South pacific underwater medicine society

C O N T E N T S

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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.

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Notes to Correspondents and Authors

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OFFICE BEARERS

Editorial

The range and variety of articles included in this and previous newsletters should go far towards demonstrating to even the most complacent or cynical of divers that our upstart speciality of Diving Medicine is not only of use when diver damage occurs but is the only source of expertise towards understanding the physiology and pathology of those who venture underwater. The merely mechanical problems can be solved by any competent engineer once the protocols have been stated, but the highly complex system of checks and balances in the body is still little understood. It should be apparent that a purely mechanistic approach is not only too simplistic but downright dangerous. The occurrence of "rogue bends" in association with strict use of non-decompression dive schedules demonstrates this fact. For far too long the generality of divers have worked empirically, equating survival with correctness of procedure, while the majority of experts gave blind belief to theory as written, regarding what they witnessed as evidence of diving technique failure always and never as indicative of imperfection in their understanding of matters. The mind-blocks that hamstring Scientific progress are as well developed in our infant speciality as in the most ancient of Royal Colleges. Anyone who reads the DCIEM 1973 report on decompression sickness will suffer an acute case of deja vu if they have previously read the brief review of the subject by Dr Charles Shilling (USN Med Bull 39.367376, 1941). Therein he noted the opinions of himself and others that DS was much more than a simple bubble effect, that helium could be used by divers, that bone damage occurred and that oxygen therapy was highly useful. The commonly used texts for non-specialist diving physiologists still, over 30 years later, keep to the simple old time faith of the founding fathers of diving of discrete recompressible bubbles as explaining decompression sickness. While the finer details of the charges are of no importance to the practicing diver there are serious and observable ill effects flowing from the belief that if one cheats a little and gets a bubble trouble the complete and simple answer is to squash it flat with a quick blast of pressure. Would that it were so easy. This leads to the well known demand by abalone divers, for example, for bigger and better recompression chambers rather than an educated approach to diving prophylactic procedures. Surely there has been enough talk now about "bone rot" to persuade divers that "Doc may know best", at any rate in this matter. But in truth our understanding is still incomplete.

Progress in the refinement of knowledge can only be firmly based if a sufficient fund of accurate information is available. It is also a great help to have a few clues as to the problem that one is seeking to remedy. For such reasons the newsletter welcomes papers on observations of a general (surveys) or specific (case notes) type. The article by Crockford and Dyer indicates that the small number of deaths associated with professional divers may conceal an unacceptably high mortality rate. It is to the shame of all parties involved in the North Sea oil rig diving activities that the mortality among divers, guessed by some to run at a yearly rate of 1% of rig divers, was ignored until a newspaper started to run the story. Then everyone started to get in on the act. It is hoped that the newfound though belated interest in diver training and diving safety procedures will be maintained and spread world wide. It is hoped, though this is less likely to occur, that co-operation will evolve and information sharing become a reality. There is a real danger of "Empire building" producing frictions between the different groups involved in diving safety in the North Sea area already. A paper dealing with the need for an Undersea Life Support Specialist, by WJ O'Neill of Westinghouse Electric Corporation will appear in the next issue. Such people would in the future hold the balance between the many forces effecting diving decisions and could become a source of vital information. But until such information sources evolve there will be great dependence on clinical reports such as we have Drs Cave and Hattori. The present day hoarding of clinical information

about diving incidents is a crime insomuch as medical opinions at present lack sustained assessment against results. The Editor will be pleased to receive all offered information under seal of continued confidentiality should readers not wish to write up details themselves for publication. The clinical observations by divers of vertigo and post dive symptoms will be welcome. It is the Editor's private opinion that the occurrence of small air emboli is far from rare but because many think that a cerebral air embolus is necessarily fatal the non fatal cases are not correctly ascribed. This is combined with a disinterest in reporting cases where cure occurs without the doctor's assistance.

Readers will be pleased to have the opportunity of reading another chapter of the book by Edmonds, Lowry and Pennefather. They continue their effort to educate us either slowly through these columns or more rapidly (their preference!) should we buy and read their book.

Once more we are much indebted to the British Sub-Aqua Club and the individual experts who spoke to the OCEANS 2000 meeting. There is much to stimulate thought in what they have to say. Some final papers will be printed in the next newsletter dealing with the cost efficiency of diving modes and other problems. The Crystal Ball approach to advances in diving involves a return to basic principles if any degree of accuracy is to be expected. No more distinguished experts than those whose papers are here printed could be imagined. We thank them for allowing this use of their papers.

One sign of the questioning of diving shibboleths is the suggestion that the very tables of depth/time for divers are therapeutic rather than prophylactic regarding decompression sickness. This has very important implications in the field of the prevention of dysbaric osteonecrosis. It could mean a rush back to the computers by the back-room boys if the Duke University workers are correct. The modern ability to monitor asymptomatic bubbles will be very useful in the investigation of the safety of diving tables and the article by Dr Mecklenburg helps to understand what is involved.

The report on the AGM is not available but it is known that the members there present declined the opportunity offered by the New Zealand members to do some practical hypothermia research, suggesting in their turn a return to the Isle of Pines. The recent meeting at Melbourne is reported briefly. Two papers given there are being made ready for later publication in this newsletter.

Yes, you are quite right. The Editor has given up the unequal struggle to produce four issues of the newsletter this year. This issue is extra large as some sort of recompense for the shortfall. It would be easier to publish regularly if members contributed in larger numbers. Sincere thanks are offered to all contributors, those who choose to remain anonymous included. To the others, a suggestion that your New Year Resolution is to produce a contribution and so join the ranks of the blest. It really is amazing how many things can be legitimately included as relating to diving medicine. Perhaps it is the most all-embracing of Specialities.

Safe diving ... or write it up!

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DON’T FORGET THE SHARK-O-GUARD, BEN!

There is a large and eager public awaiting anyone who can produce a fully guaranteed, sure-fire, all-sharks repellent. The most practiced, though still unintentional, deterrent appears to be the screen of pollution that exists off-shore near human habitations, large conurbations naturally being at a considerable advantage over resort islands in this matter. Unfortunately there may exist currents and gulleys that allow sharks to come close to the shore, there to produce alarm through their presence regardless of their actual aggressive activities. Naturally! So further protection is desired, and even more so is it necessary for plane or shipwreck victims. An artificial pollution has been tried by means of chemicals, dyes combined with copper compounds becoming popular during World War II. Unfortunately when sharks get the feeding frenzy they ignore even their own partial destruction in their insensate compulsion to attack. Bubble walls, beside being ineffective for most sharks, are hardly practical on the open sea situation. Similarly electrical barriers are unworkable and the search for acoustic devices appears to have found how to attract them, not to repel!

On the premise that prevention is better than cure a promising line of investigation has been the development of a plastic bag so that the shark remains unstimulated, the potential victim’s many “It’s feeding time” chemical (and other) broadcast messages being retained within the bag he/she wears. Although Dr Walt Stark has shown that some sharks avoid a diver who wears a bright striped wet suit, at any rate near Lord Howe Island and off the Solomon Islands, others have found that sharks have good eyesight and are attracted by bright colours. As shark behaviour is not understood and they have labile temperament on occasion, caution is indicated before regarding even the most eminent of observers as a guide for oneself. The days when one was told to blow bubbles and all sharks would flee seem to have themselves flown. It is therefore of interest to note that one group of fish seem to bear a charmed life when sharks are around.

Pardachirus marmoratus, the Moses Sole of the Red Sea, owes its protection to a milky secretion from glands at the base of the dorsal and anal fin rays. The Australian Peacock sole apparently has a similar protection. Anything that can stop a shark from shutting its mouth must really pack quite a kick! So if anyone sees Ben Cropp rubbing a sole over his wet suit they will know they can safely bet on him in the greatest piece of showbiz piscatorial may-be of the coming year. Otherwise, in fair fight, poor Ben ...

