South pacific underwater medicine society

Newsletter

C O N T E N T S

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EDITORIAL

If Medical publications such as this appear to dwell unduly upon the mishaps that afflict man in his passage through life, one must remember that just as without crime there could be no police so would a truly trouble free environment make the medical profession redundant. It is in this sense a matter of benevolence on the part of Inscrutable Providence that such a plethora of interesting yet tricky problems bedevil our attempts to explore and utilise the portions of our planet that are covered by the waters. Luckily the dangers cause more morbidity than mortality and appear to be capable of avoidance, though always at the expense of creating even more complex problems. Once upon a time submersion created a simple surface-or-drown option. Thanks to mankind's ingenuity our choice of evils is greatly expanded. Pandora appears to be the patroness of Man, the Master of His World, and to have a special interest in diving medicine! These complex problems are being progressively illuminated by the extracts from "Diving and Subaquatic Medicine", a further chapter of which appears in this issue.** "Accidents" are the raw ore from which we fashion our Speciality.

The recent Melbourne meeting provides two articles, covering High Pressure Oxygen and Dysbaric osteonecrosis (a disease of many names). The bone damage is still regarded by many divers as something that only effects other people, so we can confidently expect many cases to "occur" in the near future now that it is being sought by doctors. The only known prophylaxis is the conservative interpretation of the diving tables for "shallow" dives and great and continued caution when deep dives are undertaken. You <u>can</u> get decompression sickness from "no-decompression" dives and this is one reason for the rethinking now occurring regarding the design of safer tables of depth/times/stages. This new approach was applied to the Deep Dive we are kindly permitted to reprint and is the basis of new decompression meters now being designed in Texas. Only one thing is certain, diving by luck and acceptance of minor symptoms of DS is stupid and damaging. Dr Knight keeps DS before us with a case report of an incident.

Accidents are by definition unexpected by the victim. Just how unexpected is illustrated in the case of Pulmonary Barotrauma due to a diver at the surface being alternately covered and exposed by large waves while himself remaining at a fixed point and breathing from his air supply. This practical application of the pressure differential cause of air embolism is not one usually considered. The Provisional Report on the 1975 diving related deaths contains other examples of the unexpected being potentially preventable.

Our curiosity concerning these tragedies is justified only if we use the information to further our understanding of the factors that most critically effect the outcome of the ever present context between the pro and anti-life forces of the environment, Divers, even more than most, must be aware of this critical equation while about their business within the waters.

** Extracts from "Diving & Subaquatic Medicine" will appear in the next issue of SPUMS Newsletter.

SUBSCRIPTIONS

Members pay $15 yearly. Associate membership for those neither medically qualified nor engaged in hyperbaric nor underwater related research is available for $10. The journal is sent up to four issues yearly to both full and associate members. Those resident outside the immediate Australasian area should write for the special terms available.

Treasurer: Mr Phillip Rubenstein, 4 Highfield Crescent, Kew, Victoria 3101.

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Notes to Correspondents and Authors

Please type all correspondence and be certain to give your name and address even though they may not be for publication. Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide same in a presentation suitable for photo-reproduction direct. Books, journals, notices of Symposia, etc will be given consideration for notice in this journal.

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Disclaimer

All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of SPUMS.

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OXYGEN TOXICITY
Dr John Knight

General Effects of Hyperbaric Oxygen

If the arterial partial pressure of oxygen is raised to over 2.25 ATA there is sufficient oxygen dissolved in the plasma to supply the body's needs. So a person can be kept alive even if his haemoglobin is useless. This effect is made use of in treating carbon monoxide poisoning with hyperbaric oxygen. At the same time the partial pressure of oxygen throughout the body will be raised well above normal, which is used clinically in the treatment of gas gangrene.

However high partial pressures of oxygen have their inevitable effects. Above 2.25 ATA the haemoglobin remains fully saturated so carbon dioxide transport is interfered with. There is vasoconstriction and damage to various organs and enzymes. The organs that have been shown to be adversely effected by raised oxygen partial pressures include the central nervous system, the lungs, the eye, the bone marrow, the kidneys, the gonads and the liver. I will mainly be discussing the effects of oxygen on the central nervous system and the lungs.

Tissue Effects of Hyperbaric Oxygen

Life developed when the atmosphere contained little or no oxygen but plenty of carbon dioxide and water. Oxygen release into the atmosphere started as a result of photosynthesis. Enzyme systems developed and are now adapted to work at 0.21 ATA oxygen. It is not surprising that we get trouble when we expose these enzyme systems to higher pressures of oxygen. Oxygen is known to be biochemically toxic at a number of sites. Some of these are sulphydryl enzymes; thio containing co-enzymes, such as lipoic acid, co-enzyme A and reduced glutathione (GSH); flavoprotein enzymes, particularly those containing non-haem iron and sulphydryl groups; enzymes requiring pyridoxal phosphate as a co-enzyme (of particular interest here is glutamic acid decarboxylase (GAD) which forms gamma-amino butyric acid (GABA) in the nervous system); and lipids undergo peroxidation. Possible mechanisms involved in the inactivation of enzymes by oxygen are; firstly an enzyme with two SH groups could have them both oxidised to form a disulphide linkage, this may be the mechanism for the inactivation of Glyceraldehyde phosphate dehydogenase (GAPD); secondly, an enzyme with a single SH group could react with another enzyme molecule also carrying a single SH group but this reaction is not as likely as the third reaction which represents the prior oxidation of non-protein cellular compound such as glutathione, followed by mixed disuphide formation. This last reaction is reversible and is also the reaction by which oxidised SH enzymes can be reactivated, by a substance such as reduced glutathione (CSH).

There are two possible ways that sulphydryl groups can be oxidised. Increased concentrations of oxygen may drive, by mass action, reactions such as the oxidation of glutathione towards the right. An alternative is that free radicals are formed during hyperbaric oxygenation and that these inactivate the sulphydryl groups by forming disulphide linkages and water.

Carbohydrate mechanisms are susceptible to the toxic effects of oxygen in at least five places.

1. In glycolysis, glyderaldehyde phosphate dehydrogenase is quite easily inactivated. It can be reactivated by incubation with an SH donating agent.
2. The next step that has been found to be inactivated by oxygen is the oxidation of pyruvate, and this may involve the oxidation of either lipoic acid or co-enzyme A.

3. In the tricarboxylic acid cycle several dehydrogenases contain SH groups which have been demonstrated, in vitro, to be inactivated by oxygen.

4. In the respiratory chain there are a number of flavoprotein enzymes that are exceptionally vulnerable to oxygen toxicity.

5. Finally oxidative phosphorylation, the formation of ATP linked to the reactions of the respiratory chain, is also vulnerable to oxygen as it depends on the presence of free SH groups.

Not all enzyme systems are oxygen sensitive, some are oxygen resistant such as the gas concentrating mechanism of the swimbladder of fish. Below 100 metres (11ATA) swimbladder gas is 85-95% oxygen. The swimbladder P O2 can be approximated to 0.09 x depth in metres, which gives pressures of 100 to 200 ATA in some species. It is thought that the low temperature at which deep sea fish exist (less than 5°C) may protect against oxygen toxicity as may pressure itself by preventing any oxidation in which water is an end product.

General forms of Oxygen Toxicity

The two major forms of oxygen toxicity were both described many years ago. In three years time it will be 100 years since Paul Bert described convulsions in animals exposed to high pressures of oxygen. Twenty years later in 1899 J Lorraine Smith described the other important effect of breathing increased partial pressures of oxygen. This came on at lower pressures and was the inevitable result of breathing oxygen at more than 0.5 ATA. This form of oxygen toxicity affects the lungs leading through a sequence of sore chest, a decreased vital capacity, cough, increased respiratory rate and eventually to respiratory failure and death. That is of course in animals as experimental humans are not usually exposed long enough to develop respiratory failure. If an animal is exposed to high oxygen pressures and convulses to death it does not live long enough to develop the toxic changes in the lungs.

Oxygen toxicity can creep up on us completely unexpectedly. There is a disease called retrolental fibroplasia which causes blindness in babies. It occurs occasionally and the pathology is a growth of fine blood vessels into the vitreous humour of the eye, which is normally without blood vessels, and then cellular infiltration blocking light from reaching the retina. About 25 years ago there was a sudden epidemic of retrolental fibroplasia in premature infants. After a few years it was worked out that this was due to the apparently commendable practice of giving extra oxygen to all premature babies as they lay in their humidicribs. Stopping the oxygen stopped the epidemic, although sporadic cases still occur. That was oxygen at less than 1 ATA.

Oxygen at 3 ATA is toxic to the eye and three hours exposure has led to a symmetrical contraction of the visual fields. Vision remained but had been reduced to a cone of 10° and the normal field is a cone varying between 60° and 80°. The subjects in this experiment retained their limited vision until they went unconscious from the other central nervous system effects of oxygen. Within an hour of being returned to sea level their vision had been fully restored. But another man was not so fortunate. He had had eye symptoms, due to retrobulbar neuritis, some time previous to his exposure to oxygen at 2 ATA. By the end of two hours he had developed almost complete loss of vision in the eye that had been affected before. He had been exposed to less oxygen than most patients having hyperbaric oxygen therapy. He was most unlucky and was left with a permanent visual defect in the middle of the field of the affected eye.

