fairly stringent protocol of assessment and education. And I document it.

I would like to see the Thoracic Society guidelines move to a system of a little bit more like that, in recognition of what James and Cathy have pointed out.

But no one is saying “Let’s let all asthmatics go diving”. Definitely not. It is really important we understand that.

Robyn Walker

We have to be careful that people reading about asthmatics and diving do not get the impression that diving is for every asthmatic. It is not. It is only for those who are at no greater risk than the general population.

I do not believe any of us rigidly follow the guidelines but we interpret them according to our own experience and the patient’s background. Twenty years ago we did not have the data on inhaled steroids which we do have today. And we do need to move on, but I feel the need to caution doctors, who may not do diving medicals very often, that not every asthmatic can dive safely. We need to be very careful in how we document our position.

Drew Richardson

PADI will still be sending divers to you, even when they utilise the RSTC form, as asthma is still screened, so affected divers are going to be ticking it and looking for advice. Perhaps what Simon has just put forward is the best approach to come up with: advice to provide to clinicians around the world who have to review prospective divers who have ticked in the yes column that they have a history of, or presently are suffering from, wheezing or asthma.

Mike Bennett

Points well made. Can we resolve then from this discussion that at some level SPUMS and the Australian Thoracic Society will investigate a joint and possibly modified position?

References


ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

MECHANISMS OF DECOMPRESSION ILLNESS

III

Bill Hamilton

Key Words

Barotrauma, cardiovascular, cerebral arterial gas embolism, decompression illness, equipment, physiology, reprinted.

Organised and chaired by James Francis, this series of popular one day precourses originally created by David Elliott and now being ably continued on this topic by James Francis, examines decompression disorders in a comprehensive way. One objective is to show what is not known as well as what is. Although intensive discussion was encouraged and did indeed occur, Dr Francis makes the point that these are not workshops, and there is no attempt to reach consensus on complex issues. This year’s program collected topics not yet covered, mainly limb and skin bends, cardiopulmonary disorders, spinal DCI, and ear barotrauma.

Simon Mitchell of Brisbane summarised the status of the search for the mechanism of pain in joint bends. He offered several hypotheses, including gas formation in the joint itself, gas in the bone marrow cavity, venous sinusoids, or under the periosteum autochthonous bubbles in pain-sensitive tissues like tendons and ligaments; a central location from which pain is referred to the joint; or the relief of inflammatory substances. The latter may explain the resistance of some cases to recompression and the tendency of some pain to migrate; bone medullary gas is consistent with the characteristic of deep and poorly localised pain.

Tom Buttolph of NMRC told about the various forms of skin bends, spanning the familiar itching and rash due to inert gas bubbles and related to skin blood flow, to cutis marmorata, the potentially more serious marbling type of lesion which may be neurogenic and related to neurological decompression sickness. He mentioned also the distinctive white lesions seen in counter diffusion situations. The final common pathway of skin rash and itching may be histamine release.

Alf Brubakk of Trondheim covered cardiopulmonary decompression illness, the most prominent form of which is the always-serious chokes, due to bubbles lodging in the lung vasculature. These bubbles cause endothelial damage. The lungs are also a site of long-term effects of diving such as reduced CO diffusivity; these effects may also be due to oxygen exposure. Some pulmonary artery bubbles (VGE) are present after most decompressions, but lung symptoms, although rare, are statistically related to DCI. In a slide
Hugh Greer of Santa Barbara led off the section on spinal cord diseases with a description of spinal DCIS as a unique trauma model, because single clinical lesions can be precisely located, whereas spinal DCIS is multifocal. For example, if symptoms are generally one-sided one should think of arterial gas embolism, but a painful, paralysed limb is often spinal cord disease. In an interesting hypothesis he suggests that apoptosis (programmed cell death) might be a pathway, the mechanism for initiation being the withdrawal of metabolic support, presumably due to bubbles or other congestion. Loss of circulation for half an hour causes no effect, after 1 hour paresis results, and blockage over 1 hour may lead to late changes. Dr Greer did not offer other mechanisms for spinal injury; three of these were covered by the next three speakers.