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LIQUID BREATHING

Prof. Johannes A Kylstra
(Oceans 2000, 1973)

As some of you may recall, during the Second World Congress of Underwater Activities held in London in 1962, Jacques Cousteau startled his audience with a vision of the diver of the future - homo aquaticus - a human creature with liquid-filled lungs, breathing like a fish, with a surgically implanted gill. Homo aquaticus would be free to roam the oceans from the surface to great depths, protected against decompression sickness by an incompressible liquid in his lungs. As all of you know, one of the main hazards in diving is the presence of compressed air in the lungs, which prevents the chest from being crushed while, at the same time, sustaining life. The pressure causes gases to dissolve in the blood, with potentially serious consequences. At heightened concentrations in the blood and tissues, most gases are toxic: oxygen can cause lung damage and convulsions; nitrogen can produce a state of altered consciousness, and usually incapacitates a diver at a depth of 300ft or thereabouts. These complications can be avoided by using helium, which is not narcotic at high partial pressures, and by minimising the fractional concentration of oxygen in the inspired gas mixture so that the partial pressure of oxygen remains within safe limits.

However, regardless of the gas mixture used, the inert gas dissolves in the blood and tissues and, whenever a diver surfaces too rapidly from depth, releases bubbles that result in decompression sickness. Theoretically, decompression sickness and gas toxicity could both be avoided by filling the diver's lungs with a liquid instead of compressed gas. This liquid would resist the external pressure without a change in the volume of the chest and, at the same time, no gas would dissolve in the blood flowing-through the lungs since there would be no gas in the lungs in the first place.

To supply the diver with enough oxygen and an avenue for the disposal of the carbon dioxide that is continually being produced in his body would necessitate the use of a device similar to the ones now being used by surgeons to keep patients alive while repairing their hearts; an artificial lung, but this time fashioned after the gills of fish, et voila - homo aquaticus.

The essential feature of homo aquaticus is the incompressible liquid in his lungs. The artificial gill - a technical and surgical "tour de force" - is necessary to protect him from drowning, or is it? Could not the diver's liquid-filled lungs be made to function like gills? As it turns out, the answer is "Yes", and the advantages over the surgically implanted artificial gill are obvious.

Animal life on our planet began in the sea in an environment in which oxygen is relatively scarce. Early forms of animal life, making the best of the conditions imposed by the environment, evolved breathing organs such as gills that are capable of extracting adequate amounts of oxygen from water. Eventually the evolution of lungs made it possible for animals to emerge from the sea and to benefit from the physical characteristics and advantages of an oxygen-rich gaseous environment. Throughout the span of evolution, however, the function of the respiratory organs has remained basically the same: in both gills and lungs oxygen diffuses from the environment, across thin membranes, into the blood, and carbon dioxide coming out of the blood diffuses in the opposite direction to be discharged into the environment. Nevertheless, to reverse evolution and to resume water breathing presents formidable problems for a mammal. I have already mentioned one: the fact that under normal conditions, at ordinary atmospheric pressure, water contains too little dissolved oxygen. Another problem lies in the fact that natural water, be it fresh or seawater, usually has a composition which is very different from that of blood. Hence, when

such water is inhaled it causes lung tissue damage and, provided enough of it is inhaled, fatal alterations in the volume or composition of the body fluid.

Now it is a simple matter to prepare a liquid that overcomes both of these difficulties. Suppose we take an isotonic salt solution that is like blood plasma in composition, and charge this solution with oxygen under greater-than-normal pressure: the solutions' similarity to the blood will prevent any alterations in the volume or composition of body fluid by diffusion or osmosis. Under pressure, the solution can be charged with about the same concentration of oxygen as is normally present in air at sea level. Could a mammal breathe such a solution?

Using a small pressure chamber partly filled with an isotonic salt solution, I performed the first experiment, with a mouse, at the University of Leiden in 1961. The mouse was introduced at the bottom of the pressure chamber through a lock like the escape hatch of a submarine; the chamber had transparent walls so that the mouse could be observed. In the first few moments after entering the chamber , the animal tried to swim to the surface of the water, but was prevented from doing so by a grid below the water level. After a short period the mouse quieted down and did not seem to be in any particular distress; it made slow rhythmic movements of respiration - apparently inhaling and exhaling the liquid. It moved about in the chamber occasionally and would respond to a rap on the wall. Some of the mice so tested survived for many hours, the length of survival depending on the particular conditions of the experiment such as temperature and the chemical composition of the liquid. In each case, however, the mouse eventually ceased his respiratory activity.

From the results of variations of the applied environmental conditions, it appeared that the decisive factor, limiting the survival of the mice was not the lack of oxygen - which could be supplied in ample amounts simply by increasing the oxygen partial pressure in the liquid - but the difficulty of eliminating carbon dioxide at the required rate. The mousse that survived for the longest time - 18 hours was assisted by the addition to the solution of a small amount of tris(hydroxymethyl)-aminomethane, which is a substance that minimizes the untoward effects of carbon dioxide retention in animals and man. Lowering the temperature of the solution to 20°C, about half the mouse's normal body temperature, also lengthened the survival time by cooling the animal and thus reducing his metabolic rate, and consequently his rate of carbon dioxide production.

Now with mice in a small pressure chamber it is difficult to determine how much oxygen is actually taken up by the lungs, how well the arterial blood is oxygenated, and how much carbon dioxide the animal retains. Consequently, my associates and I resorted to more elaborate procedures using dogs in a large pressure chamber provided with additional equipment.

The entire chamber was pressurised with air and an anaesthetized dog was lowered into a tub of oxygenated saline. The animal was kept cool at about 32°C in order to reduce his oxygen requirement. While submerged, the dog continued to breathe, and jets of water rising from the surface showed clearly that he was pumping the solution in and out of his lungs. At the end of the observations, the dog was lifted out of the tub and his lungs were drained of water and re-inflated with air. One of these dogs was later adopted as a mascot by the crew of the Royal Netherlands Navy vessel HMS Cerberus.

We had now shown in measurable terms that under certain conditions a mammal could indeed maintain respiration by breathing water for a limited period of time. The blood pressure of the dog was slightly below normal while he was breathing the

oxygenated liquid, but it remained stable; his heart rate and respiration were low but regular and his water breathing kept the arterial blood fully saturated with oxygen. However, the carbon dioxide content of the blood steadily increased, indicating that the dog's vigorous respiratory efforts were not enough to remove sufficient amounts of carbon dioxide from the body.

I continued my studies at the State University of New York at Buffalo, using apparatus that makes it possible to measure the actual exchange of gases taking place in the lungs of water-breathing dogs. As before, an anaesthetized dog breathed the salt solution oxygenated under pressure. This time, however, the animal did not have to move the water into and out of the lungs on his own, and it was possible to measure accurately the gas content of the inhaled and exhaled water. Oxygenated liquid was delivered to the dog via a tube from a reservoir, and was drained back into a reservoir underneath the dog. A motor driven valve system regulated the respiration. The amount of oxygen taken up from the liquid in the lungs, and the amount of carbon dioxide discharged into it, was measured by comparing the relative amounts of these gases in the inspired and expired liquid. Samples were taken, so we knew the oxygen content of the liquid going into and out of the lungs; the dog was not cooled , and it extracted about the same amount of oxygen from water as it normally would have from air. However, in spite of the mechanical assistance to its water breathing, the animal did not eliminate sufficient amounts of carbon dioxide in the exhaled water, so that the partial pressure of carbon dioxide in the arterial blood gradually increased. At the end of the period of water breathing, which lasted up to three-quarters of an hour, the water in the dog's lungs was drained by gravity through a tube in the trachea and the animal's lungs were inflated with a few breaths of air blown into the tube. Several of these water-breathing dogs later became healthy and pleasant family pets.