Even if we keep the oxygen partial pressure down below 0.5 ATA we can cause the body trouble if there is no other gas present. Gemini 4, 5 and 7 were space flights where the oxygen pressure was below 0.5 ATA and above 0.21 ATA. There was no other gas present in the space craft. The crews all suffered from a large decrease in the red blood cell mass. There was a similar but smaller decrease in red blood cell mass in the crew of Apollo 9 but no change occurred in the crews of Apollo 7 and 8. The difference was that the Apollo missions started with 0.6 ATA of nitrogen in the cabin atmosphere. Apollo 7 and 8 retained this nitrogen for the whole trip but Apollo 9 was depressurised in flight for a space walk and repressurised with oxygen only. So the crew was exposed to 6 days of pure oxygen.

Central Nervous System Oxygen Toxicity

We know that high oxygen partial pressures decrease GABA levels in the brain, that the decrease precedes the convulsions, and is reversible, the decrease is specific for GABA among amino acids. Susceptibility to convulsions correlates with the rate of GABA decrease for different species, for different pressures and for different carbon dioxide concentrations. The same oxygen pressure that produces convulsions decreases GABA. And GABA given intraperitoneally protects some animals from oxygen convulsions.

GABA oxyglutaric transaminase, the enzyme that destroys GABA is normally only found in the mitochondria. Glutamic acid decarboxylase, the GABA forming enzyme is found normally in the nerve endings astride the mitochondria. GABA levels are normally determined by the GAD activity rather than the ABA-T activity. Membrane permeability probably plays a major role in the control of GABA levels by keeping GABA away from GABA-T. Extracellular GABA is involved in the inhibition or modulation of nerve transmission. The reductions in brain GABA, induced by high oxygen partial pressure, could be brought about by any one of the following mechanisms.

1. Inhibition of glutamic acid decarboxylase

2. Activation of GABA oxyglutaric transanimase.

3. Increased membrane permeability which would allow GABA more rapid access to GABA-T.

There is good evidence that glutamic acid decarboxylase is inhibited by high oxygen pressures and that increased catabolism by GABA oxyglutaric transanimase also occurs, which is thought to be due to greater permeability of the membranes to GABA.

GABA is an inhibitory transmitter in the central nervous system and it is assumed that the oxygen induced decrease of GABA reduces CNS inhibition so allowing the incoordinate actions that lead to convulsions.

For the diver the most important form of oxygen toxicity is the acute nervous system effect as this can lead, without warning, to convulsions and if you have a convulsion underwater and lose your breathing apparatus you drown. It is also highly inconvenient for a patient to have a convulsion in the confined space of a single man hyperbaric chamber.

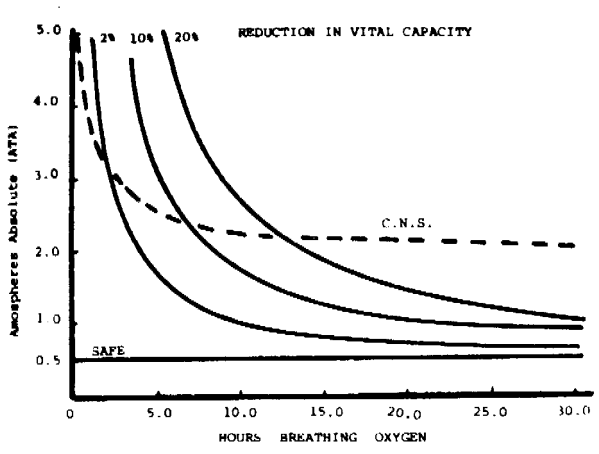
The toxic effects of high oxygen pressure on the central nervous system can be likened to acute poisoning. The victim suffers cerebral changes, twitches, inco-ordination and convulsions. The symptoms often occur immediately after the oxygen pressure has been reduced and before the arterial partial pressure has had time to drop. In one series 40% of convulsions occurred during decompression. We know that there is an

extreme variation of tolerance to oxygen not only between individuals but also for the same individual on different days. The time of exposure before the onset of symptoms is decreased as the pressure is increased. Symptoms occur sooner with men in water than with men in a dry chamber. Work greatly reduces the tolerance to high oxygen pressures. Both these effects are probably due to increased P CO2, the result of the inability of the standard oxygen diving set to absorb completely high carbon dioxide outputs.

In one experiment firemen, who might have had to fight fires in pressurised tunnels, were exercised wearing oxygen sets in a pressure chamber at the RN Physiological Laboratory at pressures equivalent to depths of 20- 47 feet of seawater. They were wearing 57.5 lb of equipment and were exercised for 40 minutes, 2 minutes work and one minute rest, at a rate that left them almost exhausted. They had approximately 87% oxygen in the breathing bag. There were no signs or symptoms in these men working hard at pressures equivalent to 20 to 23 feet of seawater. The first signs of oxygen toxicity that occurred were fasiculations and small twitches of the facial muscles (described as "the lips"). After 29 feet, which is just below two atmospheres, approximately 50% of the men had "the lips". They were not usually noticed by the subject, appeared during rest periods and disappeared during exercise. There was no trouble keeping the mouthpiece in place. These were the minor symptoms.

Young defined major symptoms as those that endanger a man under pressure, severe nausea, dizziness, light-headedness, confusion, euphoria and convulsions. One man convulsed during exercise and one during decompression. These serious symptoms started at 35 feet, just over 2 ATA, and became more frequent after 41 feet. The signs that these men had intensified during decompression while they were still breathing oxygen. 14% of the signs started during decompression, usually within 5 seconds of starting decompression and always within 10 minutes. They diminished rapidly during decompression and were gone by surfacing.

In the Navy oxygen sets are limited to a depth of 25 feet, a total pressure of 1.75 ATA, which has been shown to be safe for the endurance of the sets. This graph shows the levels of oxygen exposure, expressed as pressure and time, at which one may expect central nervous system and pulmonary toxicity. The dotted line marked CNS shows the exposures at which central nervous system toxicity leading to convulsions can be expected. The solid line across the bottom shows the level at which no toxic effect on central nervous system or lungs have been reported, although blood changes do occur at this level. It appears to be safe to breath oxygen at 0.5 ATA in nitrogen indefinitely. The other three curves show the exposures that have been found to give the indicated reductions in vital capacity. A small reduction in vital capacity develops quite quickly even when breathing oxygen at 1 ATA.



Patients in hyperbaric chambers are usually exposed to pressures of 2.25 to 2.5 ATA for periods of not more than two hours which gives them all the advantages of, and as few of the disadvantages of, hyperbaric oxygen treatment. They are in the safe zone. Safe from central nervous system toxicity, unless they are very intolerant

of oxygen, and safe from the pulmonary effects while their bodies are being drenched in oxygen. They are at rest which reduces the chances of CNS symptoms.

Divers being treated for decompression sickness are usually given oxygen at 60 feet, 2.8 ATA, and oxygen breathing is interrupted every 20 minutes by 5 minutes air breathing, which retards the onset of oxygen toxicity and also allows the patient to drink if he wants to. He doesn't always want to, as nausea and vomiting are some of the earliest symptoms of oxygen toxicity. My authority for this statement is Geoff Macfarlane, who has had considerable experience treating divers who have developed decompression sickness in Bass Strait. Even with air breathing to delay the onset of toxicity, therapeutic exposure to oxygen at 60 feet, 2.8 ATA, is limited to a total of 60 minutes, in all 75 minutes at 60 feet after which the chamber is depressurised to 30 feet, 1.9 ATA. Again the patient is at rest. Divers who have decompression sickness are exposed to high oxygen levels in an effort to increase the excretion of inert gases from their blood into their lungs. So one is walking a tightrope between oxygen toxicity and inadequate excretion of inert gas. This becomes quite a problem after long exposures to high pressures.

Pulmonary Oxygen Toxicity

Divers breathing compressed air are breathing more than 0.5 ATA below 50 feet. However the effects of nitrogen narcosis will come on and incapacitate them long before they reach 300 feet, the level at which the partial pressure of oxygen in compressed air is 2 ATA. 2 ATA of oxygen is known to have caused convulsions in divers. Luckily humans are more resistant to the pulmonary effects of oxygen than most experimental animals. A few hours exposure to a raised oxygen partial pressure, followed by a rest period on the surface, does not appear to do any permanent damage. But for saturation dives it is normal practice to keep the oxygen levels below 0.5 ATA to prevent the onset of pulmonary oxygen toxicity.

One situation where non-divers may develop pulmonary oxygen toxicity is in being rescued from a submarine. Australian submarines are fitted with buoyant ascent equipment. The sub has an egress hatch surrounded by a twill trunking coming down close to the deck. To escape the compartment is flooded, compressing the air in it to the outside pressure when it is possible to open the hatch. The twill trunking prevents the air from whooshing out of the line hatch. Each man in turn dips under the trunking, inflates his life jacket, and is borne irresistibly upwards. Over his head is a plastic hood, open at the bottom to vent excess gas, which allows him to breathe normally on the way up. Such ascents have been made by the RN from as deep as 300 feet. But the escapees are exposed to the risks of decompression sickness and of being lost when they surface. Another approach is that used by the USN and the Swedish Navy. They have built and are building underwater rescue vessels designed to mate with a hatch on the stricken sub and transfer the crew at failure. So we are forced back to the animal model and the nearest animal to humans that has been well documented histologically is the monkey (Macaca Mulatta).

