

QUESTIONS I THINK WE SHOULD CONSIDER.

Was this really a spontaneous pneumothorax or was it pulmonary barotrauma?

What is the likelihood of a 5 cm x 3 cm bulla being detected on a chest x-ray?

How do we diagnose the small blebs apart from waiting for a pneumothorax?

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EDITOR: Readers may like also to read the case report presented by SYNEK VM and GLASGOW OL. "Recovery from alpha coma after decompression sickness complicated by spinal cord lesions at cervical and mid-thoracic levels" in Electroencephalography and Clinical Neurophysiology, 1985; 60: 417-419.

CEREBRAL ARTERIAL GAS EMBOLISM A CASE PRESENTATION

Peter Chapman-Smith

In mid March 1984, a pleasure dive ended in disaster. G, an experienced, trained scuba diver aged 22 years, ventured to his first dive site in the outer Cavalli Islands, off Mautauri Bay, a little north of the Bay of Islands on New Zealand's East Coast. He recalls a brief 15-17 minute descent to 27m (90 feet), but feeling uncomfortable in the prevailing current, he decided to surface with his buddy. At 0930 he reached the surface conscious, inflated his buoyancy compensator, and then fell suddenly unconscious within 2 or 3 minutes. In this brief time he had complained to his buddy of some numbness and paraesthesiae in his arms and legs, particularly the latter. He did not vomit nor cough up any blood.

On the previous day he had done a single scuba dive at 1500 for 25 minutes to a maximum depth of 24m (80 feet). That evening he had consumed approximately 6 cans of beer and a third of a bottle of wine at a party.

The ensuing evacuation was notable for its considerable tardiness. They contacted Whangaroa, a nearby deep sea fishing harbour, by CB radio immediately. A rescue helicopter based in Auckland, 200 miles away was requested by 1015, the nearer helicopter in Whangarei being unavailable. The diver was given oxygen via a mask when they arrived at Te Ngaere Bay at 1000. He was taken by ambulance to nearby Kaeo Hospital and regained consciousness en route. He was apparently confused but could give his name. Within an hour he developed laboured respiration and hypertonicity in all four limbs.

The helicopter requested for 1015, eventually arrived at Kaeo Hospital at 1330. He was stabilised for transport, then the aircraft headed further away from Auckland to Kaitiaki to refuel! They flew at 500 feet down the West Coast, stopping approximately every 15 minutes for reassessment by the paramedic on board. G finally arrived at the Devonport Naval Base in Auckland at 1600, some six and half hours after surfacing.

The Naval Hospital medical team recompressed him for 6 hours on a 60 foot Oxygen table. In spite of treatment he was a spinal paraplegic when he was transferred to the Critical Care Unit at the Auckland Public Hospital. He required 2 1/2 weeks of intermittent positive pressure ventilation (IPPV) and a tracheotomy. At this point he was conscious, paraplegic and with an executive dysphasia. He was transferred subsequently to the Otara Spinal Unit for 6-7 months, then to Northland Base Hospital for a year.

During his hospitalization, various investigations were done. CSF was normal. CAT scans 2 days and 5 days post accident were normal. Repeated EEG's showed initial Grade III abnormality and a subsequent return to normal. Somato-sensory evoked potentials (SSEPS) showed bilateral high cord lesions with demyelination in sensory pathways. Rapid onset lesions of the spinal cord and cerebrum were reported, which is consistent with multifocal CNS damage caused by gas emboli.

Now he is glad to be flattening again, engaged to be married and commencing work again in a local Whangarei Laboratory.

He has patchy neurological recovery with an incomplete paraplegia at roughly T5 to T7 levels. It is more like T4 on the right and T10 on the left. He has normal upper limb power, but with poor right sided co-ordination. He still has symmetrical numbness of his first and second finger tips, poor sensation in his feet, but with return of sensation in his legs, this being better on the left. He has no bowel or bladder control. His hearing remains good, as does his speech. Although improving, he has a residual short term memory deficit. A reduction in eye-hand co-ordination and in particular visual co-ordination has made reading difficult. His balance is poor. He feels as though he will fall to the left. He maintains erections, but is unable to ejaculate ("as yet" he tells me). Muscle spasms in his legs are a problem, and are controlled with relaxants. His only other medications are aperients and Ubretilid, an anticholinestase. He has frequent urinary tract infections as he self catheterises once or twice daily.

He is a non smoker. He had completed his diving training 5 1/2 years previously. He had been diving three times a week in the 5 months before his accident. These dives included bounce dives to 69m (230 feet). He had several minor undiagnosed (at the time) bends. Elbow pain came on in the boat after the dive on three occasions. He observed arm and leg numbness whilst still in the water after several dives in the last 2 years of his diving. He usually dived with twin 80 cu ft tanks, carrying a DCP, a watch and a single hose regulator with an octopus regulator.