It was now abundantly clear that inadequate elimination of carbon dioxide was the main problem in water breathing. There are two reasons for this. First of all, we now know that when a breath of fresh air or water is drawn into the air sacs of the lung, the oxygen molecules are at first concentrated in the centre of the sacs and have to traverse a substantial distance by diffusion before they reach the walls to enter the bloodstream; this distance is many times greater than the thickness of the membranes that normally separate air from blood in the lungs. If the breathing medium is air, the situation is of little consequence: oxygen diffuses in air so rapidly that freshly inhaled oxygen is distributed homogeneously in a matter of milli-seconds. However, when the medium is water, in which the respiratory gases diffuse about 6,000 times slower than in air, a gradient of oxygen tensions persists over the distance between the centre of the air sacs and the walls at the periphery. Throughout the cycle of each respiration the oxygen tension is greater at the centre than at the walls; the same being true of carbon dioxide discharged from the blood: it is more concentrated near the transfer membranes than at the centre of the sacs. Thus, at a normal resting respiratory frequency, the average carbon dioxide partial pressure in exhaled water is considerably lower than in exhaled air, at the same partial pressure of carbon dioxide in the arterial blood. Furthermore, the situation may be expected to get worse as the respiratory frequency increases and less time is available for carbon dioxide to diffuse during each breath.

Secondly, at normal body temperature, the solubility of carbon dioxide in water is less than in air, which is to say that water contains fewer carbon dioxide molecules than an equal volume of air at the same partial pressure. Hence, an increase in the partial pressure of carbon dioxide in the arterial blood, and consequently, in the air sacs in the lungs, would eventually restore the balance of the production of carbon dioxide in the body and elimination through the liquid-filled lungs. Unfortunately, the body tolerates only minor variations of carbon dioxide partial pressures in the

arterial blood. Obviously then if we put all these factors together, we find that in order to maintain his arterial carbon dioxide partial pressure within tolerable levels - to prevent a sense of suffocation or even loss of consciousness - a water-breathing diver would have to move a substantially greater volume of water per minute in and out of his lungs than the air-breathing diver moves air. At first sight this would not seem to be an insurmountable problem, since a suitable motor-driven pump could relieve the diver of the extra work of breathing, but unfortunately the maximum rate at which air or water can flow out of the lungs is effort independent: the flow initially increases with the increase in expiratory effort, but only up to a point, after which the flow no longer increases no matter how much pressure is applied to the lungs. The reason for this is the pliability of the walls of the airways so that they collapse once the critical expiratory flow has been reached.

David Leith and Jerry Mead at the Harvard School of Public Health, Boston, have measured the maximum expiratory flow of water from the lungs of dogs, and on the basis of their findings predicted that the maximum minute ventilation of a saline breathing diver would be approximately 3.5 litres. If one realises that a resting man must breathe normally almost twice this amount of air per minute and much more when he is performing work, then it becomes clear that the water-breathing diver could not possibly eliminate carbon dioxide at the necessary rate, even if he remained absolutely at rest in the water.

Now does this mean that we must find other ways to eliminate carbon dioxide from the body such as, for instance, an artificial gill? Not necessarily. Theoretically, the problem could be solved by using a liquid in which carbon dioxide is more soluble than in water, or by adding a substance which chemically binds carbon dioxide; the effect of either of these measures would be the same, namely to increase the number of carbon dioxide molecules present in the exhaled liquid at a given partial pressure.

We are mainly interested in the solubility of carbon dioxide at a partial pressure of 40 millimetres of mercury; that is a partial CO pressure normally found in arterial blood. Under these conditions, one litre of saline contains approximately 30 millilitres of carbon dioxide, while FC80 - a synthetic fluorocarbon liquid - contains 84 millilitres (almost three times as much), whereas one litre of air at the same partial pressure contains approximately 60 millilitres. A solution of tris(hydroxymethyl)-aminomethane in a 0.3 molar concentration and adjusted to a pH of 7.4 contains 320 millilitres of carbon dioxide per litre.

Since, in normal resting conditions a man produces about 250 millilitres of carbon dioxide per minute, it would follow that carbon dioxide elimination might not be a problem if a diver were breathing a tris buffer solution, even if his maximum minute ventilation were no more than 3.5 litres, as predicted by Leith and Mead. In fact it can be shown that such a diver would be able to perform work requiring up to 1,300 millilitres of oxygen per minute - that is, approximately four times as much as under resting conditions - and still have a normal partial pressure of carbon dioxide in his arterial blood.

Unfortunately, the solubility of oxygen in a tris buffer solution is no different from that in normal saline, thus an inspired oxygen partial pressure of approximately 18 atmospheres would be required to provide the diver with the 1,300 millilitres of oxygen per minute. If our diver were to breathe fluorocarbon liquid instead, his maximum oxygen consumption at a normal arterial carbon dioxide partial pressure would only be about 300 millilitres per minute - barely enough to support him while completely at rest. On the other hand, only one half of an atmosphere of inspired oxygen pressure would be needed to provide him with the necessary amount of oxygen.

In general then, the high carbon dioxide-carrying capacity of a tris buffer solution would allow productive physical activity of a liquid-breathing diver but, with the oxygen solubility being so low, prohibitively high partial pressures of inspired oxygen would be necessary. The solubility of oxygen in a fluorocarbon liquid is high enough to provide the diver with sufficient oxygen at safe inspired partial pressures, but the solubility of CO in fluorocarbon liquids is still not good enough: even at complete rest in the water and while breathing at his maximum capacity, the diver would barely be able to maintain a normal carbon dioxide partial pressure in his arterial blood.

Would it be possible to combine the advantages of a fluorocarbon liquid and of a tris buffer solution by mixing the two in suitable proportions? Fluorocarbon liquids do not mix with water and are in general very poor solvents for all but a few organic substances. However, it is possible to make stable emulsions of fluorocarbon liquid droplets in physiological salt solution containing tris buffers. Such emulsions have been prepared as a blood substitute, the fluorocarbon liquid droplets functioning as liquid blood cells to carry oxygen from the lungs to the tissues.

We can now estimate the maximum oxygen uptake through human lungs filled with different liquids at various arterial carbon dioxide partial pressures. The calculations are based on the known oxygen and carbon dioxide solubilities of various liquids, on an estimated maximum effective alveolar ventilation of three litres per minute, a minimum arterial oxygen pressure of 100 millimetres of mercury and a gas exchange ratio of 0.8. On the basis of such calculations one can predict that the maximum oxygen uptake of a saline-breathing diver could be no more than one third of his resting oxygen requirement at a normal arterial carbon dioxide pressure, that is to say, he could not survive under these conditions. In order to be able to extract the minimum required 300 millilitres of oxygen per minute from the oxygenated saline in his lungs, the partial pressure of carbon dioxide in his arterial blood would rise to 110mm of mercury and cause him to lose consciousness. As you may recall, these predictions are in accordance with the experimental findings in saline-breathing animals which I discussed earlier.

If a diver were to breathe FX80 Fluorocarbon liquid instead of saline, the situation would be somewhat better: the estimated maximum oxygen uptake would be slightly greater than his minimum oxygen requirement at a normal arterial carbon dioxide partial pressure. However, the slightest amount of physical activity, raising his demand for oxygen by no more than about 60 per cent, would cause an increase in his arterial carbon dioxide partial pressure to 60mm of mercury, and give rise to a sensation of impending suffocation. For this reason a fluorocarbon-breathing diver would not be of much use in the water.

If we estimate the maximum uptake of oxygen through the lungs of a diver breathing an emulsion of fluorocarbon liquid in an isotonic tris buffer solution, the prospects brighten considerably: the diver would be able to perform work that requires an oxygen uptake of 1,100 millilitres per minute, at a normal arterial CO_2 pressure of 40mm of mercury, and he could increase his oxygen consumption to almost 1,500 millilitres per minute before he would be so short of breath that he would have to stop. The diving women of Korea, who harvest abalone and oysters and other materials from the seabed at depths of up to 60ft, require about 1,200 millilitres of oxygen per minute while they are working underwater. So this would seem to be a reasonable figure for a reasonably active diver.

