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This paper was written when Dr R M Walker MB.BS., Dip DHM., was holding the position of Locum Director of Hyperbaric Medicine at the Townsville General Hospital, Townsville, Queensland, Australia.

CLINICAL MANIFESTATIONS OF THE DECOMPRESSION ILLNESSES

N.K.I. McIver

Introduction

"Many cases are on record to show the danger of lost time".

This sombre warning first issued in 1982[1] still holds good today. Despite training of both amateur and commercial divers in the recognition of decompression illness, delays at all stages are all too frequent. The implications in the light of the work of Palmer, Calder and Hughes are obvious in attempting to avoid permanent residual spinal or cerebral damage.[2]

Dysbarism embraces all the illnesses caused by changes of pressure and volume: barotrauma and decompression sickness.

Barotrauma

Changes in volume in air or gas containing spaces cause the largest proportion of morbidity and mortality, by tearing lung tissue and leading to cerebral arterial gas embolism (CAGE). This is seen in its purest form in submarine escape training where there has been no prior inert gas load taken up by the tissues. In the diving context, the distinction between arterial embolism and decompression sickness may be less clear cut when there is an inert gas tissue load as well. Collapse and unconsciousness, with or without convulsions may occur (the commonest presentation in the Royal Naval submarine escape tank training series), due to gas in the cerebral or coronary arteries. This usually occurs on ascent or immediately on surfacing and the patient may die before being recompressed.[3]

A second form may have variable symptoms, cortical in origin and may respond to recompression initially but relapse later and a third form may make a full recovery on recompression.

Where there is a fast or uncontrolled ascent followed by a rapid onset of serious symptoms, arterial gas embolism should be suspected. This, in most cases, will have occurred within 5 or at the outside 10 minutes after reaching the surface.

The following diagnostic criteria are suggested.[4]
1 Collapse or unconsciousness occurring without warning immediately or within the first few minutes after decompression.
2 Inco-ordination.
3 Confusion.
4 Weakness or paralysis of limbs.
5 Visual disturbance.
6 Unilateral paraesthesiae.

An unresponsive stupor, possibly with eyes open, has been described.

Decompression Sickness

AETIOLOGY

Gas as bubbles in the circulation or separated "autochthonous" gas in tissue can occur when local supersaturation is reached during decompression.[5-7] Extra- vascular bubbles can cause damage by occlusion of the circulation with tissue hypoxia, disruption of cells, compression of adjacent tissues as in the spinal cord, tearing of tissue or tissue planes. Intravascular bubbles can trigger a cascade of secondary events. These include activation of complement, haemorrhage or clotting, and respiratory changes causing reduction of venous drainage of the spinal cord.[8] The differing aetiology can explain both the quite bewildering variety and severity of presenting symptoms and signs described in decompression sickness (DCS).

CLASSIFICATION

DCS is classified as Type I (mild or pain only) or Type II (serious or neurological).[5] There are differing approaches to treatment of dysbaric illness based on successful regimes around the world. Most have different therapy for DCS and arterial gas embolism (AGE), and also for Types I and II DCS.[9] Inappropriate therapy is just as significant as delay in the failure of therapy or the occurrence of relapse in the treatment of DCS.

PRESENTATION

Type I DCS is obvious to the patient and to observers but it must be emphasised that this refers to pain in, or very close to, a major limb joint. All other pain must be considered a Type II event and treated as such. Examples include the unpleasant raw burning discomfort in a limb, deep back pain or abdominal pain which may be spinal in origin.

Type II DCS may also present insidiously with alteration of sensation of the lower limb (common in commercial DCS cases). This may be incorrectly ascribed to local or positional discomfort and is not infrequently unrecognised initially with potentially serious consequence later.

Type II decompression sickness was regarded as predominantly a spinal cord disease with infrequent cerebral involvement[10] but confirmation of concomitant cerebral perfusion deficits in divers with apparent spinal cord DCS raises once again the point made by Professor Lambertsen that decompression sickness is a diffuse, multifocal disease.

The clinical presentation may vary from apparently localised spinal cord trans-section (Brown-Sequard syndrome) to bizarre diffuse neurological or psychiatric syndromes and it may be extremely difficult to localise the site of the lesion.