Many reports of patchy collapse as a complication of breathing pure oxygen 1 ATA have been published based on postmortem evidence. However Kapanci and his co-workers showed that this is a postmortem effect, and that if the lungs are fixed in the inflated position immediately after death there is quite a different picture. It is from their work that this section of my presentation is taken. Their monkeys were exposed to oxygen at 1 ATA for up to 13 days.

Changes in Lung Tissue in Monkeys

In the monkey 15% of lung volume is tissue and the rest is air. After a week of breathing oxygen there is a vast decrease in normal lung tissue. After twelve days the lungs have virtually no normal tissue left and the total tissue volume has nearly doubled. Some monkeys removed from oxygen and allowed to recover. To get them out of the oxygen environment safely they had to be weaned by gradually reducing the oxygen partial pressure as rapid reductions made them anoxic. The monkey which was sacrificed 56 days after its 7 day exposure recovered so that almost three-quarters of its lung was normal tissue but it still had more lung tissue than the controls. The monkey sacrificed 84 days after its 13 day exposure had about 90% of its lung tissue normal and the septal volume was almost back to normal. Both had patches of abnormalities scattered haphazardly throughout the lung. The abnormalities were of various grades of disorganisation.

Both monkeys and humans have a destructive and exudative phase as the first signs of pulmonary oxygen toxicity. There was a steady increase in interstitial thickness with exposure to oxygen. During the first few days this was due to an increase in interstitial fluid which more than replaced the volume of the cells destroyed. This was the early destructive and exuderive phase. If the monkey survived this there was a later proliferative phase in which there was a steady increase in the volume of cells and fibres.

After four days the alveolar walls were severely damaged. The alveoli contained oedema fluid and cellular debris and macrophages. 90% of the membranous, type 1, pneumatocytes were damaged. The cells were swollen and had ruptured membranes and fragmented cytoplasm. Some were detached from the basement membrane which was left bare or covered with fibrin strands. There was a small increase in the air-blood barrier. By seven days the type 1 pneumatocytes, normally 85% of the alveolar lining, had been almost completely destroyed. Their replacement by type 11, granular, pneumatocytes thickened the alveolar walls. The epithelial part of the air blood barrier was now 1.7 mu instead of 0.6 mu in the controls. The endothelium varied in thickness from region to region and the interstitum was filled with fibroblasts and leucocytes. About this stage many of the monkeys died from respiratory failure.

At 12 days the alveoli were lined by cuboidal, type 11, cells. The alveolar spaces were decreased by increase in volume of the septa which were thickened by many fibroblasts and inflammatory cells as well as by the thicker epithelial cells. The air blood barrier was over three times as thick as in the control animals.

Following exposure to oxygen there is a large increase in the volume of the epithelium complete destricution of the type I, membranous, pneumatocytes and overgrowth of the type II, granular, pneumatocytes. In those animals that survived exposure the normal proportion of epithelial cell types was not restored even after many weeks.

To recap. The main changes in monkey's lungs are a large increase in epithelial thickness, a lesser increase in interstitial thickness and little change in endothelial thickness. The process can be divided into an early destructive and exudative phase, peaking at about 4 days, and a later proliferative phase.

Species differences exist in response to the same exposures. The rate doubles the thickness of his blood air barrier in three days while the monkey in the same time has no significant change.

Human Symptoms and Signs

Now to leave the animal world and come to humans. Clark and Lambertsen reported the symptoms that were complained of by people exposed to oxygen at 2 ATA. The symptoms started with mild carinal irritation on deep inspiration, went on to occasional coughing, then pain on inspiration, then frequent coughing, intense carinal irritation, uncontrollable coughing, severe pain on inspiration and then dyspnoea. The decrease in vital capacity was correlated with the symptoms but came on before the subject complained. Clark and Lambertsen chose the decrease in vital capacity because it was something that they could measure, whereas symptoms are very difficult to measure. It would be very nice to have this early evidence of oxygen toxicity when treating patients in a recompression chamber. But there are problems. Water spirometers are excellent until the pressure is reduced and the air in them expands and water goes everywhere. Electrically driven portable spirometers like the Vitalograph cannot be used in pressure chambers if you do not want to risk a fire as it is inadvisable to take electric motors into high oxygen environments. There is a mechanically driven recorder on the market but it is made by the Japanese for the Japanese and has a maximum of 4.5 litres which is not large enough to cope with the average Australian diver. Vane spirometers, such as the Wright Respirometer and the Drager Volumeter, are affected by the increased density of the compressed gas and the rate of flow past the vanes.

The vital capacity decreases and other oxygen toxicity effects do not clear up immediately. The vital capacity changes in three individuals who had been breathing oxygen at 2 ATA were all different. All subjects had a continuing decrease in vital capacity in the first four hours post-oxygen when they were breathing air. The man with the least decrease in vital capacity had returned to a normal vital capacity by the third day after discontinuing oxygen. The man with the greatest decrease however had a normal vital capacity by the second day, while the man with the intermediate decrease took eleven days to regain a normal vital capacity. This emphasises the individual variation in susceptibility to oxygen toxicity.

To sum up

Oxygen is not a harmless drug if given for long periods at more than 0.5 ATA. Certainly if given for 24 hours at 0.75 ATA the subject will complain of chest symptoms and will have suffered a decrease in vital capacity. At higher pressures pulmonary toxicity comes on quicker and progresses more rapidly. At pressures above 2 ATA central nervous system symptoms and signs can be expected. This is a limitation in hyperbaric therapy. In treating decompression sickness in divers who have been deep and so require a prolonged therapeutic decompression pulmonary oxygen toxicity can be a complication of great severity preventing the use of raised oxygen partial pressures. The only way to avoid oxygen toxicity is to keep within the experimentally determined safe limits and even then there can be surprises owing to the great variations in individual susceptibility.

Clyde Cameron, when Minister for Science (September 1975) put the case against ill considered scientific programs very succinctly. “To repose confidence in crash programs in science is as realistic as advising a woman that she could produce a baby in a month by putting nine men on the job.” Such remarks surely require no relating to diving to be worth repeating!

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Commons' Speaker: "Rigs are Ships"

The UK House of Commons was in uproar (28 May 1976) , the votes evenly decided as to whether Oil Rigs were ships, so should be Nationalised, or Oil Rigs. The Speaker had the casting vote and disregarded the Government's wishes by His decision that such platforms would be included in the assets that would be Nationalised. He also voted to ensure that the bill was passed. Everyone being displeased, a brawl ensued. As Divers in Australia seem to be governed under the Scaffolding Acts we can hardly laugh too loud.

Graeme Henderson, the Perth maritime archaeologist investigating the wreck of the James Matthew in Cockburn Sound, has disproved the cherished mariners' theory that if you tickle an octopus it becomes your friend. Mr Henderson was measuring timbers when a large tentacle grabbed his steel tape measure. He tried the tickle test, but the octopus made a grab for his watch. Mr Henderson wants to pass on the information that octopuses are the bowerbirds of the sea .... so avoid swimming wearing anything that glitters. (Australian 31 March 1976)

DYSBARIC OSTEONECROSIS

Dr Gordon Donnan

AETELOGY

It has been assumed that all types of decompression sickness are due to the formation of bubbles of nitrogen during decompression and that more efficient decompression would prevent this. These assumptions are being questioned and alternative theories of the pathogenesis of decompression sickness are being put forward, but at present no convincing and coherent explanation of all these phenomena of decompression sickness has been constructed. Effect on serum enzymes and coagulation factors may be significant.

The earliest radiographic features are areas of increased density adjacent to the articular surface and these result from new bone laid on dead trabeculae resulting in an overall increase in bone bulk. These develop about eight months to a year after initial exposure to a hyperbaric environment and may be seen within four months. These lesions may remain static or progress to a structural failure of the joint surface and then to secondary osteoarthritis. A lesion of the shaft causes no symptoms or disability.

The diagnosis of dysbaric osteonecrosis is not usually difficult but all other causes of aseptic necrosis of bone must be kept in mind. The most important ones to be excluded are those following fracture of the femoral neck or dislocation of the hip, the idiopathic form sometimes associated with large doses of steriods, the haemoglobinopathies, Gaucher’s dieases and Schandler’s disease.

Revascularization of both medullary and juzaarticular lesions may begin but halt short of completion, the revascularization front becoming collagenous. Bone trabeculae adjacent to this fibrous tissue are often greatly thickened and may give rise to a sclerotic line on clinical radiographs. When such a radio-dense line is seen traversing a bone and it is highly probable that the tissue between it and the joint surface is often followed by formation of osteophytes at the living joint margins. At first the joint space remains normal and the articular cartilage covering dead bone is relatively well preserved but later it and underlying dead bone may be ground away, the end result sometimes being difficult to distinguish from primary osteoarthritis. A similar pattern of events and morphological changes may be seen following juxaarticular bone necrosis due to other causes.

