To the Editor:

Dear Sir,

VERTIGO IN DIVERS

Thank you for asking for my comments on Noel Roydhouse’s letter which I have read with interest. He is technically correct in stating that vertigo is not inevitable when the tympanic membrane ruptures. Vertigo is not dependent on the size of perforation that is sustained, but is dependent on the rate of ingress of cold water into the tympanic cavity. The caloric effect produced by rapid ingress of water is entirely dependent on the temperature difference between the water that has entered the tympanic cavity and body temperature. Once the water temperature reaches approximate body temperature then the caloric effect ceases as does the vertigo. As ENT surgeons we use this caloric phenomenon for testing labyrinthine function (vestibular function); and under test conditions we use water at 30°C and 44°C, in ears with intact tympanic membranes, in order to induce a vertigo. If an article were to be directed at “diving doctors” I think that it would be fair comment for it to be stated that a vertigo sustained on descent should be assumed to be due to a tympanic membrane rupture with rapid ingress of water, until proven otherwise, by inspection of the ear; if the tympanic membrane is found to be intact, then a diagnosis of perilymph fistula must be made, which will only be proved or disproved by performing an exploratory tympanotomy. The above statement refers to vertigo sustained on descent, NOT ASCENT.

Of course in both conditions of ruptured tympanic membrane and suspected perilymph leak, the subjects involved must undergo puretone air and bone audiometry as soon as possible, post incident; so that a baseline audiogram is obtained. Needless to say it should be mandatory for all divers to undergo a puretone audiogram before they ever start to dive, only then will pathology, sustained while diving, of an otological nature be picked up early.

Ruptured tympanic membranes sustained on ascent do occur, usually on free ascent, such as on submarine escape, and at a depth greater than 30 metres. I never saw a patient who had escaped from the submarine escape tower at HMS Dolphin, who had sustained an isolated tympanic membrane rupture, without sustaining pulmonary barotrauma. I have fitted grommets into the tympanic membranes of volunteer submariners who were unable to equilibrate their middle ear pressures, so that they could undertake the submarine escape tank training, none sustained a caloric effect, nor did any subject develop an infective otitis media, I am certain that as the subject ascends the expanding gas in the tympanic cavity passing through the iatrogenic perforation, prevented water entry. My experience of this is of fifteen subjects, not statistically significant, but I think fifteen more than anyone else.

The vertigo of ascent is not a caloric phenomenon, but is the manifestation of an alternobaric vertigo, in which one middle ear pressure has a greater positive pressure than the other. The only vertigo of ascent that I have seen that has not been due to a vestibular bend has been due to an alternobaric vertigo. Most subjects who suffer from this form of vertigo also tend to have an inability to equilibrate their middle ear pressures with ease, with one ear clearing before the other.

I am not quite certain as to the relevance of the two cases that are quoted who sustained a sensorineural hearing loss while diving, what is obvious is that Case Two is entirely different from Case One. Case One suffers from an intermittent eustachian tube dysfunction, having sustained an haemotympanum one year previously, and having had difficulty in equilibrating his middle ear pressures on the dive which resulted in his hearing loss. I would not be surprised that on close questioning, it would be found that the subject has had several “sticky ears”. This eustachian tube dysfunction can be cured surgically by removing the vomer-ethmoid suture. The hearing loss is hard to explain, for in my experience hearing losses of a sensorineural type sustained while diving are usually high tone. It of course may be explained by intralabyrinthine membrane rupture.

The Case Two, is a totally different problem, if this case had presented in a naval diver, one would instantly think in terms of a vestibular bend, even from 13 metres; note the comment about the rapid ascent. She should have been recompressed, if the vertigo had disappeared, the diagnosis would have been one of a vestibular bend, the history is one of a decompression injury. The hearing loss affecting the high tones together with the vertigo and the long lasting unsteadiness for at least 17 days would cause me to make a diagnosis of decompression injury to the right ear.
If Dr. Whittaker stated that sudden hearing loss sustained while diving should be regarded as suspected perilymph leak, which should be treated as an emergency; he is absolutely correct. In my experience of 59 confirmed perilymph leaks, as determined by operation, only two perilymph leaks of the round window type presented with any history of vertigo. The only case of oval window fistula sustained while diving presented with severe vertigo and sensorineural hearing loss. I would also state that it is highly unlikely that a perilymph leak to occur on ascent or for there to be labyrinthine membrane rupture in a subject with normal eustachian tube function, as the eustachian tube opens, without requiring any muscular activity when the middle ear pressure reaches 100-200 mm of water. On rapid ascent the subject is exhaling and therefore is not usually consciously equilibrating the middle ear pressures. If Case Two had normal eustachian tube function, then her diagnosis must be decompression injury. As a rule of thumb, vertigo on descent, especially if preceded by otalgia means tympanic membrane rupture until proved otherwise. Vertigo and hearing loss after ascent equals decompression injury to the vestibule, and requires the use of a decompression chamber, or further descent and slow reascent if in the bundu. If recompression abolishes vertigo, diagnosis is vestibular bend, if unabolished, perilymph leak or labyrinthine membrane rupture.

I think that Noel Roydhouse has been dogmatic in his statements; in being so he has left himself open to criticism; as I have demonstrated in Case Two.

I am sorry that I have taken so long to reply, but I have been on leave and have had one or two problems in my fight to set up the definitive ENT Department at Mansfield; this as taken a fair amount of my time.

Anyway I hope that my comments will be of some use. By the way, would there be an opportunity to write an article for the BJSM on a topic, such as ENT problems in Diving and their management?

With kind regards,

Yours sincerely,

WILLIAM D. McNICOLL, FRCS(G), DLO

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OBITUARY

Dr. Samuel Leonard Simpson, MD, FRCP

Within a few days of writing the obituary for Dr. J. B. Adams we learnt of the death on August 3rd of another founder member of our Association, Dr. Leonard Simpson, who died at the age of 82. This distinguished endocrinologist qualified in 1925, having first been awarded a double first in the Cambridge Tripos. He obtained the MD in 1930, having gained MRCP in 1928, and was elected FRCP in 1940.

His first interests were in diseases of the chest but after a stay at the Mayo Clinic he became interested in the new science of endocrinology; diabetes and later the steroid treatment of Addison’s disease. His book on major endocrine disorders became the definitive text for two decades. After the Second World War he was appointed Consultant Endocrinologist to St. Mary’s Hospital, Paddington, and as a colleague of our co-founder Lord Porritt, he was one of the small group of doctors who founded the British Association of Sport and Medicine. He was able to apply his sporting enthusiasm in boxing, riding, tennis and golf to the medicine of sport, particularly to its endocrinological aspects.

Apart from medicine, his connections with sport were extensive. On his brother’s death he became an active partner in the family’s world famous business, noted especially as sports outfitters, and became Chairman of the Company in 1957.

During the last few years, he was unable to participate in many BASM activities, but he gave us his support up to the time of his death. We extend the sympathy of this Association to his widow and to his daughter.

H. E. Robson