Ian Calder from Cambridge, UK, dealt with gas embolism, the apparent blockage by bubbles of circulation to certain spinal tracts, and offered some philosophy about the process in general. He quoted the old adage, “Know syphilis and all things medical will come unto you” and applied that to a nasty little bubble. As a pathologist he has examined many spinal cords, including those of divers without overt decompression sickness or illness who died of unrelated causes, and found many focal cord lesions that could well have been caused by bubbles. He mentioned fat embolism as an alternative possibility, and he acknowledged the cascade of effects of disturbance to the intima of blood vessels.

Fred Bove of Temple University presented the hypothesis that venous infarction may be a mechanism for spinal cord decompression sickness. Brain blood flow is 80 times that of the cord so arterial bubbles should go there preferentially, but the brain is normally spared. This venous infarction hypothesis and elegant experiments to support it were presented, with colleagues Elliott and Hallenbeck, at the Sixth Underwater Symposium in Freeport in 1972. Dogs were given spinal DCIS and X-rays taken in the chamber showed no venous plexus blood flow, and this correlated with pressures measured during lung inflation. Coagulation and microscopic studies agreed.

James Francis made the case for autochthonous bubbles forming directly in spinal tissue as the cause of the spinal DCI symptoms. He pointed out that venous infarction normally takes time to develop, but that the onset of spinal symptoms is prompt, and that clinical venous infarction symptoms are not the same as those of spinal bends. Dogs were wired for somatosensory evoked responses (SSEP) were exposed to a profile of slightly higher pressure with a shorter exposure, decompressed, and monitored for a drop in the SSEP. The spines of those showing a drop were fixed and stained, and non-staining, space-occupying lesions (NSSOLs) that looked an awful lot like bubbles! were found. These were predominantly in the white matter despite the 6:1 dominance of gray:white. These NSSOLs were not found in controls. Other animals given gas embolism by injection of bubbles showed oedema of the gray matter but none of the “bubbles.” The time course of these events agrees with the concept that a short blockage does not damage a cell, but also that there are delayed effects due to inflammation and lipid peroxidation. In submarine escape accidents with no gas loading, the bubbles go to the brain.

The next presentations considered ear barotrauma. Andy Meredith of Hastings, UK, gave a succinct history of inner ear barotrauma (IEBT), followed by a review of mechanisms and methods of diagnosis. Mechanisms include perilymph bubbles, counter-diffusion, vascular embolism, impaired performance, and membrane rupture. The diagnostic process includes consideration of the profile and time of occurrence, the counter-diffusion situation, audometry, electrocochleography, and some specialised tests. He pointed out that one has to treat these cases early lest they get well on their own, but when there is damage to the hair cells they do not regenerate. He takes issue with Otto Molvaer’s recommendation not to recompress IEBT cases and feels that suspicion of IEBT should not preclude recompression. Divers who have gone back to diving after IEBT have shown no problems.

Ian Calder looked at the pathology of ear barotrauma and decompression sickness (due to “effervescing gas”). Ear damage as a result of pressure has been known for a couple of centuries, with much of this involving the middle ear and the round and oval windows. In the inner ear, vestibular hair cells may regenerate but those in the cochlea do not.

Andy Meredith began a look at the middle and outer ear with a review of the physiology and anatomy of the Eustachian tube. Septal and sinus surgery can be effective, but the physiology can be more important here. When middle ear barotrauma occurs it is treated with pain relief, occasional decongestants, steroids, and antibiotics. Outer ear barotrauma or “reversed ear” is due to blockage of the outer ear canal. This can often be prevented with perforated ear plugs.

