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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

other doctors. The history took some digging out on occasions as the patients were reluctant to complain.

DIVE HISTORY

Most had been diving in less than 18 msw. However, 1 diver had descended to 60 msw. Frequent ascents and descents were a common feature of the dives.

TREATMENT

3 divers were operated on, with tympanotomy and the application of a fascial graft. Most received either conservative or no treatment. Rest, head up posture, carbogen (CO_2), oxpentifylline, avoiding straining and tranquillizers were tried for some. Most of the cases, being late presenters, received no useful therapy. Steroids were tried in 1 case of inner ear membrane rupture.

OUTCOME

Constant tinnitus was a feature in this group, with permanent significant sensorineural deafness on the affected side, principally in the high frequencies, usually > 4,000 - 6,000 Hz.

Two were left with less than 40 dB hearing loss, both having had tympanotomy and repair. One diver with inner ear barotrauma was left with a 40 dB low frequency loss. Two had moderate (40-60 dB) high frequency loss. Six had severe (more than >60 dB) high frequency hearing loss. One of these had a 50-60 dB loss above 2,000 Hz. Another had a loss in the left ear of 60 dB above 4,000 Hz and 70 dB above 6,000 Hz in the right ear. One of the snorkellers, who presented the same day, had a 120 dB loss above 4,000 Hz.

Three patients were lost to follow up.

Goodhill[2] described one way a round window fistula occurs. The cochlear aqueduct communicating between the cerebro-spinal fluid (CSF) and perilymph is of variable structure and calibre. If a short duct fails to damp a pressure wave from the CSF, the sharp rise in pressure in the scala tympani could cause a window rupture. The round window membrane is only 3 layers of 4 or 5 cells thick and of variable thickness, 10 µ centrally to 70 µ peripherally. Alternatively, the pressure wave could rupture through the basilar and Reissner's membranes to involve the utricle and saccule. A further mechanism is excessive rapid inward or outward movement of the ear drum. The first raising the inner ear pressure by pushing the ossicles in and the second, by suddenly pulling on the ossicles in the oval window, dragging the round window inwards beyond its tolerance. This explains why inner ear barotrauma can be associated with sudden middle ear barotrauma or a forceful Valsalva manoeuvre.

I have made no attempt to consider the possible causes of sensorineural hearing loss and vestibular symptoms as seen in DCS or inner ear haemorrhage on ascent. No doubt some "occupational hearing loss" cases seen in general practice stem from these causes.

These cases are only those that were recognized. I suspect there are in fact many more that pass unrecognized. The preponderance of middle ear barotrauma is as expected. The extremely common sinus barotrauma and cases of otitis externa have not been shown in this study in their true numbers because they are so common, and were not recorded.

The new data on right to left cardiac shunts with potentially patent interatrial septa, suggests that inner ear air embolism may be more common than previously considered. As exact diagnosis is not easy and with their late presentations, one can only urge medical colleagues to take an often lengthy history, to use an auriscope, to use an audiometer and to try to identify these patients with inner ear barotrauma, who can do so well with early appropriate treatment.

Case 1

A 26 year old farmer presented in February 1990 with a story suggestive of bilateral round window rupture. He had been diving 2 or 3 times a week, using a 5 mm wetsuit and hood, for 3 years. He first had problems diving in a fresh water lake in Northland. In 1984, experiencing difficulty clearing his ears underwater, he developed a sudden pain in his left ear. He felt generally unwell for a week after. He recalled imbalance and falling over to one side.

Some 3 years later, whilst cray hunting, he swam hard on scuba to a cave at 6 m for about 30 minutes. Feeling well he had a 45 minute surface interval. The next dive was to 12 m, with a number of descents and ascents. 15 minutes into the dive he felt unwell with marked nausea and vertigo. Surfacing, he felt quite giddy, was vertiginous and vomited a lot. He remembers a yellow mucoid post-nasal drip after the dive. On closer questioning, he admitted to recurrent episodes of vertigo, giddiness, nausea and vomiting underwater. He had had approximately 12 attacks over the previous 2 years. The onset of symptoms was usually when diving in a swell or near the surface. At presentation he felt he was deaf bilaterally. He clearly recalled the two episodes of acute onset pain before the vestibular symptoms being on opposite sides.