I have prepared a Graph showing the relationship between oxygen uptake, effective alveolar ventilation, and oxygen and carbon dioxide partial pressures in the arterial

blood of a hypothetical fluorocarbon emulsion-breathing diver. As you can see, the effective alveolar ventilation increases linearly with an increase in the oxygen consumption, while the arterial carbon dioxide and oxygen partial pressures at the end of an expiration remain 40 and approximately 1,300mm of mercury, respectively. Once the maximum alveolar ventilation of three litres per minute has been reached - which occurs at an oxygen consumption of approximately 1,100 millilitres per minute - a further increase in activity and consequently in the oxygen consumption, causes a rise in the arterial carbon dioxide partial pressure and a drop in the arterial oxygen partial pressure. An inspired oxygen partial pressure of approximately 4 atmospheres was chosen to prevent blackout as a result of arterial hypoxaemia before the diver's arterial carbon dioxide tension rises to a value that would force him to rest. In spite of the high inspired oxygen partial pressure, the partial pressure of oxygen in the arterial blood remains within acceptable limits, at least for a dive that would not last much longer than an hour or so. With liquid-filled lungs that would be long enough to descend several thousand feet into the water, work at depth for perhaps half an hour and return safely to the surface - all within an hour.

Granted that it has been shown that animals can breathe oxygenated salt solution or fluorocarbon liquid; granted that my estimate of the respiratory capabilities of a hypothetical fluorocarbon emulsion-breathing diver is approximately correct, what evidence is there that a real diver could tolerate the sensations arising from the presence of a liquid instead of air in his lungs?

During the past six or seven years my colleagues and I at Duke University Medical Center have been treating patients suffering from a variety of lung diseases by rinsing their lungs with a physiological salt solution in order to remove harmful secretions.

The patients are anaesthetised and a double tube catheter is inserted into the trachea: one lung breathes anaesthetic gas and oxygen while the other has its air replaced by physiological salt solution at normal body temperature. Once all the gas in the lung has been replaced by liquid, the rinsing procedure, which more or less resembles the normal breathing process, begins. A tidal breath of 500 millilitres of saline is made to flow into the liquid-filled lung and immediately after this the same volume of liquid is siphoned off. We continue this treatment for up to two hours and may use as much as 10 gallons of salt solution on one lung; so far we have done this on about 200 occasions, and the patients have suffered no harmful effects.

The same procedure was performed on a healthy volunteer whose windpipe was anaesthetised to introduce the catheter; he otherwise received no medication and was wide awake throughout the entire experiment. He told me afterwards that the liquid-filled lung had not felt noticeably different from the gas filled one, and that he had experienced no unpleasant sensations arising from the flow of saline in and out of his lung. Of course, such a test is very different from trying to breathe liquid with both lungs, but it did at least show that filling the human body with liquid will not necessarily damage the tissues or produce unacceptable sensations, provided a suitable liquid is used and provided the proper technique is employed.

With the advent of this volume-controlled lung lavage technique it has also become possible to measure accurately the rate at which a saline solution can flow out of the human lung. We have been able to drain 500cc of saline from one lung in 7 seconds, so it should be possible to exhale twice that amount, that is one litre of saline, from both lungs in the same time and, since inhalation and exhalation normally require about the same amount of time, it should be possible to move more than 4 litres of saline per minute into and out of both lungs. This is slightly better than Leith and Mead's estimate of 3.5 litres per minute.

The total amount of liquid inhaled and exhaled per minute will always be greater than the effective alveolar ventilation since part of it is wasted in ventilating the dead space. Consequently, the maximum effective alveolar ventilation may be expected to be somewhat less than 4 litres per minute, depending upon the size of the anatomical dead space, the respiratory frequency, the distribution of the flow of inhaled liquid and blood in the lung, and the presence of absence of partial gas pressure gradients within the liquid-filled gas exchange units of the lung.

During volume-controlled lung lavage in patients and again in a healthy volunteer, my colleagues and I found that diffusive mixing in the liquid-filled gas exchange units of the human lung is complete, provided that the respiratory frequency is no more than two or three breaths per minute. In addition, we found no evidence of a gross imbalance between the flows of inspired liquid and blood in the saline filled human lungs which, by the way, is in complete agreement with observations in dogs' lungs made several years ago by John West and his Clinical Research Physiology Group at Hammersmith Hospital in London.

It seems therefore safe to conclude that at a respiratory frequency of two or three breaths per minute the physiological dead space in a liquid-breathing man would not be much greater than the anatomical dead space of some 200 millilitres. Thus, with 4 litres ventilation per minute the effective alveolar ventilation of a liquid breathing diver could be as high as 3.5 litres per minute. As you will recall, my predictions were based on the assumption of an effective alveolar ventilation of 3 litres per minute, so they are in fact a little bit on the safe side.

This is in essence what is known now - 12 years after it was first shown that mice could be kept alive breathing on their own in oxygenated salt solution, instead of air. Many questions remain unanswered and much work remains to be done. Nevertheless, looking to the future, it seems likely that some day - soon, perhaps - some courageous man will take a deep breath of specially prepared liquid and that, by the turn of the century, divers with liquid-filled lungs will carry out critical rescue and salvage operations at great depths in the oceans, where gas-breathing divers would have failed.

Surgeon Commodore Rawlins: I think our last speaker gave an extremely interesting and erudite presentation, but I have always personally felt that homo aquaticus could be equated with man-powered flight: extremely interesting but not too practical, I could be wrong about that.

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Brief Profile

Johannes A Kylstra, widely known for his liquid breathing experiments, is Associate Professor of Medicine and Assistant Professor of Physiology at America's Duke University. Born in the Netherlands - he received a medical degree from the University of Leiden in 1952. From 1952 to 1954 he was an intern at Albany, NY, and from 1955 to 1958 he was a Lieutenant in the Royal Netherlands Navy Medical Corps.

In 1958 he obtained his PhD in physiology from the University of Leiden, and after three more years in the US, he served from 1961 to 1963 as Assistant Professor of Physiology at that University.

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DECOMPRESSION SICKNESS

Extracted with permission from Diving and Subaquatic Medicine (1975)
by Edmonds, Lowry and Pennefather

Following the development of the air pump by von Guericke in 1650, Robert Boyle was able to expose animals to decompression. In 1670 he reported these experiments and included the first description of decompression sickness - a bubble moving to and fro in the waterish humour of the eye of a viper. Hoppe-Seyler repeated these experiments n 1857, and correctly explained the mechanisms concerned. Admiral Sir Thomas Cochrane's pneumatic technique was used by Triger in France in 1839 in the development of a caisson for the sinking of coal shafts through the wet soils of Chalons. This was followed by the widespread use of the compressed air environment to allow for tunnelling, mining and caisson sinking. Trevessart and then Pol and Watelle observed Triger's subjects and the physiological and pathological manifestations of decompression.

Hoppe-Seyler repeated the experiments of Boyle. In 1857 he described the obstruction of pulmonary vessels by bubbles and the inability of the heart to function adequately under those conditions. He also recommended recompression to remedy this.

Le Roy de Mericourt in 1869, and Gal in 1872, described an occupational illness of sponge divers, which was also attributed to the breathing of compressed air and equated this with the caisson workers' disease. A host of imaginative theories was proposed during the 19th century to explain the aetiology of this disorder. It was followed by equally diverse mathematical models during the 20th century, also purporting to explain the same illness.

Paul Bert, in 1878, demonstrated in a most conclusive manner that decompression sickness is primarily the result of an inert gas (nitrogen in the case of compressed air divers and caisson workers) which had been dissolved according to Dalton's and Henry's Laws, and then released during or following decompression.

Haldane, Boycott and Damant in 1907 supported the concept of stage decompression where the diver ascends with a series of stops. It is based on the hypothesis of multiple tissue saturation rates and a critical super-saturation ratio necessary for bubbles to form. Most decompression tables still in use have their basis in this work, although the single critical ratio has now been replaced with multiple critical ratios for the different tissues at different depths.

Leonard Hill in 1912 produced both experimental and theoretical evidence questioning the value of stage decompression over continuous decompression, and this latter technique is now applied to decompression from saturation exposures.