Frank Butler of the US Naval Special Warfare Command dealt with equipment barotrauma. Such cases can result when a heavily-weighted diver falls off the stage. Suit barotrauma or squeeze is caused by not inflating the suit during descent and may lead to skin lesions. A case shown had a pattern resembling a lightning strike. Failure to equalise a mask can cause eye damage. He presented a case of vision-threatening orbital haemorrhage with a
successful outcome. He admitted, however, that bar fights cause more orbital haemorrhage than diving. He also mentioned the reverse problem, such as over-inflation of a buoyancy device leading to uncontrolled ascent and the possibility of pulmonary barotrauma and arterial gas embolism.

This report is reprinted with minor editing, by kind permission of the Editor and the author, from PRESSURE, the Newsletter of the Undersea and Hyperbaric Medical Society, 2001; 30 (4): 2,6-7.

RW (Bill) Hamilton PhD, who was one of the guest speakers at the 1996 SPUMS Annual Scientific Meeting, is principal of Hamilton Research, Ltd., Tarrytown, New York 10591-4138, USA. Telephone + 1-914-631-9194 Fax + 1-914-631-6134. E-mail <rwhamilton@compuseve.com>.

INTERNAL CAROTID ARTERY OCCLUSION FOLLOWING SPORTS DIVING

P J Hughes

Key Words
Case report, injury, medical conditions and problems, reprinted, trauma.

Abstract

A case of internal carotid artery occlusion following a sports dive is described. The investigation and management of this condition is discussed with particular reference to blunt trauma being a risk factor for carotid artery occlusion.

Case report

HISTORY

A 28 year old lady returned from a two-week holiday in the Indian Ocean where she had been sports diving during the beginning of the second week. The day before returning home she experienced some diarrhoea. This rapidly settled with kaolin and morphine.

On the day of her return to the United Kingdom she complained of headache. She awoke several times that night and when she attempted to talk had great difficulty with word-finding. This was attributed to tiredness.

By the following morning this problem had worsened and there was evidence that both her short- and long-term memory were impaired. For example, she was unable to remember her own age, address or even her boyfriend’s surname. Her writing had become all “jumbled” and her boyfriend felt that her walking was impaired and described her walking as “slow”. There had been no further bowel disturbance and no urinary disturbance. Although she felt rather stiff there had been no rigors and no symptoms to suggest seizure activity.

Whilst on holiday she had sustained numerous mosquito bites. She had drunk only bottled water. Previous surgical intervention included an appendicectomy and right knee cartilage operation. Her only medication was the oral contraceptive pill.

EXAMINATION

She was orientated in person but not time nor place. Her speech was slow, answering “Yes” or “No” to most questions with evidence of a profound nominal dysphasia. Her memory was poor. The formal examination of the cranial nerve territory was otherwise normal. On examining the motor system she was able to obey simple commands. The limb examination revealed normal tone and power. There were no involuntary movements. There was no reflex asymmetry but the right plantar response was consistently extensor. Light touch was normally perceived. The general medical examination revealed a pyrexia of 38°C per axilla. Her pulse rate was 100 per minute and in sinus rhythm: she was normotensive. The remainder of the clinical medical examination was normal.

INVESTIGATIONS

The haematological and biochemical profiles were normal. Blood cultures were negative and films were negative for malaria parasites. Chest X-ray and CT head scan both normal. A CSF examination was undertaken, this revealed clear, colourless CSF at normal pressure; the protein was slightly elevated at 617 mg/l (NR<500); less than 1 WBC, no organisms were seen; virus was not isolated in cell culture. The CSF IgG was also increased at 40 mg/l (NR up to 30 mg/l) isoelectric focusing for intrathecal oligoclonal IgG bands was negative. An EEG examination revealed left central to temporal slow wave disturbance.

MANAGEMENT

The combination of pyrexia, speech disturbance and alteration of higher function with mild EEG changes resulted in a presumptive diagnosis of viral encephalitis. She was commenced on acyclovir.

A subsequent MRI head scan revealed areas of increased signal on the T2-weighted scans in the territory