His past medical history was not abnormal. He took no regular drugs. But he had some allergic rhinitis. Occupational exposure to noise had occurred.

On examination, apart from minor otitis externa, only a severe bilateral high frequency hearing loss was noted, 60 dB above 4,000 Hz in the left ear and 70 dB above 6,000 Hz in the right.

The diagnosis was presumed bilateral round window rupture.

Case 2

This 28 year old worker presented 1 month after diving in the sea at Taupo Bay on the Northland coast in 1987. He recalled a day with a lot of surge. He had scuba dived to 10-12 m and suddenly developed nausea and vertigo underwater, vomiting several times. He experienced tinnitus and a dull feeling in his right ear. He often had problems equalizing. Examination showed a serous middle ear effusion and an 85 dB loss at 6,000 Hz.

Following admission to the local base hospital, there was no improvement in his serial audiograms and he was taken to theatre three days later. At tympanotomy, a round window rupture was noted and the fistula was patched. He had a splendid result with only a 35 dB loss in that ear and no vertigo, however some tinnitus still persists.

Case 3

A 22 year old ticketed scuba diver of 1 year's standing, who had had no prior problems with his ears went spearfishing in the Whangaruru Harbour region in January 1989. Snorkel diving to recover a spear, he developed considerable vertigo when back on the surface. Climbing out onto the nearby rocks, he noted nausea, left sided deafness and tinnitus. These symptoms persisted for 5-6 hours. At that time his balance was poor.

The next day he had a planned scuba dive lined up. The first scuba dive was to 12 m chasing crays and 45 minutes went by without problems. After a surface interval of 20 minutes, they dived again to 12 m. He developed vertigo in the water with marked nausea and surfaced feeling unwell. Having lost his buddy, he dived again to seek him! Not locating the other diver and feeling worse, he surfaced and vomited in the boat a number of times. Marked vertigo continued.

Hewent to the local hospital casualty room. Next day he still had nausea, giddiness, deafness and vertigo. His audiogram showed a 60 dB loss above 6,000 Hz. The local consultant was away he was sent to hospital in Auckland. Although he had presented acutely his round window rupture with a perilymph fistula, was treatment conservatively with rest, carbogen (CO_2), oxpentifylline, and steroids.. His audiogram never improved, although his vestibular symptoms were reduced. He was given a hearing aid and advised that to resume diving in 2 months would be safe !

Case 4

This 33 year old, experienced diver presented in December 1989 after scuba diving with a friend looking for crays. During the first descent he had some difficulty in clearing his ears. The dive profile of the first dive was to 18 m for 20 minutes, followed by a surface interval of 30 seconds. A second descent to 18 m for 20 minutes. He then surfaced again for 1 minute as his buddy had lost his weight belt. Then a further descent to 18 m for just 5 minutes. Then there was a surface interval of 50 minutes.

The second dive was to 18 m for 25 minutes, then he slowly ascended to spend sometime at 9 m and to do a 6 minute stop at 3 m depth. Following the second dive he noticed a left hearing loss, but no vestibular symptoms such as nausea, vertigo or tinnitus. The ear felt blocked.

He sought advice from the on-call GP and was prescribed an antihistamine. He presented to his usual GP ten days afterwards. A middle ear effusion was noted with a negative Valsalva manoeuvre on the left, poor mobility of the tympanic membrane and an apparent conductive hearing loss. In fact a severe high frequency loss was present, 75 dB above 6,000 Hz in the left ear. There was minimal improvement 1 month later, after a course of prednisone, which was suggested by the local ENT consultant, for presumed pure inner ear barotrauma. His hearing loss appears to be permanent.

Case 5

A 41 year old professional man presented acutely in January 1989 after scuba diving for scallops in the Bay of Islands. There was no significant past medical history. Trained 3 years previously, he dived regularly.

The first dive involved many descents and ascents to 7.5 m over 45 minutes. His catch bag was attached to his buoyancy compensator. Developing cramps in his legs, he started to feel generally mildly unwell. On his last dive he coughed, ingesting compressed air from his regulator. On surfacing he felt quite unwell and noted the sudden onset of left upper abdominal pain. He snorkelled 70 m to the waiting yacht. Clambering aboard, his abdomen felt distended. Passing urine and having a bowel motion slightly relieved his discomfort. He then vomited, lay down, and was taken to the wharf at Russell.

