Having carefully predicted death, and before contacting you, I treated the animal with:

6mg Dexamethasone 1/peritoneal; 2/100mg Atropine; 2 minimums 1:1000 Adrenaline 1/peritoneal and20mg Doxapram HCl 1/peritoneal. This was obviously based on the principle of 'kill or cure', and only the adrenaline appeared to produce much response, basically an improved resp. rate for a short period.

Endotracheal intubation was performed and initially oxygen administered but the basic inability of the practice to supply someone to give 1 pump to a respirator bag each 15 minutes for an indeterminate but obviously long period meant that we finally disconnected the oxygen though the tube remained in place.

After your advice, we then did nothing further but admit the anomal and keep a watchful eye, and occasionally prod it to be sure life still remained. It got gradually more limp - if such was possible - slower corneal reflex and dehydrated looking over the period, and was finally declared dead on the morning of 11th March.

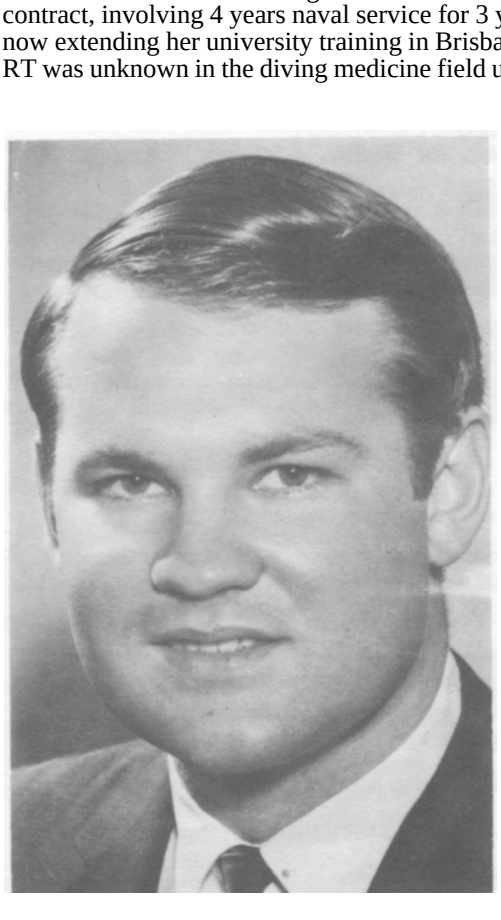
No post mortem examination was made as the owner wanted the skin. If I never see another crocodile I will be quite happy.

This may be something to add to knowledge of tetradotoxin poisoning of crocodiles.

Sincerely
(Sgd) E Fisher, Veterinary Surgeon, Deagon, 4017

PERSONALITY PROFILES
(or Dangerous Marine Animals That I Have Met)

Robert Thomas is a graduate of the University of Queensland (1968) and a self confessed victim of the RAN Short Service Comm contract, involving 4 years naval service for 3 years undergraduate training. He married Denise, an exuberant and attractive arts graduate, now extending her university training in Brisbane, and they have a bouncing baby tadpole named Natalya. Although having an interest in d RT was unknown in the diving medicine field until he joined the RAN School of Underwater Medicine - following negotiations which were fully explained - for a period 'which must not exceed 6 months, I don't want to be a spec



He then became involved in a series of publicity gimmicks centred around the emer decompression sickness treatments to both divers and caisson workers. There is no acknowl relationship between his enthusiasm in making emergency dashes to Melbourne, and the fa his family now live in the same capital city. Then came a series of publications, in both m journals and books, which demonstrated that RT could write as well as he could cure. One books soon topped the best selling list of the Australian Medical Publishing Company, a of his articles had reprint requests exceeding 600, within the first 12 months of publishin a non-specialising general practitioner, this is not a bad effort.