Of particular note, he developed a cough 6 months before the fateful day. This progressed to pleurisy so he consulted his GP. He had several subsequent chest infections in the 3 months before his accident. One week before his accident he felt short of breath while snorkel training in a pool and consulted his GP again. A chest x-ray was ordered and was reported as normal. He sought specific advice about the safety of diving and was evidently reassured.

This is a tragic story, memorable for several features. He was an experienced diver, undoubtedly pushing his luck with regard to the USN tables which are the accepted sports diving tables in New Zealand. He

probably had numerous minor bends, and ignored them before his accident. His temporary unfitness to dive, with multiple chest infections, raises the question of how long divers should be banned from scuba use after a chest infection. He was given questionable medical clearance to dive. The tardy transport to a recompression facility delayed recompression. What was the diagnosis? Was it CAGE or was it rapid onset decompression sickness?

POSTSCRIPT

This case stimulated discussion and consultation with the emergence of a rapid evacuation network for diving accident victims in the far North of New Zealand.

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A CASE OF ALTERNOBARIC VERTIGO

John Knight

Alternobaric vertigo, which was first described by Lundgren in 1965, is usually noticed on ascent.¹ Lundgren was interested in the condition as he had suffered "sudden rotational vertigo during ascent from diving as well as in the hyperbaric chamber." His paper was based on the replies to a questionnaire about vertigo sent to 550 members of the Swedish Association of Sports Divers. He received 354 answers of which 92 (26 per cent) had experienced vertigo during scuba diving or when breath-hold diving which could not be explained as due to some known cause.

26 divers had suffered vertigo due to such causes as caloric stimulation, decompression sickness, seasickness, sensory deprivation, food poisoning, over-distention of the gut with air, exertion, hypoxia or hyperventilation and were excluded from the study. In this condition one middle ear pressure is higher than the other. This results in the labyrinth on that side being exposed to a higher pressure, transmitted through the round window, than the other and the result is a mismatch of messages from the two labyrinths and this is misinterpreted by the brain as a rotatory movement. The usual cause is failure of one Eustachian tube to function, so retaining air in the middle ear. Eventually, the pressure in the affected side is sufficient to open the Eustachian tube and air blows out. This equalises the two middle ear pressures and all is well again. Failure to equalise one ear completely during descent is the basic cause. The result of a failure to equalise is at least some degree of swelling of the lining of the middle ear. If this swelling is sufficient to obstruct the Eustachian tube the scene is set. Edmonds classifies alternobaric vertigo as a middle ear barotrauma of ascent in most cases but it can be due to unequal vestibular responses.² Edmonds' classification is given in Table 1.

I wish to present a case of alternobaric vertigo which not only did not commence on ascent but also continued long after the dive was over and recurred with every dive.

TABLE 1

VERTIGO IN DIVING

Due to unequal vestibular stimulation

1. Caloric
 - 1.1 Unilateral external auditory canal obstruction
 - 1.1.1 Cerumen
 - 1.1.2 Otitis Externa
 - 1.1.3 Miscellaneous
 - 1.2 Tympanic membrane perforation
 - 1.2.1 Shock wave
 - 1.2.2 Middle ear barotrauma of descent
 - 1.2.3 Forceful auto inflation
2. Barotrauma
 - 2.1 External ear barotrauma of descent
 - 2.2 Middle ear barotrauma of descent
 - 2.3 Middle ear barotrauma of ascent
 - 2.4 Forceful auto inflation

3. Inner ear barotrauma
 - 3.1 Fistula of inner ear window
4. Decompression sickness
5. Miscellaneous

Due to unequal vestibular response

1. Caloric
2. Barotrauma
3. Abnormal gas pressures
4. Sensory deprivation

The diver is a man in his 30s who has been snorkelling for 16 years and diving for 8 years. For the first seven years of his snorkelling he was quite unable to equalise his ears, which limited the depth he could dive to when spearfishing. Nine years ago he was spearfishing in a competition and was not doing as well as those who went deeper. So he went down and this time was able to equalise. However, it could not have been proper equalisation, as he was completely deaf in one ear for two weeks! Having learnt to equalise he learnt to scuba dive and had no problems with his ears until 1985.

His work involves many snorkel dives and two shallow tank dives every day that the weather is suitable. He has done this for a few years. However, in about April 1985, he started to develop vertigo and nausea after about half an hour of snorkel dives. At first this did not occur after every occasion but it soon became a regular event. As his livelihood depends on his diving he continued to work. Although he had vertigo with snorkel diving he did not have any when using scuba. After about 6 months his vertigo came on after ten minutes with the snorkel. At this time he decided to take a holiday and was out of the water for some three weeks. During this holiday he went for a trip in a boat and was seasick for the first time in his life.