REFERENCE: *J. Clin. Path.* 25: 1004-1006

A RAN Diving Achievenment

A team of nine divers from HMAS Curlew recently took it in 45 minute duties to march back and forth along a 200m length of rope underwater off Balmoral Beach, Sydney. They required 9 kg lead shoes and 13 kg lead weight belts so were, as a spokesman said, pretty tired when they finished. The aim had been to achieve 40 km in 32 hours but in fact they did 72 km. As the team supervisor said afterwards, “We are very pleased with the distance. Civilians would find that very hard to beat.”

Some people hope that nobody is going to try.

Investigation during and after construction of the Type Road Tunnel (1962-66) Report of Decompression Sickness Panel, Medical Research Council (British Journal of Industrial Medicine 28: 1-21, 1971)

INCIDENCE

15/124 developed definite lesions
7/124 suspected lesions
10 had 1 definite lesion
1 had six lesions
4 required operative treatment
First definite lesion nine months after starting work in compressed air
Not earlier than 4 months
Definite or suspected lesions:

LOCATION

Upper end of femur	14
Lower end of femur	30
Both ends involved	3

Since the Clyde Tunnel experience of 1963, all compressed air workers in the United Kingdom have had a radiographic skeletal survey and the MRC Decompression Sickness Registry has examined 1694 men showing an incidence of 19.7% with definite osteonecrosis and 11% with a juxta-articular lesion.

RADIOLOGICAL REQUIREMENT OF THE VICTORIAN MINING ACT

All persons working in or entering compressed air shall have an X-ray of the chest at intervals of not more than 12 months.

All persons working in or entering compressed air where the gauge pressure is over 13 psi shall have an X-ray of the heads of the long bones to detect bone necrosis not later than six months after first commencing work in or entering such compressed air and at intervals of not more than six months thereafter.

SKELETAL SURVEYS PERFORMED FOR THE BOARD OF WORKS

First record of Skeletal Surveys, January, 1969.

Number of examinations up to and including 23 July 1975 = 608

(This is the number of patients not all of whom were subsequently employed in compressed air)

X-rayed 6 or 12 monthly depending on the pressure - 28 ppsi lowest and 38 ppsi upper limit to date.

Full size chest X-ray initially, Micro each 12 months.

One case of bone necrosis. Many disc degenerations and spondylolisthesis. Workers are no longer excluded because of bond islands at initial examination.

History of patient

(whose films were demonstrated by courtesy of Mr Leo Lenaghan)

6 October 1970	No previous experience. X-rays NAD.
16 April 1971	860 hours at 14 psi
24 February 1972	1150 hours at 29 psi
29 January 1974	851 hours at 38 psi. No symptoms.
23 May 1974	Evidence of aseptic necrosis. X-ray appearances unaltered but has pain. Continued employment out of pressure. Ultimately total hip replacement. Films demonstrated

The patient had been a professional diver for 30 years. His usual depth of operation was about 50 feet below the surface. However, whilst he was working in the Eildon Dam in 1953 and at a depth of 200 feet the hose broke and he had to surface quickly. He then went down again to a depth of 200 feet and said that after this episode he suffered from an attack of the bends. He said that after this episode he suffered from an attack of the bends. He said that he was semi-conscious for a period of four to five days. He had cramping pain in his arms and legs. After this episode in 1953 he had no special treatment, just went home and was off work for some weeks. He said that he had had no trouble with his hips and shoulders prior to this incident but after this incident in 1953, he used to get some pain in the shoulders and in the legs and, in particular, in the right leg. He said his right leg used to go weak on him.

He then described another episode of bends in 1970. At this time he was working on the sunken ferry, the “Wahini” and it was at that time that he was taken to Auckland to the naval establishment and was recompressed in a chamber for a period of sixteen hours. He was then flown back to his previous place of work and continued on working.

The patient is now aged 57 years and is working as a taxitruck driver.

Clinically, the patient is a person of slender build and is rather tough and wirey. He had a considerable reduction of movement in both shoulders and both hips and pain in all four joints. He also had a considerable amount of coxa vara deformity in his right hip. He also had some arthritis in both knees but only of a minor nature. I could not find any evidence of arthritis in other joints.

BOUNCE DIVING IN 450 - 600 feet WATER DEPTHS AND DEEPER
Donald M Taylor, Editor

Recently, four divers in a hyperbaric chamber at Duke University made a simulated dive to 1,000 feet in only 33 minutes. The men were breathing a new mixture of helium, oxygen and nitrogen. They arrived at the simu-depth with none of the usual losses of mental or physical capacity that afflicts divers breathing the traditional helium-oxygen mixture. They returned to the simulated surface pressure in 96 hours.

By the way of comparison, the normal time for compression in 1,000 feet is 24 hours; and the decompression period now used by the US Navy for a similar dive is 11 days. This amounts to a reduction of 7 days in total elapsed diving time. At lesser depths, the percentage reduction in time runs even higher.

What will this mean to the offshore oil industry? It could mean plenty. Officials of Oceaneering International, Inc., one of the participants in the Duke experiments, say the new technique could extend non-saturated diving beyond its present depth limit of 600 feet to as much as 1,000 feet. And this could reduce diving costs by almost $300,000 per year for rigs operating within this range. The basis for this reduction in costs can be found in current diving practices. To begin with, the time required on bottom for actual work is usually quite short. “An analysis of 4,000 drilling rig dives showed the average time spent on bottom was only 17 minutes,” says D Michael Hughes, Oceaneering’s chairman. “The expense of diving then, is almost entirely a function of the time, effort and materials expended in going to and from bottom. This is why non-saturated or bounce dives hold such promise in the 400 to 600 foot depths.”

The time required for a bounce dive may run as little as 10% of that for a saturated dive. In the bounce dive, the diver is compressed to bottom pressures in a matter of minutes, quickly does his work, and usually starts decompression within the first hour. Because of the short exposure to high pressures his body does not become saturated with the breathing gas, and the period of decompression runs only a fraction of the time that would have been required had he stayed longer under high pressure. Why then, aren’t all dives bounce dives? Hughes, a tall affable man in his mid-30s who started as a diver in the Gulf of Mexico, explains, “Primarily, it’s because we lack precise knowledge about short-duration diving beyond 400 feet. Although a large number of short duration dives have been made in the 500 to 600 feet range, most of them produced an unacceptable incidence of bends. The ones which were not successful pointed up a need for further work if these dives are to become a commercial service.”

One of the most difficult problems starts with the compression cycle. In depths below 430 feet, high speed compression in the helium-oxygen mixture can produce high pressure nervous syndrome (HPNS) which is characterized by nausea, dizziness and tremors. The symptoms become more severe with increasing depth, eventually resulting in somnolence or convulsions. Even if the diver’s mind is clear, he may be physically incapable of working or even of saving himself.

To Dr Peter B Bennett, Professor of Anesthesiology at Duke University Medical Center, this represented a challenge. Animal experiments had previously indicated that the effects of HPNS could be negated by adding a certain amount of anaesthetic or narcotic gas such as nitrogen to the helium-oxygen diving mixture. Experiments had also shown that the effects of nitrogen narcosis, which would then result, could be relieved with increasing pressure.

In August, Bennett, who is an international authority on the physiology of diving, began a series of simulated human dives designed to see whether just the right balance

could be found between helium and nitrogen so that narcosis and HPNS might both be negated. Four divers participated in the dives, three from Harbor Branch Foundation and one, Erik Geerts, from Oceaneering.

Surprisingly, success came early. During a dive to 720 feet in August, using the three gas mixture, the four divers showed no signs of HPNS but did complain of slight narcosis.

In the 1,000 foot dives that followed later in the year, the nitrogen content was decreased. "This greatly reduced the narcosis they had experienced without causing any of the symptoms of HPNS to come back," Bennett said. "The divers had no tremors, giddiness or sickness and felt no pain in their joints."

Advantages of New Mixture

Everybody involved considered the experiments a nearly perfect success. Lad Handelman, Oceaneering's president was particularly happy with the possible time saving because his company has a contract to provide diving services in water depths to 1,000 feet. If divers can be compressed to 1,000 feet in 20 to 30 minutes, then spend 20 minutes working on bottom, they can be decompressed in a fraction of the 11 days normally required. Because divers would not be tied up for such long periods in the decompression chambers, the size of the diving crews could be reduced by, say, 25%. This is a much needed saving which can be passed along to the customer.