Clinical Manifestations

Grading: A classification was presented as an attempt to differentiate non-serious and serious cases, so that identification prognosis and therapy could be more appropriately standardised. This clinical classification of Type I and Type II decompression sickness is not wholly satisfactory. The classification was neither defined nor applied in the same spirit. Type I is defined as (musculoskeletal) pain only bends. Type II included those presenting with symptoms other than pain, or with abnormal physical signs. The central neurological, cardiovascular, respiratory and gastrointestinal manifestations are potentially serious. Nevertheless in most series peripheral nerve symptoms are allocated to the same group as spinal and cerebral. Nor is there a differentiation made between vestibular and cerebellar presentations.

Although the appellation of Type I is reasonably clear when it is applied to acute decompression sickness affecting the musculoskeletal system ("joint bends"), the conglomeration of Type II manifestations, ranging as they do from an inner ear lesion to abdominal cramps to patches of hypoaesthesia to cardiovascular collapse, is of much less value. The classification is so commonly modified, even by its own proponents, that it appears to have lost any merit it originally had. The qualifying designation of the organs affected appears to be the only logical approach, eg. decompression sickness affecting the inner ear and musculoskeletal systems.

Onset. Decompression sickness develops after the subject has commenced decompression or ascent. Most cases present within 6 hours of the dive. In long or deep dives, cases may commence during the ascent. The delay in onset between initiating decompression and developing symptoms is influenced by individual and operational variations. The figures available must only be interpreted with an appreciation of the population being considered and the diving being performed. Some of the typical figures are presented in Table 1.

TABLE 1

POPULATION CHARACTERISTICS	TIME OF ONSET FROM SURFACING (percentage incidence)								
	0-30 mins. to 1 hour	30 mins. 2nd hour	3rd hour	4-6th hour		7-12 hour	13-24 hour	>24	Unknown
Royal Navy Divers	57	10	13	14		6	1		
Canadian Navy Divers	62	12	9	6	9	3	1		
US Navy divers (1)	54.7		12.1		19.5	6.6	2.3	0.3	4.5
US Navy divers (11)	43.9		27.4				3.9		
Hawaiian Civilians	66	6	4	3	2	5			14

The Naval series were heavily weighted for saturation exposures, whereas the civilian series was characterised by totally omitted decompressions. Thus the latter had few cases during decompression, the former had many. The civilian group was characterised by rapid and uncontrolled rates of ascent thus the incidence of early symptoms attributable to intravascular bubbles was greater. With an early presentation of symptoms, there are two diverging effects. The illness is potentially more severe, but treatment is more likely to be available.

Correlation of Symptoms

Although it is often stated that patients in the quoted series suffered from more than one symptom, the correlation between the different manifestations is not usually given. In some series, especially from cassion workers, the cumulative totals equal 100%, this suggests that not only the major symptom is documented. The two diver series which do show correlations between symptoms are fortunately the two extremes - one with US Navy controlled divers often in recompression chambers, and the other with uncontrolled diving civilians. The Navy divers represented a less severely affected group, the civilians more affected.

TABLE 2

N = 935

SIGN OR SYMPTOM	NO. OF INSTANCES	% OF INSTANCES	NO. OF INSTANCES MANIFESTED INITIALLY	% OF INSTANCES MANIFESTED INITIALLY
Localised Pain	858	91.8	744	79.6
Numbness or Paraesthesia	199	21.2	41	4.3
Muscular Weakness	193	20.6	8	0.8
Skin Rash	140	14.9	42	4.4
Dizziness or Vertigo	80	8.5	24	2.5
Nausea or Vomiting	74	7.9	8	0.8
Visual Disturbances	64	6.8	14	1.4
Paralysis	57	6.1	2	0.2
Headache	37	3.9	5	0.5
Unconsciousness	26	2.7	6	0.6
Urinary Disturbances	24	2.5	0	
Dyspnoea (“Chokes”)	19	2.0	4	0.4
Personality Changes	15	1.6		
Agitation or Restlessness	13	1.3		
Fatigue	12	1.2	2	0.2
Muscular Twitching	12	1.2		
Convulsions	11	1.1		
Inco-ordination	9	0.9		
Equilibrium Disturbances	7	0.7		
Localised Oedema	5	0.5		
Intestinal Disturbance	4	0.4		
Auditory Disturbance	3	0.3		
Cranial Nerve Involvement	2	0.2		
Aphasia	2	0.2		
Haemoptysis	2	0.2		
Emphysema - subcutaneous	1	0.1		

TABLE III

N = 100

Clinical Manifeatations	Incidence	Cerebral	Percentage incidence of associated clinical manifestations									
			Inner Ear	Spinal	Peripheral Nerves	Respiratory	Gastro-intesti-nal	Spinal or Back pain	Headache	Musculo-skeletal	Apathy and Malaise	Skin
Cerebral	24%		29	17	4	33	17	13	25	38	21	8
Inner Ear	18%	39		11	17	11	28	-	11	33	28	-
Spinal	22%	18	9		-	32	27	23	9	27	14	-
Peripheral Nerve	11%	9	27	-		18	36	-	9	64	9	9
Respiratory	21%	38	10	33	10		14	19	19	52	10	10
Gastro-Intestinal	16%	25	31	38	25	19		6	19	38	25	-
Spinal or Back Pain	11%	27	-	45	-	36	9		27	45	-	-
Headache	11%	55	18	18	9	36	27	27		27	36	-
Musculo-skeletal	54%	17	11	11	13	20	11	9	6		7	7
Apathy and Malaise	4%	38	38	23	8	15	31	-	31	31		-
Skin	3%											

Cutaneous

These manifestations range from being local and innocuous, to generalised and ominous, with a complete spectrum in between. They have been variously described as follows:

1. *Pruritis* A common complication of "diving" in compression chambers. It is often a transient effect, presenting very soon after decompression, and is not considered a systemic manifestation of decompression sickness.

   It is noticed mostly after short deep exposures, often with only one or two decompression stops. The areas affected are the forearms and wrists and hands, the nose and ears, and the thighs. The symptoms are transient and there is usually no objective sign available. In other cases there may be a slight folliculitis observed as red punctate areas, when this presentation merges with the next. The symptoms are attributed to small gas bubbles in the superficial layers of the dermis, and especially near its entry through the epidermis and the sebaceous glands.

2. *Scarlatiniform rash* This is an extension of the above manifestation. It is associated with pilo erector stimulation and perhaps a tissue histamine release and appears as a red punctate rash. The distribution is predominantly over the chest, shoulders, back, upper abdomen and thighs, in that order. The duration is from minutes to hours.

3. *Erysipeloid rash* This is an extension of the scarlatiniform rash and occurs over the same distribution, but with the involvement of endogenous gas interfering with venous drainage, it is a definite sign of systemic decompression sickness. Some of the skin appearance is thought to be a reflex vascular reaction. The lesions are collections of papules which may merge to form large plaques with flat and firm borders. Coughing or performing the Valsalva manoeuvre will accentuate the venous markings (Mellinghoff's sign).

4. *Cutis Marmorata Marbleization* This commences as a small pale area with cyanotic mottling. It may spread peripherally becoming erythematous with extension of cyanotic mottling. The area is warmer than the surrounding skin. Swelling and oedema result in a mottled appearance. Recompression gives dramatic relief. The area may become tender to palpation in a few hours, but the other signs may have diminished or disappeared by then. Marbling of the skin is a cutaneous manifestation of what is occurring elsewhere in the body, and thus is a serious sign of decompression sickness. Gas bubbles are present in both tissues and blood vessels.

5. *Subcutaneous emphysema* This has the typical crepitus sensation on palpation, either in localised areas or along tendon sheaths. It can be verified radiologically and should not be confused with the supraclavicular subcutaneous emphysema extending from the mediastinum and due to pulmonary barotrauma.

6. *Lymphatic obstruction* This presents as a localised swelling which may be associated with an underlying decompression sickness manifestation. If it involves hair follicles, a peau d'orange or pigskin appearance with brawny oedema is characteristic. It is common over the trunk, but is also seen over the head and neck.

