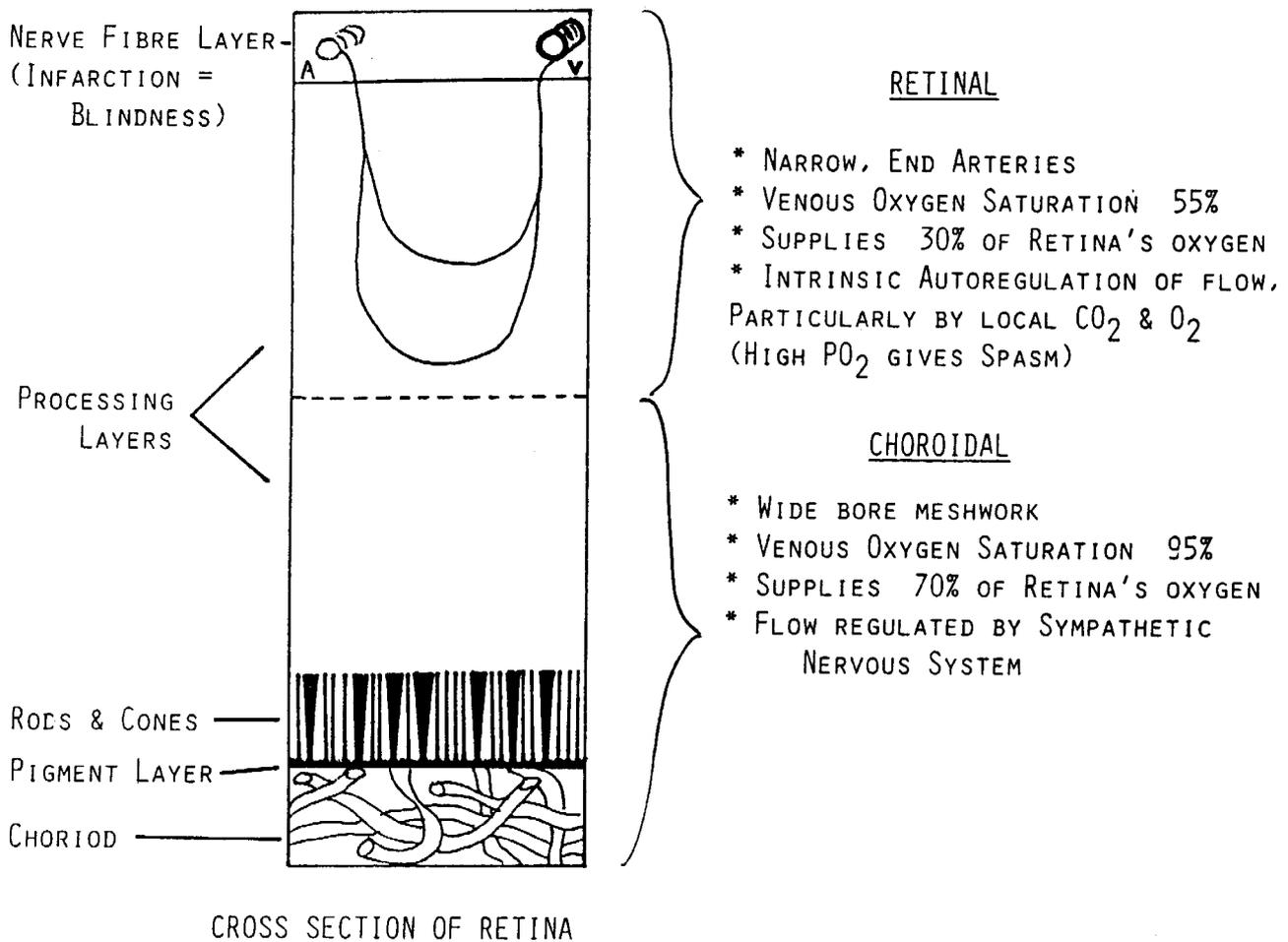


THE RETINAL AND CHOROIDAL CIRCULATIONS



If compression and hyperbaric oxygen had not been used he would have been a candidate for enucleation. At the time of writing, both his eyes appear normal, and move normally. There is only a small supero-temporal area of visual field left in the left eye. There is no pain or discomfort. The patient has made a good adjustment to his injury, and is grateful for the treatment as he realises that the injury could have resulted in him losing his eye.

Thanks are expressed to Drs E Ryman, Deputy Director, Royal Hobart Hospital; Michael Treplin, Ophthalmic Consultant to the Royal Hobart Hospital, David Griffiths and Michael Martyn, Department of Anaesthesia, Royal Hobart Hospital; and G Brian, Department of Ophthalmology, Royal Hobart Hospital.

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PULMONARY BAROTRAUMA OR SPONTANEOUS  
PNEUMOTHORAX  
A CASE PRESENTATION

Beris Ford

Let me introduce you to my area. I live in Whangarei which is 160 km north of Auckland, New Zealand, and is in the middle of a long isthmus, the East Coast of which is popular for scuba diving. The Poor Knights Group of Islands lie 30 km off the coast. They are New Zealand's Mecca for scuba divers and about 20,000 dives per year are done here and perhaps 250,000 in all of Northland.