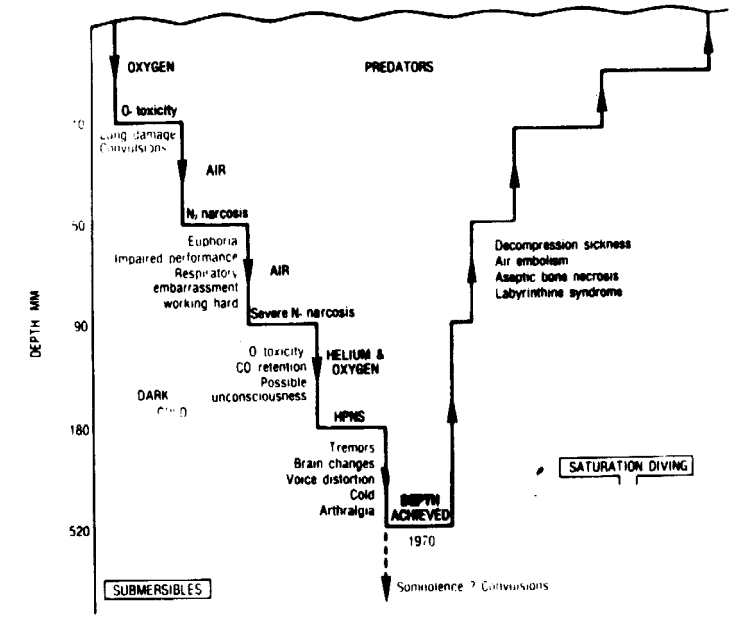
Another saving comes from the speed of response to emergencies. "It costs $40,000 to $50,000 a day for a drilling rig to operate in the North Sea," Handelman said, "If it typically takes a diver 24 hours to compress to 1,000 it, that's a waste of a day right there."

Oceaneering believes the new technique can lead to still another saving. A non saturated dive to 600 feet would require only half the charging gas and no replenishment gas during decompression. A 60% reduction in consumption of expensive gases is possible using bounce diving rather than saturation dive technique. At two dives per month, this new technique could effect a major yearly saving.

The divers, too, prefer the non-saturated for obvious reasons. Following a bounce to 600 feet, the diver will need only about 15 hours in the decompression chamber. A saturated dive at the same depth will require a stay of about 6 days. And this brings up another factor of keen interest to Mike Hughes. That is the matter of diver safety. "Long periods under pressure increase the diver's exposure to possible rig catastrophes such as fire or blowout," says Hughes. "Can you imagine the feelings of a diver if a fire breaks out when he has eight more days to go in the decompression chamber!"

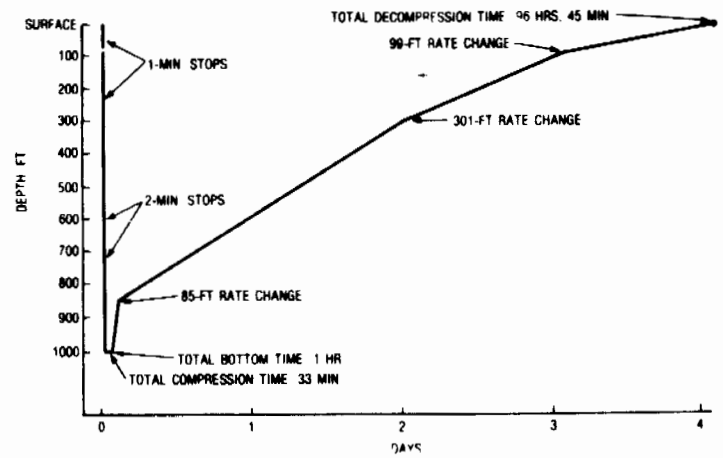
The new diving mixture takes on still greater importance in light of the oil industry's expansion into deeper water. As Dr Bennett put it, "One thousand feet has been the limit of man's working capacity in water, but even this has not been practical commercially because the divers had to go down and come back so slowly they lost much of their functional ability at that depth."

But this depth limit has been set by the combined effects of helium and pressure, he says, and no one yet knows how far man can dive with the helium/pressure effect eliminated by the use of the three gas mixture. It could be considerably deeper. More research is needed to complete studies of rapid compression before this technique can be considered safe for field use, but the potential savings could be tremendous.



Physiologic

Physiological problems of diving.



Graph of 1,000-ft dive showing rapid compression and decompression.

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Provisional Report on Diving Related Deaths in 1975 (Stickybeak Project)
Dr Douglas Walker

Overview

Ten fatalities have been identified as having occurred in Australian waters during 1975. There were two (2) breath-hold divers, three (3) Scuba divers and five (5) hose supplied divers. All the hose-supply divers were Professional in the sense that they were so diving for their employment: there is no evidence that any had received training, though special instruction was presumably given to Case H 5/75 before his employment. His death, from a heart attack at the early age of 34, was not foretold by the full "Diving Medical" given about eleven months previously. It could well have been fatal to him even had the illness occurred at the surface. On the admittedly incomplete evidence available it would appear that in all cases the victim was alone, though that was not the critical factor. The causes included a shark attack, drink competition spearfishing, total inexperience with scuba, failure to heed warnings of danger, ill health and inappropriate reaction to an out-of-air situation. When consideration is given to the fact that this total of deaths covers the whole of Australia and the environment underwater is unsupporting of life, the extreme safety of diving is worthy of comment.

Case Reports

Case BH 1/75. This was the only fresh water fatality. The victim, aged 30, borrowed a speargun from a neighbour and went to a nearby river with his wife and one of her friends. It was late afternoon before he started to dive at a spot well known to him. This gave time for the party to refresh themselves, his consumption being 10-12 stubbies of beer. This explains his later comment "I'm too full, I can't hit anything". After a while he placed the speargun and spear shaft back on the river bank and announced his intention to make one final dive in order to retrieve the lost spear head. His failure to emerge from the water caused alarm and several swimmers tried to find him. The Police were called and one of them dived to investigate. The victim's arm was felt within a hole beneath the bank.

This hole, about 4 ft 6 ins below the surface, was known to the victim. Its entrance was about 2 ft 6 ins diameter and the cavity extended beneath the roots of a tree for about 6 ft. The policeman correctly avoided entering the cavity himself, pulling the body out without undue difficulty. It cannot now be known whether he had entered in search of the spearhead or through error.

Case BH 2/75. The Inquest report is not yet available but witness reports have been used as basis of the comments that follow. The victim was aged 29 and presumably an experienced spearfisherman for this was a Championship Competition. The chief witness was on shore patrol with a walkie talkie and the other witness was a competitor at that time also ashore and talking to him. The latter commented that a diver, to whom was attached a float and diving flag by regulation line, seemed to have surfaced and indicated need for assistance by waving his arms. The witnesses considered the situation for a time but decided that no action was required. He was about 50 yards from the nearest other diver and 30 to 40 yards from the beach. A third person remarked that the swimmer was no longer visible so the witnesses decided to swim out to offer assistance if this was required. The first witness reached the float and pulled on the line, thereby discovering that the diver was lying unconscious on the sea floor 10-15 feet below. There was no kelp or other possible cause for entanglement in the area. He pulled the victim to the surface and commenced to tow him back to the beach. The trailing line entangled one of his feet and he would have been in peril had not

another diver arrived and cut him free. EAR was commenced on the beach, but without success. The second witness had to delay his assistance because his bootees had to be removed before he could safely swim. The absence of efficient boat cover/buddy diver cover relegates the float to the function of a floating tombstone and in no way an aid to safety, save from boats.

Case SC 1/75. This 16 year old boy came down from the country and hired diving equipment. Neither he nor his companion, who also hired Scuba equipment, had any training or experience of scuba diving. While his companion sat on a rock, the victim entered the sea for his first dive. The newspaper report states that he dived once to 40 feet, surfaced, dived again. His body was not recovered till next day. Comment is superfluous.

Case SC 2/75. At 18 1/2 he was an enthusiastic and frequent scuba diver, with 3-4 years experience of diving with others. He and a non-diving colleague were sent to a coastal town in connection with their regular employment, so he naturally took his diving kit with him. After a day-long journey they arrived at their destination, going straight to their hotel for a four course meal. Following this the victim dressed in his wetsuit etc and the two youths made their way to the harbour. It was now night. He had only to don his tank and he was ready to dive, his first solo and possibly first night dive (the latter is conjecture). He entered the water about half way along a wharf but shortly after this returned to the wharf to find his companion again. He spoke to a passing sailor to ascertain the likelihood of a moored ore ship starting its engines. The reply was presumably satisfactory because he entered the water beside the ship with the apparent intent of viewing its hull. His prolonged failure to surface from this dive caused his companion to raise the alarm (after 90 minutes). The initial police search was unsuccessful, but the body was washed ashore eleven days later. Most of the equipment had become detached and lost during the interval so could not checked. He was in good health.

Case SC 3/75. This was a planned Club Dive under ideal weather conditions on an old and well known wreck lying in 50 feet of clear water. There were 10 actual divers on the hired boat, some non-divers and the experienced skipper. The divers were paired and their names and water entry/exit times noted on a log by a non diving member deputed to the task. Such was the excellence of the visibility and the limited area of the wreck that no true buddy-diving procedures were thought necessary or followed. As everyone could see everyone else such caution was deemed superfluous. This is common diving procedure one may think. There were many artifacts beneath the sand, hidden in lumps of pitch-like material. Their collection was the object of each diver, and all set-to with a will.

It is thought that one of the divers was using hookah, the remainder scuba. One witness described how he was approached by another diver, later thought to have been the deceased, for help in opening a lump of material. Shortly afterwards the witness made a hurried solo ascent, reaching the dive boat in an exhausted condition. It was this diver's "buddy" who was shortly afterwards approached by the victim, who was making signs to indicate that he was short of air and wished to buddy-breath.