His work was acknowledged when, following 2 years full-time work at the SU became classified by the Australian Navy as a Specialist in Underwater Medicine. During he became the Officer-in-Charge of the School, and a Consultant and Adviser to many Gover Departments in both diving and hyperbaric medicine. He was one of the foundation memb SPUMS and sat on the executive committee for 2 consecutive years. He was a prime mo the Diploma of Diving and Hyperbaric Medicine, and one of the main organiser of 2 conse Annual General Meetings. During 1973 he also took over the editorial post of this Newslett was responsible for its improvement from an anecdotal loose leaf rag to a high quality bo He was co-founder of the Diving Medical Centre, which no utilises temporary centres eastern states of Australia, and received rapid acclaim from the civilian and commercial although the Diving Medical Centre is geographically located in Sydney, with the recent m RT to Queensland, it is likely to flourish in that state.

Bob left the Navy in December 1973 and entered the underwater life of Brisbane capacities. One was as a General Practitioner with interests in hyperbaric medicine and a de to diving medicine. The other was as a flood victim with all his uninsured equipment and fu at the bottom of a rather expanded Brisbane river. These floods were the only State disaste the last decade, which cannot be directly attributed to the activities of the Premier Bjelke-Pe Undaunted, although perhaps a little soggy, we expect Bob to emerge and keep up the activ of 1970-73. A hard worker, a brilliant organiser and a most competent writer, we can exp hear more from RT in future - but at this time we acknowledge the debt that diving medi the South Pacific area owes this man.

ELECTROCARDIOGRAPHIC CHANGES AND MYOCARDIAL DAMAGE FOLLOWING DECOMPRESSION SICKNESS

R John Knight, FFARACS, Surgeon Lieutenant-Commander RANR, Royal Australian Navy School of Underwater Medicine

Summary

Three Australian divers developed ECG changes following severe decompression sickness. The aetiology of the condition is discussed and the literature reviewed. Two case histories are presented.

In the past four years, three cases of divers who had severe decompression sickness followed by changes in the electro-cardiogram have come to the notice of the Royal Australian Navy School of Underwater Medicine at HMAS Penguin. None of these divers was servicing in the RAN. The three men were commercial divers diving from oil exploration rights in widely different past of Australia. They all developed severe decompression sickness with involvement of the central nervous and respiratory systems. Involvement of the cardio-respiratory system in decompression sickness is accepted as evidence of gas bubbles in the blood, the symptoms of 'the chokes' being attributed to bubble emboli blocking the passage of blood through the lungs. Under these circumstances, it is possible for bubbles to pass into the arterial circulation through a patent ductus arteriosus, an atrial or a ventricular

septal defect or via the pulmonary plexuses. It is possible for bubbles to form in the blood which has passed through the lungs. These bubbles may embolise throughout the body and occasionally the area affected is the coronary circulation.

This is not a new observation, however, it has seldom been reported in divers, and most of the reports deal with caisson workers (Breu 1940; Caccuri and Graziani 1950; Zannini 1954; Caccurri et al. 1956; Aston 1957; Zannini and Odaglia 1958; Zannini 1967). As many of the reports are in Italian or German, the work reported will be reviewed as an introduction to the Australian case reports.

Breu in 1940 reported that 41% of caisson workers complained of 'hyperexcitability of the heart'. ECGs showed abnormally high T waves which were interpreted as signs of myocardial damage.

In 1950 Caccuri and Graziani reported their findings in 28 caisson workers affected by decompression sickness. Several had myocardial 'trouble' and a tendency towards deviation to the right of the QRS axis. This became a more frequent finding and was more accentuated the longer the men worked in caissons.

Zannini reported in 1954, 14 workers treated for decompression sickness of whom 7 showed right axis deviation, 2 had ST depression, 2 had inverted T waves which reverted to normal in 3 days and 5 had intraventricular conduction defects.

In 1956 Caccuri et al. published their findings in 90 workers treated for decompression sickness. The ECG changes included 20 diagnosed as auricular and ventricular hypertrophy, intraventricular conduction defects (1 with intermittent bundle branch block and 2 with partial bundle branch block), abnormal rhythms including ventricular extrasystoles and nodal rhythm, signs of subendocardial ischaemia and two unsuspected myocardial infarcts. Using the vector cardiogram they diagnosed 'myocardial trouble' in 40 cases.

