CASE REPORTS

CASE 1 Diving on compressed air to 160', with surface decompression. In the interval between surfacing and entering the recompression chamber, he complained of pain in both knees and then collapsed. He was placed in the recompression chamber where he recovered consciousness. His symptoms were relieved at 60' and he was decompressed on a minimal recompression oxygen table (Table 6 USN). Following this he was easily tired, and a week after the incident he was exhausted after a simple surface swimming job and climbing back to the diving platform.

Nine days after his episode of decompression sickness his ECG showed ST depression in lead 2 and an inverted T wave in leads 3 and AVF. A week later the report read "T wave inversion in L3 and AVF is now less obvious suggesting a recent cause". Later he was investigated in hospital. The resting ECG showed ST and T wave abnormalities. An effort test ECG showed increased ST depression. He did not complain of chest pain during this exercise, however, immediately after stopping the exercise he had an episode of bradycardia and hypotension. During this time he experienced the same symptoms as had occurred while he was swimming after his episode of decompression sickness. Coronary arteriography showed normal coronary arteries.

A year and a half after the incident he was getting chest pains on exertion and his ECG showed T wave inversion in leads 3 and AVF, and ST segment changes in V5 and V6. The report stated that 'these changes are consistent with either old inferior infarction or with postero lateral ischaemia.'

This man had a normal ECG eight months before his decompression sickness.

CASE 2 Dived on compressed air to about 180', 22 minutes bottom time. Decompression stops in the water according to USN Diving Manual as for a 25 minute dive at 180'. While undressing, he noticed paraesthesiae and numbness in his loins, then he developed pains in legs and shoulders, difficulty in co-ordination, severe malaise, tiredness and nausea. This was followed by difficulty in taking a deep breath precipitating a coughing spasm. He also developed a rash.

He was recompressed, 27 minutes after surfacing and his symptoms were relieved at 40'. He was treated on a minimal recompression he had a recurrence of the malaise and the onset of swelling over large parts of his body. He was then again submitted to therapeutic recompression, this time without obtaining symptom relief.

The next day he noticed a 'fluttering' in his chest. It only lasted a few seconds, came on at any time and was unrelated to physical exercise. This occurred about 100 times a day. He had never experienced such a sensation before. He felt slightly faint and had a mild headache during the palpitations. An ECG taken 4 weeks after the onset of palpitations showed ventricular extrasystoles. After exercise the ECG showed slight depression of the ST segment in leads 2, 3, AVF, V4, V5 and V6 and slight elevation in AVR. The T waves became inverted in V1, slightly bifid in V2, biphasic with predominant primary inversion in V3 and biphasic with less deep primary inversion in V4.

A week later his resting ECG again showed occasional ventricular extrasystoles while after exercise there were more frequent multifocal ventricular extrasystoles, blunt slightly bifid T waves in V4 and very slight depression of ST segments in leads 2, AVF and V5. As the man had had a normal ECG recorded two and a half months before his episode of decompression sickness the final diagnosis was decompression sickness involving the skin, joints, abdomen, cardiac and neurological tissues.

On the basis of the above every deep diver and commercial diver should have an ECG recorded and retained for reference. If he is unfortunate enough to develop severe decompression sickness his ECG should be recorded when he finishes therapeutic recompression and again a week later. In this way there will be objective evidence of the presence or absence of myocardial damage due to decompression sickness available to assist in the assessment of any Workers Compensation claim that is made.

While it may be that these three cases represent the total Australian experience of cardiac changes following decompression sickness, it is possible that there are other men similarly affected whose case histories have not reached the School of Underwater Medicine.

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MANAGEMENT OF CHLORINE GAS POISONING

At 9.00am on 6th June 1973, fourteen men were admitted to Western Suburbs Hospital following exposure to Chlorine gas. The men were working in the vicinity of a chlorine supply tank which was being loaded from a road tanker. Apparently, the outlet valve was not closed before the tanker was disconnected. The result was that chlorine escaped under pressure into the area in which the men were working.

All the affected personnel left the are immediately (except one who donned a gas mask and attempted to close the valve).

On arrival, all showed signs of varying degrees of respiratory distress. All were tachypoeic and had audible rhonchi and coarse crepitations throughout their lung fields. None were clinically cyanosed. Each were given oxygen to breathe at two to three litres per minute through Edinburgh masks. A chest x-ray was taken of each patient on admission. None showed signs of recent lung disease.

Spirometry (FEV and Vc) and peak flow (PF) measurements were made on all the patients. These showed that there was a considerable degree of bronchospasm in all the patients. Twelve of the men were selected randomly and divided into four groups of three. A single dose of Orciprenaline 20mb was given to group A, Aminophylline 100mg to group B, Choline Theophyllinate 200mg to group C and Terbutaline Sulphate 5mg to group D. The FEV_1 Vc and PF were measured again at one and two hours after administration of the bronchodilators.

All of the patients showed a considerable improvement over this period. Six hours after the first dose of bronchodilator was given, the parameters, FEV_1, VC and PF were measured again one and two hours after the doses. This procedure was repeated after a further six and then nine hours so that each of the four medications was given once to each patient.