7. *Counter diffusion of gases* There have been occasional reports of skin and mucosal swellings due to counter diffusion of gases. This results in bubbles forming at the interface between 2 gases diffusing at different rates, but with the total gas pressure at the interface exceeding the environmental pressure. This is only likely when the subject's body is exposed to a light readily diffusible gas, while breathing a heavier slower diffusing gas. The result is that the light gas moves rapidly through the body surface from the environment, while the inhaled gas maintains high tissue pressure and is reducing this slowly.

8. *Others* Formication may be the presentation in any of the skin manifestation described above, or due to involvement of the peripheral nervous system or the spinal cord. The neural involvement may also result in numbness, hypoaesthesia, paraesthesia and hyperaesthesia of the skin. These signs of inflammation may also occur over affected joints. Bruising is sometimes described over the chest and abdomen in serious cases, but this is not due to genuine tissue haemorrhages, as it blanches on local pressure.

Musculoskeletal

This is also termed "joint bends", "Type I", "pain only bends", "decompression arthralgia", etc. First, there is an ill-defined discomfort or numbness poorly localised to a joint, periarticular or muscular area. The subject may protect or guard the affected area, although in the early stages he may get some relief by moving the limb. Over the next hour or so the discomfort develops into a deep dull ache, then a pain with fluctuations in intensity, sometimes throbbing and occasionally with sharp exacerbations. Limitation of movement is due to pain, and the limb is placed in a position which affords the most relief. The duration of pain is often related to the severity of symptoms. The shoulder is the commonest joint affected in divers, in approximately one third of cases. Other joints, about equally affected, are the elbows, wrists, hands, hips, knees and ankles. Often, when two joints are involved, they are adjoining and frequently the localisation is between joints over the scapula, on tendon insertions etc. Rarely is the involvement symmetrical. The application of local pressure by means of a sphygmomanometer cuff, may result in considerable relief and may be of diagnostic value. It has been claimed that the site pain can sometimes be transferred by massage of the area.

In the mild cases, fleeting symptoms are referred to as "niggles", and may only last a few hours. The pain of the more severe cases usually increases over 12-24 hours and, if untreated, abates over the next 3-7 days to a dull ache. Local skin reactions may occur over the affected joint.

Although the musculoskeletal symptoms are the commonest presentation of decompression sickness in humans, the pathology of this is not well understood. Radiological evidence of gas both in joint spaces and periarticular lesions is available, but this is not necessarily the causative lesion. Extravascular bubbles in the subperiosteal area, tendons, ligaments, joint capsula tissue, fascia and muscles are thought to cause the pain of "bends". Bubbles in the articular vascular supply and referred neurological pain have also been incriminated.

Neurological

These presentations have produced a great deal of the past interest in decompression sickness. The areas affected can be subdivided according to the level of the nervous system affected.

The following clinical subdivisions of neurological presentations are:

Cerebral Any cerebral vessel may be occluded by gas bubbles, and this result in a great variety of manifestations, analogous to those of the cerebro-vascular accidents of general medicine. Especially noted are the homonymous scotomata, unilateral or bilateral, single or multiple. Other manifestations include hemiplegia, monoplegia, focal or generalised convulsions, aphasia, alexia, agnosia hemisensory or monosensory disturbances and confusional states. Raised intracranial pressure has been noted, and this may be associated with severe headache. In cases of homonymous hemianopsia electroencephalographic slow waves have been reported over the ipsilateral occipital cortex. Repeated EEG's are usually indicated during convalescence.

Serial, non-cultural, psychometric assessments of cognitive function may be of value if given before, during and after treatment. They provide measurement of mental impairment and response to treatment. Permanent mental impairment has been claimed as sequelae of cerebral decompression sickness. Brain stem involvement may also result in cranial nerve and pupillary abnormalities.

Cerebellar This results in ataxia, inco-ordination with typical neurological signs of hypotonia, diminished or pendular tendon reflexes, dysmetria, asynergia, tremor, dysdiadokokinesis, rebound phenomenon, scanning speech and nystagmus. The "staggers" which is variously described as vestibular, posterior column, spinal cord and cerebral decompression sickness, is probably more often due to cerebellar lesions.

Spinal Involvement of the spinal cord is most common in the lower thoracic or upper lumbar segments, although cervical lesions are by no means uncommon. It is often preceded by girdle pains, and commonly results in a paraplegic state. It is more common in patients who also have respiratory symptoms ("chokes"). The symptoms and signs are those of paraplegia or paraparesia, and include urinary retention with overflow incontinence. Often there is sparing of some sensory long tracts.

Peripheral nerve Bubble formation in the myelin of peripheral nerves will result in a patchy sensory deficit predominantly involving the lower limbs. The differentiation between this and an incomplete spinal lesion is important.

Pathological lesions in the neurological system include perivascular haemorrhages, oedema and demyelination in the cortex and subcortex, cerebellum, brain stem and spinal cord. The spinal cord changes are predominantly in white matter, and are most often observed in the mid-thoracic, upper lumbar and lower cervical areas, with the lateral, posterior and anterior columns suffering in that order.

The neurological manifestations of decompression sickness are explained in the following sequence of events. Gas bubbles form in the circulating blood after a short latent period after decompression. Most bubbles are filtered out by the lungs; some

bubbles, however, pass through the lungs, either by small arteriovenous anastomoses, or through the capillary lakes of Sjöstrand. These bubbles are small, about 25μ in diameter. They pass through the heart and reach the central nervous system and occlude arterioles of lesser calibre. The clinical manifestations, depending on the site of vascular obstruction and collateral supply are largely a matter of chance.

The multiple pulmonary gas emboli have secondary detrimental effects by raising pulmonary arterial pressure, and this may predispose to paradoxical cerebral embolisms through a patent foramen ovale, atrial septal defect or intra pulmonary arteriovenous shunts. Also of relevance is the interference with venous drainage of the spinal cord and subsequent damage to the cord. Some spinal cord and anterior horn damage may be due to spinal artery obstruction from an embolus. More importance is placed on the pulmonary hypertension interfering with venous drainage through the anastomoses of the spinovertebral-azygos system, subsequent engorgement and thrombosis in the vertebral venous system and oedema and infarction in the comparatively poorly vascularised area of the spinal cord - the mid-thoracic segment.

THOUGHTS ON RECORDS

"The Australian" recently quoted some recent additions and changes in the great wide wonderful world of record shattering achievements, where non-events become Guinness Crowned. Lest you are still awaiting the information, here it is. In Victoria, Texas, at the annual Armadillo Confab and Exposition, Elyira Rose Hunt has earned the title of Miss Vacant Lot of the World by stuffing 264 pennies into her mouth. As she wore a two piece purple hot pants outfit the spectators didn't object to her speechless condition. Mr Joe Pena filled, licked and rolled 42 cigarettes in five minutes. The results of the belching, yelling and spitting competitions are not yet available, regrettably. However, the All-China Nationwide Peoples Liberation Army Games (Peking) hand-grenade throwing record of 63 metres, held by 16 year old Ma Li-Li was not equalled. The only Australian entry for the useless records section was the successful breaking of the Manly Marineland Underwater Endurance record, now held by their chief instructor and shark feeder. Few of the fish who shared the record attempt are thought to have suffered ill effects.

For the benefit of anyone seriously wishing to establish some sort of record for getting air through a hose it may be an advantage to know what the present state of the market is. In 1946 an American "hard hat" diver aged 26 set what could be an involuntary record, though his times may have been exceeded. The conditions are as follows: without any warning the diver is imprisoned in a mud tunnel, unable to move, in 40 ft water beneath a sunken barge. The only surface contact is by the air hose and telephone. Duration of dive must exceed 3 days and nights and no food or drink is allowed. The cold and a knowledge that there was a constant risk of the wreck settling harder onto the trapped man are factors included by serious contenders for the Record.

Intending contestants should contact "Project Stickybeak" first.

Gastro-Intestinal Manifestations

Patients mildly affected may present only with anorexia, nausea, retching, abdominal cramps and diarrhoea. When the condition is severe, local ischaemia and infarction of bowel, with secondary haemorrhages, may result.