In 1957 Alston reported a case of severe decompression sickness in a Norwegian diver treated on board HMS Adamant. The case report is of interest as the man was relieved of all his symptoms on recompression to 165'. He was decompressed on the RN Table III and for the first 6 1/2 hours his progress was excellent. At that time when he had been at 30' for 3 hours, there was 'sudden onset of severe cardiac and respiratory embarrassment, the patient becoming almost unconscious. His heart was fibrillating and pulse almost imperceptible at over 100. On recompressing to 60' he recovered rapidly but felt again a pain in his left lower limb, which, however was only temporary'. There was no ECG being taken, so the diagnosis of atrial fibrillation is purely clinical. The retrospective diagnosis to explain his collapse rests between the sudden onset of a cardiac arrhythmia and the sudden onset of the 'chokes'. As he has been at the same pressure for three hours, it seems unlikely that there was a sudden large increase in the rate of bubble formation in his blood, especially as the rest of his decompression was without incident. It would seem more likely that as the result of a bubble embolus in a coronary artery he developed an anoxic area in the conduction path and that this led to the sudden onset of arrhythmia. Prompt recompression presumably restored the blood flow to the anoxic area and cardiac rhythm reverted to normal. It is of interest that this man's ECG was normal approximately 4 days after his collapse. Zannini and Odaglia reported 11 cases of decompression sickness in caisson workers. These patients had had the ECG's recorded before, during and after recompression. Any changes in the pre recompression tracings had reverted to normal after recompression and therapeutic decompression.

In none of these series is there information about the ECG's before the episode of decompression sickness.

In 1965 during a symposium on decompression sickness, Zannini summarised much of the preceding work and presented his findings in 74 patients with decompression sickness. Of these 33 showed ECG abnormalities (Table 1). He summarised his views on the causation of these changes.

1. Changes in rhythm and heart position during the stay at high pressure. These were bradycardia, right axis deviation, ST elevation, clockwise rotation of the yeart. They were attributed to changes in autonomic tone, increased oxygen tension, reduction of abdominal gas and lowering of the diaphragm. The changes could regress on return to atmospheric pressure or persist for some time.

2. Changes due to the workload. These were increased potentials from left or right or both ventricles with an enlarged QRS complex and flattening of the T wave in left leads. He was unable to explain why caisson workers developed these changes more often than workers doing similar jobs at atmospheric pressure. The changes were transitory.

3. Changes usually detected in decompression sickness, but which can occur without decompression sickness. These include pulmonary P waves, notched P waves, depressed ST segment, flattening or inversion of the T wave in leads 2 and 3, and lengthening of the PQ interval. These may be due to bubbles interfering with the pulmonary circulation.

4. Changes due to coronary or myocardial damage due to aeroembolism. These are rare.

TABLE 1		
	Notched P waves	8
	Pulmonary P Waves	7
	Right ventricular hypertrophy	2
	Inverted T waves	2
	Flat T_1 to T_{1-2}	3
	Nodal rhythm	2
	Partial bundle branch block	8

ECG abnormalities detected in 33 out of 74 patients treated for decompression sickness.

The Australian cases differ from the previously reported series as two of the men are known to have had normal ECGs before their episodes of decompression sickness. One man developed ECG changes which later disappeared. One man developed multifocal ventricular extrasystoles. The third man developed chest pains and 2 1/2 years later had ECG changes consistent with either an old inferior infection or posterolateral cardiac ischaemia. As is to be expected in a disease with the protean manifestations of decompression sickness the three men sustained differing amounts of cardiac damage. All three men had an interval of days between finishing therapeutic recompression and the onset of their symptoms. A similar delay in the onset of ECG changes in carbon monoxide poisoning ha been reported by Hayes and Hall.

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.