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The above is an edited extract from Australian Animal Toxins by Dr SK Sutherland, published by Oxford University Press, covering one of the topics discussed by Dr Sutherland as guest lecturer at the 1985 Annual Scientific Meeting of SPUMS.

Dr Sutherland has copies of Australian Animal Toxins available for $45.00, which is a large reduction on the bookshop price.

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NEW ZEALAND CHAPTER OF SPUMS MEETING NOVEMBER 1985

A CASE OF CEREBRAL AIR EMBOLISM

Peter Robinson and Allan Sutherland

This 23 year old diver was referred to the Royal New Zealand Navy Hospital (RNZNH) on 24 October 1985, 3 days after her diving accident.

On the previous Monday evening, the 21st, she had made a rapid ascent from the base of a swimming pool while undergoing training with scuba. She had been practicing face mask retrieval and was breathing through scuba apparatus when things went wrong. She panicked and made a rapid ascent holding her breath. Immediately after the incident she was cold and frightened. With some reassurance, everything seemed to settle down. On her way home, she noted that her left middle finger was stiff, swollen and tense and later on that evening, it turned a dusky colour. Next day her left arm was cold and aching in the shoulder region and the finger was still a dusky colour.

Two days later she could not sleep because of the pain and aching in her left arm and shoulder. The finger had not improved, her left arm was cold with some altered sensation and clumsiness. There was no shortness of breath, no pleuritic pain and no precipitating factors which made the pain worse. At this stage on the evening of the 23rd she went to North Shore Hospital where several doctors looked at her, examined her arm only and x-rayed her hand, after which they diagnosed a case of arterial gas embolism, reassured her and sent her home.

She reached the RNZNH 24 hours later, on the Thursday evening, having mentioned to her diving instructor at Shore Watersports Ltd that she was having a few

problems. It was only through the action of her diving instructor contacting a doctor with an interest in diving medicine, that she actually got referred to RNZNH.

On examination she was pale and nervous but otherwise a reasonably well looking young woman. Her blood pressure was 160/100 in the right arm and 150/100 in the left arm. Her pulse was 70 and regular. The respiratory rate was 20. There was minimal crepitation over the left clavicle which was also tender to palpation. There was a bluish discolouration to the left middle finger, especially noticeable around the proximal inter-phalangeal joint, dorsally and ventrally. The tip of the finger was, however, a normal colour. The left arm was noticeably colder to touch than the right arm. There were a few bruises on the knees but no obvious bruises on the upper limb.'

On neurological examination she was fully oriented with no cranial nerve abnormalities noted. However, power was markedly reduced on virtually the whole left side of the body, except for the deltoid and trapezius groups. On a scale of 1 to 5 as outlined in the Undersea Medical Society (UMS) medical form, she was 4 to 4 plus in the upper and lower limbs on the left side. Tone was normal and sensation was normal. Co-ordination was abnormal, being markedly diminished on the left side. Attempting the sharpened Romberg she could stand for less than 2 seconds. Heel shin co-ordination was very poor and her gait with the eyes closed was very poor. She could, however, walk well with her eyes open. Reflexes showed clonus in the biceps and triceps muscles on the left side and clonus in the left knee jerk. There was some degree of hyper-reflexia in the right-sided triceps and knee jerks.

On the basis of left-sided weakness associated with clonus and diminished left-sided co-ordination, the diagnosis of cerebral arterial gas embolism (CAGE) was made and she was started on a therapeutic recompression table.

She was started on USN table 6. After 10 minutes, however, she felt dizzy, unwell and the oxygen was withheld. On examination at this stage she was as before with little improvement. Oxygen was recommenced and after 20 minutes it was noticed that there was no clonus on the knee or triceps on the left side, although they were still hyper-reflexic. Her hand felt as if it had improved and her finger-nose co-ordination was much improved. At 0120 on 24 October 1985, while still on table 6 at 60 feet at sea-water, she complained of chest pain and numbness on her left side. On examination her symptoms had regressed to those found at the first examination. Due to the fact that it appeared she had regressed, in consultation with Dr P Robinson it was decided to go to USN table 4 and at 0212 hours on 24 October 1985, she was commenced on table 4 with good relief before reaching 165 feet. Once there a drip was instituted and IV dexamethazone 20 mg was given. Shortly after reaching 165 feet it was noted that her reflexes were still hyper-reflexive but there was no clonus demonstrated. Throughout the rest of the decompression there were recurrences of pain in her shoulder and chest which fluctuated and lasted for a few minutes at a time.

Her peak flow was 575 on first reaching 165 feet and at 0400 on 24 October 1985 she complained of sharp retro-sternal pain which was worse when she was on oxygen. Peak flow had diminished to 530 and because of the query of possible oxygen toxicity she was taken off oxygen and continued on air. In discussion with Dr Robinson, Valium 5 mg QID was started which had quite some beneficial effect on her chest pains. At 1345 on that day however, oxygen was stopped again due to constant retrosternal pain of over an hour's duration. Her peak flow at that stage was down to 500. The rest of the decompression was fairly uneventful with the odd chest pains being reported, although intermittent. The rest of treatment Table 4 was uneventfully carried out with oxygen causing no great problems during the final stages of the table.

After the therapy she and the attendants were admitted to RNZNH for observation and although markedly improved, there was still some degree of clonus in her left knee and triceps jerks, some left sided weakness especially in the upper limb and some diminution of left-sided co-ordination, especially finger-nose and heel-shin. She complained of headaches and vague aches and pains in her shoulders.

She was discharged on Monday 28 October for rest at home.

On follow-up of 5 November 1985, it was noted that she had some diminution of power on her left arm, although only minor, a slight degree of lowered co-ordination on the left arm and clonus of her left triceps and hyper-reflexia of her left knee jerk. She complained of lethargy, headaches and the odd ache and pain in her shoulder region. She felt that she was improving day by day and apart from the weakness in her left arm which was improving, she felt much better than prior to treatment. It was interesting to note that her finger was 100 per cent normal to examination as it had been immediately after recompression therapy. She was sent off for another week's rest at home and will be reviewed after this meeting

It is worth noting that the doctors at the North Shore Hospital could reach the correct diagnosis but were unaware of the correct treatment for cerebral arterial gas embolism (CAGE). Obviously education of medical practitioners is needed.

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Dr AFN Sutherland's address is "Outspan", Bush Road, Albany RDI, Auckland, New Zealand.

The Editor congratulates the New Zealand Chapter of SPUMS on their educational efforts (see Chairman's Report page 7) and, perhaps rather optimistically, hopes that cases of arterial gas embolism will never again be diagnosed and then sent home instead of being referred for hyperbaric treatment.

DIVING KIWIS