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The victim was doubtless confident of his skill in this matter because he had acted as the “patient” in a recent Scuba Safety Skills competition which involved buddy breathing and rescues. The donor diver was wearing a borrowed set, a twin hose unit, the absence of purge valve making sharing more difficult than would otherwise be the case. After some 5 minutes (?) of sharing, ascent was attempted . The donor forebore to inflate the victim’s buoyancy vest or drop his weight belt lest panic be caused, but he indicated the need to inflate the vest and to drop the trophy bag. It was found impossible to ascend more than 10-15 ft from the sea floor, which could be explained by the fact that the bag was found to contain 45 lbs of souvenirs. When the donor’s air supply began to fail the victim became unwilling to relinquish the mouthpiece so the donor decided to make a free ascent. He dropped his own weight belt and was preparing to ditch his tank also when he found that he was floating free. He surfaced exhausted, and held onto the anchor chain until the skipper reached him in the dinghy. He was then able to give the alarm about the diver below without air. Before any real response could result from this information another diver surfaced with the victim’s body. This was rapidly taken into the boat and EAR started. The rescuer had seen the deceased lying on the sea floor, his mouth-piece floating free between his legs, the cord of the trophy bag lightly entangled in it. The buoyancy vest may have been inflated at this time. The weight belt and tank harness quick-releases worked faultlessly and the body, free from the bag, ascended easily. The victim was aged 19. He had been diving with the club monthly for 2 years and had a “C” card certification. He had dived on this wreck on several previous occasions. This tragedy quite possibly illustrates the “Tunnel vision of Thought” that can afflict and blinker anyone under stress conditions. He was skilled above the average in buddy breathing but could not use the time gained to plan the necessary ditching of his souvenirs and weight belt. His skill merely postponed his drowning. A tank contents gauge, if consulted, would have saved him, as also would an awareness that one should never dilly dally when air is running low, for the only place there’s plenty more of the stuff is at the surface.

Case H 1/75. This 40 year old diver made his living diving for scallops from his boat. It is not known what knowledge or training he had or for how long he had practiced his craft alone, leaving the compressor working in the boat while he used the hookah supply below. One day he failed to return as expected and a search was made. The boat was found with the compressor stopped from fuel exhaustion. The hookah airline was over the side, leading to the weight belt and attached demand valve assembly. There was no trace of the diver himself. The body was found floating the next day. The autopsy showed severe pulmonary barotrauma and then drowned. He was still wearing his fins and facemask when recovered. The reason for his making a rapid ascent from his 50 ft dive cannot ever be known, but several suggestions have been offered. There was the possibility that he saw a shark (there were said to have been some in the area in previous days) or become alarmed through interruption of his air supply. This could have occurred through the compressor running out of fuel, for the air reserve tank was not connected and the engine was disadvantaged by a modification of the exhaust outlet which produced increased back pressure. The correct modification would have been to raise the air inlet rather than tamper with the exhaust. The hose was kinkable but readily resumed function when tension was released. The quick release of the hose were too readily activated but in fact were not at fault. The weight belt had so many lead weights in it that the quick release was very easily worked; this could have led to the loss of weight belt with the attached air hose and demand valve without the diver expecting any trouble. There was no non-return valve on the hose to protect the diver, but this fault also was not a present factor.

Case H 2/75. This abalone diver was apparently attacked and totally destroyed by a shark, supposedly a white pointer, in 7-8 fathoms of water, 20 yards off shore. His tender was left with one glove, a glimpse of a fin, an area of bloodstained water and a hookah line attached to a backpack with its thick rubber attachments torn. No shark was caught but there had been seals in the area and the suggestion was made that the diver was mistaken for one of them by the shark. He was 37 years old.

Case H 3/75. No inquest report is yet available concerning the death of this 19 year old abalone diver. It is said that evidence of pro-existing disease was noted at the autopsy but no other information is available.

Case H 4/75. The last words of this diver were "She'll be right!", but such was not to be the case. At 36 he had been earning his living by diving for 18 years and had experienced a wide variety of jobs. He had also suffered at least two serious episodes of decompression sickness and been warned not to dive deeper than 30 feet in future. This instruction he observed, not increasing his range to 60 feet till he had a Diving Medical in August of the last year. The limitation was of a prophylactic nature, based on discussion of his diving methods vis a vis diving tables. He regarded himself as a careful diver, very safety conscious.

He became involved in the task of entering the part flooded ballast tank to free a valve when the tanker's diving contractor asked him and another diver if they were available for the job. The contractor ran his air compressor from a boat alongside the ship and the divers were hose supplied with their air. They had the choice of using their own masks or those provided, choosing to use their own. The victim had a new mask but it was found later that he had used an old one. The tank held several feet of water above the valve and there was a high concentration of petrol vapour above this. The task and risks were explained to both divers before the second diver entered the tank and undertook the task. However the valve remained closed because it required more turns to open than was often the case. While he retired to shower off the petrol that was causing skin irritation the victim prepared to enter the tank. He entered the tank holding his mask in one hand and proceeded with his descent despite the warning shouts of the others present warning him of the dangerous fumes. It was only when he began to cough that he put on his mask. Despite further coughing he completed his descent and entered the water, again refusing to return to the fresh air on deck. It was soon observed that he was in trouble, holding onto the valve stem for support. The other diver was summoned and immediately started to the rescue, minus wet suit but using the second hose supply mask. The rescue attempt failed because this diver himself collapsed shortly after reaching hold of his colleague's shoulder and weight belt. At this stage the ship's emergency breathing apparatus was used by the First Officer and both divers were removed from the hold by rope. This was a gallant action by the First Officer, and the crew also worked efficiently in the rescue and the resuscitation attempts. The second diver recovered, the victim did not.

Investigation revealed that the personal masks of both these casual-contract divers were old and ill fitting. The victim's mask leaked when used in water, requiring frequent clearing, because the feathered edge of the mask had become worn and been cut away. The masks were tested in a gas chamber and both allowed the test tear gas to enter. The second diver's mask had an additional reason for ill fitting, for he had several days beard growth present. His collapse may have been due to the excitement and rush of the descent into the tank to his colleague, plus the petrol vapour entering the mask. These masks are demand supply, not free flow, so vapour within the mask is not immediately flushed away. It is obvious that the increased necessity for a perfect air seal of the mask in a gaseous environment occurred to

neither diver, accustomed as they were to regarding themselves as “divers” rather than “users of hose supplied air” in this particular job. A semantic error with fatal consequences.

Case H 5/75. This appears to have been a truly “unavoidable” death. The diver was aged 34, working from a bell (SDC) at 240 ft using a Helium mix. He had a hot water supplied wet suit and good communications with the surface diving controller. He had passed a “Diving Medical” examination in the early part of the year. The task was heavy, requiring him to pass a somewhat inflexible cable twice round a broken pipe. Until the incident occurred the monitoring of respiration revealed nil unusual. His failure to respond to orders and a change to a laboured type of breathing caused the surface control to request the diver/tender in the SDC to investigate and report what he found. The victim was found lying on his back on the seafloor, unconscious. The tender dragged the victim back to the position of the SDC, which was then lowered to 5 feet from the sea bed. A block and tackle were attached in the SDC for such emergencies and the hook was now connected to the victim’s lifting harness. The tender had not only the exertion of pulling the victim but had needed to disengage the trailing umbilical from some debris. The victim could only be raised head and shoulders into the air space of the SDC: the mask and equipment were there removed. The victim was no longer breathing at this time. As the lower hatch could no be closed the rate of ascent was according to the USN decompression schedule, halting at 120 feet to allow a standby diver to enter to assist the diver/tender already present. Together they pulled the victim fully into the SDC and closed the lower hatch, EAR and ECC being used although they believed that death had occurred. The victim’s umbilical had been deliberately severed after a few feet of ascent as it had again snagged on debris but later testing revealed no failure of the hose or communication links. After the SDC reached the deck the divers were brought to “40 feet” and then straight to surface pressure and the SDC opened. They then proceeded to the main decompression chamber (DDC) which was about 60 feet distant. This chamber has a lock for entry but no facility to mate the SDC to it directly. The tender suffered mild “bends” pains in the legs and arms of onset before leaving the SDC on deck. The DDC was taken to 70 feet, EAR and ECC being continued until a doctor arrived and certified that death had indeed occurred. The police were notified of the fatality and investigations started. The autopsy revealed that death was due to Ischaemic Heart Disease. No evidence was given of ill health preceding the fatal incident. There was no equipment malfunction and nothing to suggest that anything further could have been done to improve survival chance. There are two points of additional note. When the diver/tender was notified that he had to don a mask and leave the SDC he felt dizzy and part fainted. This he later ascribed to the sudden apprehension at realising that he would be sharing the same hose supply gas as was being supplied to the victim so any mistake in its composition would effect him similarly. A moment’s use of the emergency bib mask and a few words with the surface very rapidly revived him and he made a good job of recovering the victim. His report of the effect of psychological factors is of great help. The other matter was the mention that the SDC and the DDC could not “mate”, a potential risk factor of importance. Sixty feet separation between them was quoted.