Cardio Respiratory

Intravascular bubbles are more common in the venous system, and are associated with sudden or severe decompression sickness. Although many of these bubbles may be trapped in the pulmonary capillaries, some may pass into the arterial circulation, either through a patent ductus arteriosus, septal defects, or via the pulmonary plexuses. The clinical manifestations may be arbitrarily divided into three types as follows:

i. Local ischaemic effect. This may follow cerebral, coronary, renal or splenic occlusion, etc. The result of these occlusions may be tissue ischaemia and infarction. The clinical manifestations will vary according to the organs involved. Specifically, an infrequent but troublesome cardiac manifestation of decompression sickness is the development of a ventricular arrhythmia, which may not respond to recompression therapy. It is not clear whether these all represent coronary emboli, or whether they result from extravascular bubbles interfering with the myoneural conducting mechanisms of the heart.

ii. Pulmonary involvement (“chokes”). Clinical manifestations are noted when approximately 10% or more of the pulmonary vascular bed is obstructed. Tachypnoea and hyperpnoea are observed. The initial symptom is chest pain aggravated by inspiration, with an irritating cough precipitated by cigarette smoking. Interference with the pulmonary circulation can result in circulatory collapse in severe cases. Pulmonary effects appear early, and are followed by either rapid resolution or a progression of symptoms. Investigations are often not possible, but ECG evidence of right axis deviation, high peaked p waves and right ventricular strain may be obtained.

iii. Post decompression shock. In very severe cases, eg. in explosive decompression or following grossly omitted decompression, there may be a generalised liberation of gas into all vessels, resulting in rapid death. The presence of gas bubbles in the circulating blood results in a bubble/blood interaction which leads to all grades of vessel wall and haematological reactions from haemo-concentration disseminated intra-vascular coagulation. Even in subjects without clinical evidence of decompression sickness, there is observed to be an increase in packed cell volume (haematocrit), haemoglobin concentration, plasma free fatty acids and prothrombin time; decrease platelet counts, plasma cortisol, complement activity and prothrombin consumption time. Those with symptoms also had increased fibrin degradation products.

The effects of hypotension, especially with air platelet and lipid emboli cause secondary hypoxic damage to capillaries, increased capillary permeability and extravasation of fluid into tissues. The signs and symptoms of hypovolaemic shock, such as haemoconcentration, postural hypotension, syncope, low urinary output, etc. are not uncommon, but like the pulmonary manifestations, they are either resolved quickly or proceed ominously. Once the cycle leading to disseminated intravascular coagulation has commenced, it does not necessarily respond to recompression therapy.

Thus such cases may deteriorate even whilst at an initially adequate recompression depth. Attention to intravenous replacement, heparin usage, correction of coagulation defects, etc. follows general medical principles, and may succeed where the recompression has not.

TREATMENT

Pol and Watelle in 1854 recorded improvement of patients who returned to a compressed air environment, ie. they were recompressed. This has remained the mainstay of therapy, but does need to be supplemented by general medical support. Treatment is likely to be more readily available under operational situations when the symptom occurs during decompression or soon after. Alternately, the illness is potentially more severe. This may be explained by the continuing tissue to bubble pressure gradient, the more likely presence of intravascular bubbles as opposed to the extravascular bubbles from slower desaturating tissues, and the consequent haematological complication of disseminated intravascular coagulation. The late development of symptoms heralds a relatively simple therapeutic regime, as exemplified by joint bends near the termination of a uniform and linear decompression from a saturation dive. In both early and delayed cases of decompression sickness, the longer the symptoms are allowed to persist and extend the more difficult is the therapeutic procedure.

Recompression Therapy

The volume of intravascular and extravascular gas bubbles decreases in proportion to the absolute pressure applied, in accordance with Boyle's Law. Thus at a depth of 165 feet or 50 metres, the pressure is 6 ATA and the volume of the bubble will be reduced to one-sixth of its size at the surface. This may be sufficient to relieve the symptoms and signs of decompression sickness and restore circulation. In this particular example the surface area to volume ratio is almost double, thus aiding the resolution of the bubble by increasing the nitrogen gradient. The standard air therapeutic tables have as their most useful depth, 165 feet or 50 metres. This was rationalised as being a depth at which further volume change would become insignificant, and at which the increase in nitrogen saturation of tissues is becoming prohibitive by increasing subsequent decompression requirements. That depth was also consistent with the working medical attendants not being incapacitated by nitrogen narcosis. The air tables, 1 to 4 of the US Navy Diving Manual, had the counterparts in most other Navies, and they varied mainly in their duration, from 7 to 43 hours.

Recompression followed by a slow decompression is the basis for treatment. There are three problems to consider in deciding the necessary form of the recompression therapy. These are the depth required for therapy; the gas mixtures used; and the rate of decompression. The gas mixtures and decompression rate are partly reliable on the depth of recompression.

In deciding this there are three different approaches which may be made, and these are as follows:

A. Recompress to a pressure (depth) dependent upon the depth and duration of the original dive.

B. Recompress to a predetermined fixed depth - ie. according to standard tables of recompression therapy.

C. Recompress to a depth which produces a clinically acceptable result.

These are now elaborated further.

A. *Recompress to a pressure (depth) dependent upon the depth and duration of the original dive.* This not a particularly satisfactory technique, as it is designed to cope with the total quantity of gas dissolved in the body during the original dive. Because decompression sickness is the clinical manifestation of a gas bubble lodged in a vulnerable area, it is necessary to recompress in order to reduce the size of that particular bubble, irrespective of the total quantity of inert gas dissolved in the body. This approach was best typified by the now defunct concept of treating aviator bends by descent to ground level.

   The one advantage of this approach is when a diver develops decompression sickness soon after surfacing from a deep dive. Under those conditions a prompt return to the original depth will ensure that there is no tissue to bubble pressure gradient which could assist bubble growth at a lesser depth.

B. *Recompress to a predetermined fixed depth - ie. according to standard tables of recompression therapy.* The standard tables of recompression clearly state the gas mixture to be used (usually air or oxygen). The application of the standard recompression tables produces relief in 90% of cases, if treatment commences within half an hour or the onset of symptoms. This proportion falls to 50% if the delay exceeds six hours. After a 12 hour delay, the results are poor.

   As a general rule the short air table, taking just under seven hours, is only used for the Type I cases and the longer tables, needing over 42 hours, are used for the more difficult cases. As most series deal with navy divers who have only marginally exceeded decompression requirements, the value of the air tables was not adequately quantified. They seemed of more value to the milder Type I cases than the others.

   The introduction of standardised oxygen tables using 100% oxygen interspersed with air breathing (Table 5 and 6 in the US Navy Diving Manual) gave far more flexibility and improved results. They are able to be extended and interchanged with the air tables at certain depths - mainly 30 feet and 60 feet. These tables became popular because of the improved results and the economy in time, needing only 135 minutes and 285 minutes respectively. The physiological advantages are in the speed of bubble resolution and increased oxygenation of tissues in countering the pulmonary arteriovenous shunting effects. Disadvantages include a fire hazard, oxygen toxicity, and the

occasional intolerance of a distressed patient to oxygen, or a mask, and the less immediate reduction in bubble size, ie. to less than half the volume achieved with the 165 foot standard air tables. Although the pressure gradient of nitrogen from the intravascular bubble to the blood is increased with oxygen breathing at 60 feet, if the diver has previously dived in excess of 60 feet, then there could well be a positive pressure gradient from tissue extravascular bubble still in effect during the early phase of recompression.

Despite the above qualifications, the use of oxygen has received world wide acceptance as a supplement to the air tables.

C. *Reoompression to a depth which produces a clinically acceptable result* allows a more flexible approach. The gas mixture which is likely to result in the maximum safe inert gas and oxygen pressure gradients may then be administered. For example, if the diver requires recompression to a depth of 100 feet, a 50% oxygen mixture may be used, and the oxygen percentage may be increased on reaching the shallower depths. In these circumstances, the rate of decompression must initially be decided arbitrarily - eg. 1 foot ascent per 4 minutes using 100% oxygen, and increasing the time by one minute for every 10% decrease of oxygen in the inspired mixture. The percentage of oxygen to be used may be calculated on the depth of the patient.