It was traditionally taught that Type II DCS would have a latent interval from several minutes to 1 or 2 hours (or more) before onset of symptoms. This has been seriously challenged in a thought provoking paper[11] where the latent interval to presentation in 1,070 human cases of DCS was reviewed. By attempting to include only cases of DCS rather than arterial gas embolism cases, three broad categories were defined. Cerebral, spinal and cerebral and spinal.

The cerebral cases included only those with grand mal seizure, reduced level of consciousness or with a cortical picture of motor or sensory deficit. The rather vague cases of headache, fatigue, malaise or vague psychiatric symptoms were excluded, nevertheless these may be manifestations of Type II DCS and should be treated as such.

The spinal cases included only those with bilateral signs and symptoms or the cord hemisection syndrome and those cases affecting bowel, bladder or sexual function. The authors showed that Type II DCS presents within a shorter time interval (latency) when classified in this manner and removing the Type I cases which have longer latency. 56% of all cases arose within 10 minutes of surfacing, with 50% of cerebral cases arising within 3 minutes and 50% of spinal cases arising within 9 minutes.

However, the CNS cases may involve inner ear, brainstem, cranial or peripheral nerves also. The location of the site of the lesion may be in doubt leading to less precise diagnosis and inaccuracy of this classification.

Neurological DCS may present in a most rapid and malicious form with swift progression to motor loss and paraplegia. This is more common in our experience in scuba divers and may be due to a greater decompression insult or a different aetiology.

Cases have been described after dives of short duration, where there was no need for decompression stops, and yet serious spinal DCS has arisen. DCS would not have been expected. Causes of bubbles getting through the lung filter include a patent foramen ovale or other pulmonary vascular shunts.[12]

A recent paper describes cases of transient serious symptoms following a no-stop dive, suggestive of cerebral arterial gas embolism (CAGE) which resolve spontaneously. There then follows the development of spinal cord symptoms.[13] This has been called Type III DCS and may be a recognisable entity casting more light on aetiology.

Classification of Symptoms

CEREBRAL AND CRANIAL NERVE

It was believed, for unexplained reasons, that aviators were more prone to cerebral symptoms than divers. However, if one asks patients carefully there are frequent transient symptoms suggestive of cerebral gas formation. Common symptoms are alteration of balance, vision, hearing or speech. More diffuse alteration of affect, thought or memory may be obvious to trained observers (diving supervisors) whereas the patient may be the last to recognise this. Specific cranial nerves such as the facial or trigeminal may be affected.[14]

SPINAL

Any motor or sensory tract of the spinal cord may be affected by DCS. Post-mortem lesions may be haemorrhagic or thrombotic and are visible later as areas of demyelination and gliosis with sub-pial sparing.[2,15] The symptoms reflect the area of pathology but may be extremely patchy and difficult, even for a neurologist, to assess and explain.

Conclusion

Any doctor or casualty officer may be consulted by a diver who has presenting symptoms. All symptoms should

be considered decompression-related until proved otherwise in order to prevent the consequences of delay.[16] The main concern is that what is apparent clinically may be the tip of the "neurological iceberg" pathologically. No symptoms should be ignored. Of 470 cases, treated by the North Sea Medical Centre over the last 24 years, 115 patients turned out not to have had dysbaric illness. Decompression sickness can masquerade in many different guises.

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DECOMPRESSION ILLNESSES

18 months experience at the Alfred Hospital Hyperbaric Service

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Abstract

One hundred divers presented to the Alfred Hospital Hyperbaric Unit with decompression sickness (DCS, 95 divers) or cerebral arterial gas embolism (CAGE, 5 divers) were reviewed with particular attention to potential predisposing causes, response to treatment and determinants of outcome.

Twenty-six divers presented with DSC following dive profiles outside current table recommendations. The remaining 78 divers developed DCS despite diving within tables. Other commonly identified potential risk factors were multiple dives and/or multiple ascents (55 divers), rapid ascent (17 divers), previous DCS (12 divers), alcohol (6 divers) and altitude (5 divers). No risk factor could be identified in 17 divers. Presenting symptoms were often