Comment

These cases are few compared to the large number of divers at risk, but any avoidable factors are worth consideration. Undoubtedly many “near misses” have occurred. The reports are made on the basis of information at present available. This is always incomplete, sometimes grossly so. It is hoped that readers will recognise the value of considering these tragedies as a mirror to common diving practices and so improve safety to even higher levels. It is hoped also that they will recognise that without

more full and truthful reports of incidents of all types this investigation cannot achieve much. All reports are treated as confidential, all the information in this report being available from open sources with a little trouble. The confidential reports assist in better understanding the underlying factors that influence the outcome of incidents. It is noted that the holding of an inquest often clarifies matters written in depositions. The practice of not holding an Inquest when the fatality seems "a natural death" is correct but regretted because many matters effecting the understanding of why the outcome was fatal remain for ever unresolved. The function of the Coroner as an important link in the prophylaxis of accidental death is worthy of further emphasis. In considering these cases further one can divide them into breath-hold, scuba, hookah and deep diving. The ignorant solo diver will always be a problem and well represented in any accident survey. Sudden illness cannot be predicted but the availability of assistance may critically effect its outcome. Those who organise breath-hold spearfishing competitions should be aware that the competitors are at greater risk of hyperventilation blackout than are the ordinary sport spearfishers. It should be apparent by now that a one-for-one check on competitors is the only effective safety mode, a surface cover requirement that should be obligatory however difficult this may be to apply. Competition deaths are NOT to be regarded as "unavoidable accidents".

The Club dive (Case SC 3/75) brings out two points. First, it is not sufficient to go through the motions of safe diving, one must actually dive safely. The buddy procedure was only given lip service, as is probably a very common matter in diving everywhere. The lack of knowledge of one's air reserve status is a negative safety factor, at least two of the divers on this dive running short of air. The second point is of wider significance, concerning the very philosophy of Emergency Procedure. This diver had been involved in recent exposure to testing of divers in rescue and buddy breathing, yet he died. In the emergency situation he reacted in the wrong manner. An "overlearning" is required evidently so that one cannot help but react by a stop-think-ascend decision over-riding all other thoughts. Here the thought seized-up at the getting of air stage, forgetting the imperative need to surface. This is similar to a deep dive fatality in a previous report. The failure to drop the weight belt and trophy bag and inflate the buoyancy vest were aspects of this inappropriate response. Undoubtedly ANYONE could do the same under similar conditions. So please PLAN NOW for your next accident.

That commercial and amateur users of hookah are blasé should be no surprise. Until disaster strikes there is no skill required to use the apparatus. The resultant emergency ascent can be relied upon to cause the occasional fatality from pulmonary barotrauma. Those who sell such hookah apparatus should advise their clients to learn something about diving. It is remarkable, and a compliment to the equipment, that so few fatalities do occur.

Other points have been noted in the case reports. It is hoped that the statements of fact and opinion are both accurate and helpful. Further details on these and other cases are of interest and comments are welcome.

ACKNOWLEDGEMENTS

This report would not be possible without the reports supplied by many people. Of greatest importance has been the continued assistance of the Attorney General's and Justice Departents in all States. Their understanding and aid continues to be invaluable.

PROJECT Stickybeak

Further reports are always welcome and will always remain confidential as to source and victim. Cases are welcome whether serious or minor. Of the greatest interest are reports of instances where an Emergency Situation either occurred or seemed likely to occur. Comments and additional advice concerning cases in this or previous Provisional Reports are welcome.

Please write to: Dr DG Walker
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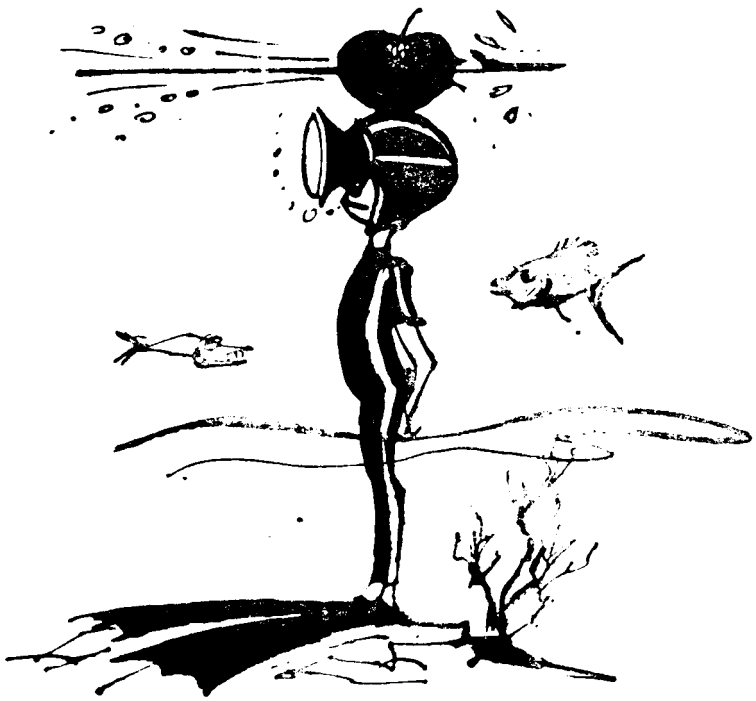

Rule 1 – Avoid dangerous situations!

Anxiety Induced Hyperventilation Danger to Divers

Boris Innocenti
(NAUI A-78)

Any condition which can render a diver unconscious or immobile should be of great concern to the diving public. These effects can result from hyperventilation. The production of unconsciousness following the voluntary hyperventilating practiced by breath-hold divers intending to increase their underwater breath-hold times is a problem well covered in texts on Underwater Physiology, but the result of involuntary hyperventilation due to anxiety, stress through apprehension or other strong stimulus, anticipation, excitement or other cause has not been documented well (if at all) relative to divers. This is despite its common occurrence on land. It is naive to assume that it neither does nor could occur in diving. This form of uncontrolled breathing may result in muscle cramps, aches, stiffness, convulsions, tingling of the feet, hands, mouth and tongue and carpal spasms. The changes in the blood chemistry and lung filling/buoyancy effects are outside the scope of this article.

If the victim is caught in time and can be brought to dry land or a boat and allowed to rest, the (tetany) symptoms will gradually subside and the person return to normal. This return to normal with regard to breathing and the other symptoms can be speeded by administering 5% Carbon Dioxide or forcing the victim to rebreathe expired air by breathing into and out of a paper bag for a short period of time.

If a diver undergoes such an occurrence, quite apart from the obvious danger to himself there is a severe scare to the diving buddy or instructor, they at once suspecting that air embolism or some other of the demons they were told about in scuba course lectures has struck. I'm sure that every diver has seen a relative novice diver suffer extreme anxiety at being presented with a new and less than satisfactory diving situation. This is the perfect set up for trouble if that diver is also prone to hyperventilate under stress conditions. The following incidents, seen through the eyes of a medical layman, will hopefully serve to bring out the significance of this reaction to stress. I have trained many thousands of students during my 20 years of teaching and have not lost any in a diving accident. In fact there have been so few "close shaves" that it is not too difficult to recall them. Until a few years ago all such close calls for which I could find no satisfactory answer were filed away mentally with the appropriate question marks. However, about that time a close relative started to hyperventilate during moments of stress and armed with this new experience and with a mental picture of the symptoms, etc of the "new affliction" of the divers, the many pieces of a puzzle began to fit together, albeit in a loose manner. I hesitate to class the following as Case Histories but they appear to be worth presenting as evidence for incriminating involuntary hyperventilation as a cause of diving accidents.

Case 1 A fellow scuba instructor was personally conducting several novices on a tour of a shelf 60-70 feet below the surface. One diver was not relaxed so the instructor came in close and decided to escort him to the surface. On approaching the diver he noticed that he was quite stiff. He grabbed him by the arm and proceeded to ascent. As he was not breathing the instructor forcibly pushed him in the abdomen, causing him to exhale. The ascent completed, after surfacing the diver admitted to near panic. The instructor felt that the victim was unconscious of events at the start of the ascent.

Case 2 Mr Jack Albert was accompanying a group of surgeons diving from the 85 footer "Cayman Diver" when one of the group surfaced, apparently in great distress. He stiffened up and passed out (though not necessarily in that order). His colleagues

felt that the gentleman had undergone a heart attack. Upon returning to New York the diver consulted a number of heart specialists. Results indicated no abnormalities. In the meantime he also got in touch with Mr Albert, who offhandedly mentioned my concern about apprehension and possible blackout, tetany, etc, from hyperventilation. He immediately felt that this could well have been the case with himself since he was extremely apprehensive prior to the dive. He further commented that failure to recognise the symptoms did not surprise him since he and his colleagues had been away from diagnostic medicine for many years.

It is surprising how frequently tetany and paralysis are confused by both lay and medical people, at least in the situations I have encountered. Conversations with a number of friends in the medical profession were the first to bear this out. The confusion seems to be not with the general practitioner or the internal medicine specialist but with highly specialised surgeons. My relative spent several hours on pure oxygen in a hospital emergency room because the only doctor present, a highly qualified surgeon, erroneously felt she was undergoing a heart attack. If this is a real situation, as I feel it is, perhaps it might be wise to educate scuba instructors and divers into recognising the symptoms of hyperventilation and the use of a paper bag or whatever to restore normal breathing.