Here he was very well received and resuscitated by the local GP and his nurse. He was placed in the left Trendelenberg position and given oxygen. Advice was sought over the phone. He complained of back pain and left upper quadrant abdominal pain and paraesthesiae and mus-

cle twitching in his lower legs.

He was transferred by helicopter to Whangarei. Here he was further assessed. He was pale and clammy, but alert and orientated, with an irregular pulse. His cardio-vascular system was otherwise stable with good cardiac output and respiratory status. Arterial blood gases showed a PO_2 of 250 mm Hg (on 6 l/min O_2), PCO_2 of 52.9 mm Hg, otherwise they were normal. An ECG and chest X-ray were normal. An intravenous drip was inserted and he was sent on to RNZN Hospital in Auckland for recompression.

In transit, despite 30 mg of papaveretum given intramuscularly he was in constant discomfort, rubbing his abdomen and some shakiness of his left leg continued. Treated with a table 6A in the naval recompression chambers, his symptoms resolved readily under pressure. He was neurologically normal the next day and was sent home symptom free. The differential diagnosis included aerophagy, mesenteric arterial gas embolism, free peritoneal gas or a combination of these.

References

1 Edmonds C, Lowry CJ. and Pennefather J. *Diving and subaquatic medicine*. 2nd edition revised. Sydney: Diving Medical Centre, 1983 392

2 Goodhill V. Sudden deafness and round window rupture. *Laryngoscope* 1971; 81: 1462-1474

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THE DEVELOPMENT OF THE RECREATIONAL DIVE PLANNER

Ray Rogers

Summary:

Depth increments in the US Navy tables are too great. The repetitive dive table is based on the slow-responding 120 minute tissue compartment, so little surface credit was obtained. This compartment was largely irrelevant to recreational diving. The repetitive group format was unsuitable as the time/depth benefits were unharmonious and times were excessively rounded off, creating anomalies. Research suggested lowering of no-stop limits. It become apparent that wholly new tables were appropriate as the USN tables were too "coarse" were not planned for extensive repetitive diving and the USN tables do not permit multi-level diving.

The compartment structure seemed wrong as there were too few compartments and they were internally inconsistent. I added compartments and adjusted their values. The 120 minute compartment never seemed to have an effect while the 40 minute tissue usually controlled the dives. The 60 minute tissue occasionally controlled and was chosen for controlling the repetitive dive calculation. The resulting table was more conservative and the time penalties were not great. The 120 minute compartment is important in long, deep dives with staged decompression.

The basic concept of theoretical model was Haldanian, retaining exponential gas exchange and a spectrum of tissue compartments. The modifications were variable maximum allowable tissue pressures, an increased number of compartments, Hempleman's power function used for the non-stop curve, shallow and deep asymptotes added, the no-stop curve was smoothed and faired, "M-values" were derived from this curve, discontinuities were eliminated and no-stop limits were lowered.

The table was designed as a circular slide rule in polar format. Multi-level capability was included. The procedure is that the dive always goes from deep to shallow. Adjustments were made to keep pressures within limits by minimum depth differentials on ascents and time restrictions were added to no-stop limits. Safety stops at 15ft/5m were recommended after all dives. These stops are required after some dives, those deeper than 100ft/30m and when within 3 pressure groups of any limit. The advantages of stops are: a dramatic reduction of tissue pressures, compensation for staying too long, compensation for diving too deep, compensation for gauge or timer error, and a slower ascent rate. There are special rules which require long surface intervals occasionally. These are when pressure groups become very high, as after repeated long, shallow dives. The rules are seldom required, but they exist.

Introduction

It is a great pleasure to be able to speak to SPUMS about the development of the Recreational Dive Planner, so enthusiastically and overwhelmingly embraced by over 95% of the medical community Down Under. Unfortunately those people have been so struck dumb by the brilliance of it all that they have remained absolutely mute and have not been able to comment. But the five per cent who are not excessively enthusiastic have written horrible letters and and numerous articles. So I would ask that they briefly give a few moments of their attention to this talk about the development of the Recreational Dive Planner (RDP) and the corporation that was created to develop it, Diving Sci-