Had the effects of the chlorine been more prolonged, it may have been possible to use this information as the basis of a clinical trial to determine the relative efficacies of the four medications. As it happened, all but one of the men were fully recovered within six hours so that the last three doses of medication had no significant effect. They were discharged after 24 hours with the exception of the one man. He developed a cough and crepitations in his lung fields. He was treated with IPPR and tetracyclines and discharged the following day.

In summary, the management of acute chlorine gas poisoning was as follows:

1. History - initially brief with reference to severity of symptoms, pervious history of heart and lung disease and smoking habits.
2. Physical examination - a rapid assessment of pulse, BP, cyanosis, respiratory rate was made together with auscultation and percussion of the lung fields.
3. Investigations - Chest x-ray, FEV_1, Vc and PF rate.
4. Treatment - oxygen, bronchodilators, antibiotics (in one case)

All but one of the patients were clinically recovered within six hours and discharged in 24 hours.

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NOTE: Dr Roger Tuck is an intern of the Professorial Unit of Sydney Hospital on secondment to the Western Suburbs Hospital, Croydon. Sent in by Dr P Hamilton.

LABYRINTHINE DYSFUNCTION DURING DIVING - SYMPOSIUM 1973

This is a very subjective and personal impression of the papers at the workshop sponsored by the Undersea Medical Society on Feb 1 and 2, 1973 at Duke University Medical Centre, Durham, North Carolina.

The meeting was chaired by Joe Farmer, Assistant Professor in the Division of Otolaryngology. The opening remarks by David Elliott suggested that the proceedings of the meeting would not be published as such, as this would allow more genuine discussion on the controversial issues. For the sake of the South Pacific Underwater Medicine Society, these rough notes were made:

Lecture 1 - Dr Hugh Barber, Professor of Otolaryngology, University of Toronto. Barber described the functional anatomy of the vestibular endorgan proposing that this be looked upon as a transducer. The central connections were considered to be programmed to compare the vestibular input on each side.

Lecture 2 - Dr Brian McCabe, Professor of the Department of Otolaryngology, University of Iowa. McCabe described also the functional anatomy of the central vestibular system supporting the concepts of Barber. He reiterated that there was a resting discharge in the vestibular system which increased on one side as it decreased proportionately on the other. In normals the vestibular responses are considered to be equal and opposite and if this does not occur then is orientation and vertigo would result.

Lecture 3 - The Clinical Evaluation of Vestibular Dysfunction. The Clinical Evaluation of Vestibular Dysfunction was again given by Brian McCabe. McCabe described two axioms in clinical history. Firstly that continuous labyrinthine vertigo cannot last more than one to three weeks. Secondly, that labyrinthine vertigo of any consequence will always have labyrinthine nystagmus. He then described the vestibular function tests.

Lecture 4 was from Hugh Barber again, and this was on the principles and clinical uses of electronystagmography.

Lecture 5 was also from High Barber, and was on the symptoms, signs and pathology of vestibular end-organ disease.. This was a classical textbook description of the symptoms and signs of this disorder. The only new feature to this narrator was the information that occasionally when one inserted a needle in the oval window one could obtain endolymph. This occurs when the saccule is greatly expanded with the endolymph.

Lecture 6 was from High McCabe on central vestibular disease and was a reiteration of the textbook teaching.

Lecture 8 The eighth lecture was given by Dr Robert S Kennedy, Lieutenant Commander and head of the Human Factors Engineering Branch at the Naval Missile Centre, Point Mugu, California.

Dr Kennedy reiterated and summarised the work that he has already presented in his NMRI Report - certainly the most complex bibliography on vestibular function abnormalities in diving.

Lecture 9 was given by Dr Carl Edmonds, School of Underwater Medicine of the Royal Australian Navy, and five cases of inner ear damage from the effects of barotrauma were presented. Audiogram and electronystagmograms were presented showing that the effects of middle ear barotrauma of descent may have either temporary or permanent damage effects on the cochlea and/or the vestibular apparatus. In the 5 cases, 3 were shown to have surgically correctable lesions by repairing the round window perforations. The clinical history of these cases all seemed consistent, ie. there was initially a difficulty in equalising the pressures within the middle ear clefts during descent, and usually there was an excessive attempt at forceful Valsalva manoeuvres. Following the dive there was usually a history of tinnitus with or without vestibular and cochlear damage; in the cases of round window fistulae the vestibular and cochlear damage progressed following the dive and was corrected only after the surgical repair was performed. Another case demonstrating the effects of ascent on middle ear pressures and the production of nystagmus was illustrated with ENG's performed during compression and decompression. A case was made for the inclusion of 2 other provocative tests in electronystagmography as it applies to divers. The firs to these was the use of bilateral equal and simultaneous caloric stimulation thus mimicking the environment of the diver, the second was the use of electronystagmography while undergoing compression and decompression changes. This was termed 'dysbaric ENG' tests and again mimics the influence of pressure changes on the diver it is pointed out that the latter test could only be performed with the diver in the upright position whereas the former test ie. the bilateral simultaneous caloric could only be performed in one of the Hallpike positions. Following this lecture there was some degree of criticism regarding the absence of any publications from the Australian workers. However this was considered invalid as the work is mentioned in at least 3 medical journals and one book.

Lecture 10 was the vestibular disorders occurring at stable depths and this was prepared by Dr Sundermacher of the University of Pennsylvania and Dr Lambertsen, the Director of the Institute of Environmental Medicine at the University of Pennsylvania.