Case 3 The diver surfaced next to his instructor and shouted “I’m paralysed! I can’t move, I can’t breath!” Since the boat was very close he was rapidly brought aboard, there to lie stiff and having a difficult time breathing. The instructor and other observers assumed all sorts of possible reasons for such behaviour, mostly associated with those “demons” of the diving medicine lectures. The first thought was to administer oxygen but fortunately it was late in coming and recovery was uneventful. The consensus of opinion is that the use of oxygen would prolong the trouble. This incident was followed up and it was found that the victim had had other bouts of hyperventilation associated with excitement and anticipation.

Case 4 A dive at a deeper than normal depth resulted in a witnessed anxiety with subsequent passing out of the diver. The witness, a doctor, was not certain whether or not exhalation bubbles were present during the ascent of the unconscious diver. Possibly laryngeal spasm had occurred, for lung damage and evidence of air embolism were found at the autopsy.

Case 5 This may throw may throw light on the preceeding case. The diver was exploring a depth not reached by him before. It was dark and barren. He was observed to become stiff and “frozen”. An instructor who happened to be present grabbed him and started to bring him up. As he remained stiff and neither breathed nor exhaled, the instructor had to squeeze him to ensure that he exhaled during ascent. It is possible that during such an anxiety period the throat may close through laryngeal spasm.

Case 6 This diver admitted later that she had felt apprehensive on this her first dive in the Catalina island kelp beds. She felt that something was wrong with her regulator as she was unable to satisfy her breathing demands. She therefore headed for the surface, inflated her vest, removed her mouthpiece and breathed directly through her mouth. But she found that she was still unable to satisfy her breathing demands. Her plight was recognised by the Dive Master, John Schultz (NASDS, PADI), who immediately jumped in and brought her to the boat. Upon reaching the boat breathing returned to normal. The regulator was checked and found to supply more than sufficient air for (normal) diving at that depth (25 feet.)

Case 7 This lady was hanging from the anchor line waiting for an escort for her snorkel swim back to the shore. She was very apprehensive, for it was her first time in the water. All those on the boat faced in the opposite direction for a minute: when

they next looked, she was gone. Several divers present jumped into the water and dived to searched for her but it was several minutes before she was located in about 30 feet of water with her weight belt still on. Several more minutes passed before mouth-to-mouth resuscitation could be administered. Despite the delay she responded successfully and showed no residual ill effects, although the doctor who treated her had feared that anoxic brain damage would occur. Her successful survival may be due to the increased partial pressure of the alveolar oxygen at 30 foot depth (plus the dive reflex). The victim did not remember passing out but admitted to anxiety preceeding the event. Hyperventilation is a possible cause of this incident.

There are numerous situations where difficulty of breathing has been reported to myself or my teaching staff. I have now incorporated a screening of anxious or apprehensive divers. On the medical questionnaire we inquire as to the existence of a history of hyperventilation and in the pool we screen for the presence of anxiety. Buddy breathing is a good test, a positive direct correlation existing between apprehension and reluctance to part with their regulator. Rate of breathing is another indicator used. Students who fall in this category are handled on an individual basis. If the problem seems permanent they are asked to give up. We also spend a lot of time in assuring our students that at no time will they be asked to make any radical entrance into this new environment. This has been found necessary since sensitive, intelligent people with active imagination anticipate a more radical experience on their first open water introduction. I feel the use of extensive snorkelling experience prior to scuba would lessen this problem considerably. The instructor who raises his or her entrance standards too high (ie. previous snorkelling experience) will merely lose the prospective student to another instructor who is not so strict. I feel that higher standards of prerequisites will almost certainly have to be enforced by the certifying agencies.

Several of the people in these incidents later proved to have had a bout with hyperventilation before. Most all had consumed a far greater amount of air than their buddy. Perhaps we should also look at that “air hog” in a different light.

It is interesting to speculate whether or not this tendency to involuntary hyperventilation under stress conditions might not exclude a person from taking up the sport of scuba diving.

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* This article first appeared, for a lay readership, in NAUI News June 1974. We are greatly indebted to both the Author and NAUI for the opportunity to republish it. It has been amended by the insertion of some additional case histories most kindly supplied by the author. This article not only draws our attention to a neglected facet of diving medicine but also demonstrates the valuable role that can be played by observant divers/instructors in the identification of problems otherwise overlooked.

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David Brown, the Queensland Gold Coast marine biologist and curator of mammals at Marineland there, is to open an underwater oceanarium in Tahiti. The project, the first of its kind in the world, is to be developed in a lagoon where people descend in mobile underwater observatories to spy on marine creatures. Already the observatory modules are being built in Sydney. “In Tahiti, instead of people going to see creatures in tanks, the people will be in the tanks”, says Mr Brown, who admits he “cherishes” such an idea.

The Australian, 5 May 1976

Case Report of an Untreated Type 1 Bend
John Knight

One Saturday two divers dived at Picininny Ponds. They went to about 110 feet and then looked about, going up and down a bit before starting for the surface. They did not go any deeper as the light was poor below where they were. They did the proper decompression stop by going into a cave at about the right depth for a while. Their dive lasted about 20 minutes. Four hours later they were at the Little Blue Lake and did a dive to 100 feet. Because it was a repetitive dive they added the two times together to obtain the correct decompression. The decompression stop was carried out using a depth gauge instead of a shot rope. They were in the water about 20 minutes. 15 minutes after leaving the water one of the divers developed a pain in his right elbow. At first he though that it could not be a bend as his buddy was quite unaffected. However when the pain had lasted three days he decided that it must be a bend and sought medical advice. Within 24 hours of talking on the telephone to a doctor his pain went and he did not keep his appointment. The history was obtained over the telephone.

He claimed to have done the proper decompression, but he was only sure of one time, the time to onset of his pain. Assuming that he was using the RAN tables published in CZ18, which he was not, his dive to 110 feet which required a decompression stop, must have been for a bottom time of more than 17 minutes. Adding this to a 4 minute dive to 100 feet on his second dive he should have had two stops on the way back to the surface. His second dive would count as 21 minutes at 110 feet, which if taken to the next higher time is 25 minutes and the stops for that are 5 minutes at 20 feet and 5 minutes at 10 feet. He was in fact using the tables printed on his Fenzy buoyancy vest. The tables are metric and the instructions are in French, which he could not read, but he “could understand them alright”.

As the dive was being limited by poor light he did not have the best of conditions for reading his unfamiliar tables. Kiessling and Maag showed in 1962 that there is approximately a 25% loss of performance in reasoning at 100 feet in a chamber. Davis, Baddeley and Hancock have shown that there is a marked performance decrement in mental arithmetic and other tasks with cold. The water in the Mount Gambier area is cold.

On the second dive the two divers were aware of the need for care and were trying to treat their dive as a “repetitive dive” according to the RAN tables. They were aware that the rule is to add the two times together and use the greater depth to obtain the decompression profile. However cold forced them to cut short their second dive and all the factors mentioned above were working even more against them on the second occasion. But one must not forget that bends do happen to people who have followed the tables precisely. This diver was probably not in that class, on his own admission he did not know the depth of the stops nor the length of the dive or of the stops.

He was advised to plan his dives before leaving the surface when there is plenty of light and no narcosis or cold to complicate mental arithmetic. He was advised to dive within the no-decompression limits and that if he was going to do a decompression dive he should work out his bottom time and stops before leaving the surface and write down his available bottom time and the depths and times of the corresponding stops on a piece of plastic with a waterproof pencil in letters large enough to read easily in poor light. Then when diving he would not have to do more than start for the surface at a known time.

Furthermore he should use a shot rope for decompression and have spare air for himself and his buddy on the shot rope in case he used more air than he expected at depth.

He volunteered that he and his buddy had dived a number of times at Mount Gambier and had been letting their diving standards drop and were not as careful on this occasion as they had been in the past.

References

1. Davis FM, Baddeley AD, and Hancock TR. Diver Performances: the effect of cold. *Undersea Biomedical Research.* 1975; 2: 195-214.

2. Kiessling RJ, and Maag CH. Performance impairment as a function of nitrogen narcosis. *Journal of Applied Psychology.* 1962; 46: 91-95.

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Elixir of Youth

A certain hyperbaric "unit" talked freely to the local Press recently. In an unguarded moment someone mentioned that HPO had a rejuvenating effect, bringing people back supposedly from the limbo of senility with sharpened wits. The therapeutic use of the Unit was mentioned, in passing, to also include carbon monoxide poisoning, gas gangrene and non-healing ulcers. A few days later the Director was reported as requesting a cessation of the requests for such treatment of senility as it was not being practiced there. A further few days passed and an even smaller notice appeared, a statement by the reporter that he didn't edit the article. Visitors are now advised not to mention their contacts in the newspaper industry when asking for favours!

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Thor Heyerdahl, the Norwegian scientist and explorer who crossed two oceans in primitive craft, says that insecticides and detergents are debilitating the seas. Heyerdahl was the main speaker at a weekend program in honour of the visit of King Olav V of Norway to Minneapolis, USA. He said future generations could be threatened with a shortage of fish and eventually oxygen when chemicals destroy vital ocean plant life.

Australian, 13 November 1975

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David Niven recently saw the film JAWS. The next morning while swimming in the motel pool he spotted a black form lurking under the water. "I was walking on the water to get out. Then I found it was a maintenance man in a scuba suit fixing the drain", he said.

Australian, 16 March 1976