J, a part Maori, was 20 years old when she came in to see me for a scuba diving medical. There was not much in her history, a couple of bouts of cough with sputum, and occasional nasal obstruction. There was nothing much in the examination except she had larger lungs than predicted, Vital Capacity (VC) 4.3 l, predicted, 3.8 l, Forced Expiratory Volume in 1 second, 3.45 l, predicted, 3.25 l, and a slight reduction in percentage FEV<sub>1</sub>/VC, actual 80 per cent, predicted 88 per cent.

Despite my urging, she refused to have a chest film because she would not take time off work. She worked in the Government note printing factory where security is naturally very strict. To attend for an x-ray would mean taking a whole day off work.

Because of pressure from her and her diving instructor, I passed her, but explained carefully my reasons for requesting a chest x-ray.

I next saw her 4 months later and had forgotten about her medical and her diving, and she did not bring it up. She attended with a chest discomfort, and a cracking noise in her chest. The diagnosis is so obvious now but since she gave no history of chest injury or diving at the time I did not suspect it. She was tender over the sternum and the left 2nd rib. Again she refused to have a chest x-ray and I asked her to come back if it did not resolve rapidly,

She did not do anything for another 2 months when she attended the doctor at her place of work. Then her symptoms had gradually progressed and she was short of breath and had a tachycardia. By that time she had definite signs and he was more successful in getting her to have an x-ray. This showed a complete left pneumothorax and some shifting of the mediastinum.

Taking a more detailed history disclosed that she had been diving on most weekends since her qualification. She was therefore no longer a novice but on her own admission, not very confident.

She had a dive several weeks before her first presentation. This was to 10 metres, and she just did not feel right. She had difficulty staying down and she felt she was floating up all the time. She felt uptight and panicky. After the dive she felt abnormally tired and exhausted.

It was about two to three days later that she first noticed what one described as the cracking noises and slight retro-sternal discomfort. It was a week or so later that she first sought medical attention.

You will now be aware that it took 3 months from the dive to the diagnosis of her pneumothorax. Once diagnosed, she was admitted to hospital.

A chest drain was placed and despite the use of negative pressure she lung did not expand fully. It was almost fully expanded after 8 days but by about 13 days she developed an effusion with infection. The fluid grew an enterobacteria.

A few days later, because of the failure of conservative management, a surgical operation was sought. Here, there was no delay and she was taken to theatre the next day.

At operation, she had a normal bronchoscopy. About 700 ml of blood stained fluid was present in the chest. There was a fibrin coating over the lung and it seemed that this might have been the reason why the lung would not expand. There was no evidence of any site of leaking when the lung was inflated. However, a 5 cm x 3 cm cyst was found in the apex. The cyst was removed and a pleurectomy was performed. Ten days later she was sufficiently recovered to be discharged.

She has, of course, been advised to discontinue scuba diving. Her next foray on the sporting scene was to take up parachuting. This can also have hazards for individuals with lung problems. Parachuting did not last long as she developed back pain and x-rays showed her to have a spondylolisthesis. So parachuting did not seem to be such a good idea.

## DISCUSSION

I found it quite difficult to get much out of the literature on pneumothoraces and could find nothing on pneumothoraces and diving at the Auckland Medical School. I did get some papers on spontaneous pneumothorax. This may well not be too wide of the mark as in the case of J, it is not really possible to say whether this was a spontaneous pneumothorax or whether there was an abnormal pressure change, a true barotrauma aetiology. In this case, the treatment was as for a spontaneous pneumothorax.

A variety of treatments for spontaneous pneumothorax have been used.

1. Simple aspiration.
2. Silver nitrate pleurodesis and drain.
3. Tetracycline pleurodesis and drain.
4. Under water drain, with or without negative pressure.
5. Surgery.

About 95 per cent resolve satisfactorily with no treatment or with intercostal drainage. These may be about a 20 per cent recurrence rate in the conservatively treated group.

## CAUSE

It is stated that it is much more common in young adult males, and that is nearly always due to rupture of a sub-pleural bleb or bulla. What causes the blebs or bullae is not known for certain. inflammation, ischaemia and congenital causes have been suggested.

## HISTORY

Air in the pleural cavity was described as far back as the mid 17th century. It was first called pneumothorax by Itard in 1803, and in 1826 it was seen to be caused in one case by rupture of an emphysematous bleb. Schminckle, in 1928 described multiple blebs in the apices of both lungs in a young man who died from bilateral spontaneous pneumothorax. Dolley, in 1929 performed the first known successful surgical treatment for a ruptured bleb in a child aged 3 months. A number of reports of surgical treatments appeared in the thirties and by the early forties pleurodesis in simple forms was reported.

In one review of patients coming to surgery, 94 per cent had blebs, and these were multiple in 60 per cent. The blebs varied in size from 0.5 x 0.5 cm to 1 x 2 cm. In another series 90 per cent had smoked for from two to thirty years before the incident. There was also a bias toward heavy smokers.

## 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 Ubreid, 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