When administering oxygen mixtures the inert gas will usually be nitrogen, but this should be avoided in three circumstances. The first is with depths in excess of 180 feet due to nitrogen narcosis. The second is when the subject has respiratory distress. Under this condition either 100% oxygen or an oxygen/helium mixture should be administered, depending on the depth. The third is during the “off oxygen” periods, when it is customary to delay the onset of pulmonary oxygen toxicity by intermittently administering a low oxygen gas for an arbitrary time, eg. 5 minutes each half hour. In the case of decompression sickness due to air diving, at these times 20% oxygen/80% helium may be given to the patient who is in a good clinical condition. This helium mixture could temporarily aggravate the clinical condition of a seriously ill patient.

Decompression is halted at any stage if symptoms recur or others develop. Further recompression may be needed until an acceptable clinical result is achieved. The rate of decompression can be varied according to the response in the individual cases, and this may need to be slowed by up to 50% for saturation dives.

The value of this flexible form of therapy is that it is orientated towards the individual patient, and that it does not limit the medical officer by the imposition of standard tables which may be wholly inadequate for the severity of the case. The disadvantage is that it can only be used by experienced medical personnel and that constant attention to the clinical state of the patient is required.

All three approaches have some validity and may be relevant in different circumstances.

An additional technique of considerable value when there is no significant time factor involved, and if the patient is severely affected, is the 12 hour or “overnight soak”. It may be of benefit for many reasons to halt all decompression for this prolonged time. Bubbles have a chance to resolve before Boyle’s Law comes into effect with decompression. Also, tissue supersaturation of gas will become equilibrated with the ambient pressures. Oxygen toxicity may be relieved. Last but not least, medical and chamber personnel will be able to regroup and reorganise

The administration of 100% oxygen during the short term transport of patients to recompression facilities is recommended for 3 reasons: to increase nitrogen bubble resolution, to improve oxygenation to ischaemic areas, and to reduce the hypoxic effects of arterio-venous shunts. For any recurrence of symptoms after compression therapy, the administration of 100% oxygen by mask also may prove beneficial. The post therapy intermittent use of 100% oxygen is likely to considerably reduce the requirement for further recompression treatment irrespective of which of the above regimes is applied.

Whenever oxygen is used at atmospheric or greater pressures, attention must be paid to the problems of respiratory oxygen toxicity. It is strongly suggested that the oxygen parameters should not exceed those likely to result in neurological or pulmonary toxicity (ie. pO2 of 2.0 ATA, and the vital capacity measurements should not be permitted to decrease by more than 20%).

Supportive Therapy.

Other forms of treatment have been used for decompression sickness. Heparin has been advocated because of its lipaemic clearing activity and its preventative effect on platelet clumping. It may be indicated in cases of disseminated intravascular coagulation which have no evidence of systemic infarctions and bleeding. Correction of coagulation defects seems a more logical approach to the problem of disseminated intravascular coagulation in decompression sickness. Low molecular weight dextran has also been used in these cases and may be indicated when the haematological changes become marked. Vasodilatation has been attempted by drugs and reflex stimulation. Mannitol has been used to reduce cerebral and spinal oedema, as has the administration of steroids. Dexamethisone 10 mgm IV and 4 mgm IM every 6 hours, is often used in neurological cases. It must be discontinued within 72 hours unless maintenance steroids are to be used. Hyperbaric oxygen therapy, repeated daily, may also be of value in these cases. Hypothermia has been proposed for persistent cerebral oedema. Hypovolaemic shock will require intravenous fluid replacement, with appropriate clinical monitoring. Symptomatic therapy is also required, eg. urinary catheterisation, administration of analgesics, etc.

It is essential to realise that general medical treatment is required during the recompression regimes. Patients should not be left unattended in recompression chambers, and especially whilst breathing increased oxygen concentrations. First aid and resuscitation techniques are often required as are accurate clinical assessments, and for these reasons it is desirable to have a trained medical attendant in the chamber. It may be necessary to consider the possibility of decompression sickness occurring in the attendants, especially when the patient is subjected to oxygen or oxygen-enriched mixtures. The decompression regimes are based on the gas mixtures being breathed by the patient, and not the air being breathed by the attendants.

DIVING 2001
Hannes Keller
(Oceans 2000, 1973)

It is a great privilege and pleasure for me to run through some of the problems of the future of man in the sea. I will concentrate on three topics: the scuba diver, the underwater sea quest and homo aquaticus.

Many of us are now chiselling at the limiting factors of diving; we dream of extended no-decompression diving. The problem of decompression lies in the fact that one cannot dive deep with oxygen alone because it is a very poisonous gas that men can only support in small quantities. In diving, one must mix this powerful oxygen with harmless gases such as nitrogen, helium and so on. These gases diffuse into the body, stay there like blind passengers accompanying one on the dive, and do not react with the body chemically. But, when one returns to the surface, these slow-witted non-reacting gases come out of the body, and if one is not making a slow and tiresome decompression, form dangerous bubbles in the body. This very much limits the freedom of the diver.

There are four hypothetical methods of getting rid of the need for decompression: drugs preventing bubble formation; non-inert breathing gas mixtures which are chemically absorbed by the body; liquid breathing mixtures instead of gases, and extra-corporal blood circuits (the lungs being filled with a liquid).

Drugs Preventing Bubble Formation: When the gas pressure in the body tissues versus ambient pressure exceeds a ratio of approximately 2:1, we get bubbles. This limits diving with scuba to between 100 and 200 ft. For greater depths one needs industrial diving techniques such as decompression chambers and submarines but drugs may now change the critical ratio. Today, I believe that some 20 per cent improvement could be realised, and in the future, super-effective drugs might double or treble the ratio, but at this point one certainly reaches the limits of what chemistry can do for you.

Depths for scuba diving could be doubled or trebled, but one would be forced to use helium instead of nitrogen for breathing. However, helium saturates the body 2.64 times faster than nitrogen, and this would again take us back to short decompression diving at shallow depths.

We can estimate, therefore, that a suitable drug applied to a helium-nitrogen mixture would allow the scuba diver to go to depths of between 200 and 400 ft. This does not sound terrific for the magical year 2001, but it would mean that really black depths would be open to everyone, and that certainly would be a moving experience.

However, if there were a gas that would mix with oxygen and which could be absorbed by the body without poisoning it, it would give us diving without decompression. Very hypothetically, such a thing would look like this: at 300 ft, we would have a mixture of 5 per cent oxygen, 45 per cent xx, 45 percent UU plus 5 per cent corrector-catalyzer mixture (whatever that means!). The blood plasma would form an xx plasma, and UU plus tissue fat would make U fat. Then the xx plasma plus the UU fat would form a green liquid! The green liquid would dissolve in the blood, be filtered by the liver and leave the body with the urine.

The 5 per cent corrector-catalizer mixture would do some very odd jobs indeed.

When it came to ascending to the surface, all the gases would have disappeared; nothing would be left for bubble formation and no decompression would be needed. We could expect the gases like xx and UU, being non-toxic, to have molecular weights of 60 or more. Such gases have critical points of 500 psi maximum, therefore, we cannot expect to be at depths greater than 1000 ft.

Breathing Liquid: This has been discovered by Professor Kylstra. It is fantastically simple: one just drowns in physiological salt water which is saturated with oxygen, but instead of saying "Farewell beautiful world" one stays alive. It must be done at depths in excess of 300 ft, or the farewell would be final, since the liquid cannot hold sufficient oxygen at a lesser depth. Maybe new liquids will overcome this, but for anybody who can beat his psychological barriers against it, it is a great way to solve all diving problems!

Extra-Corporal Blood Circuit: The technique for this is, in a way, similar to the liquid breathing method. One fills the lungs with a suitable liquid and one may or may not continue to breathe. An artery or vein is cut and interrupted, the blood being guided through an apparatus which one may carry under the arm. The apparatus does the lungs' work by getting oxygen into the blood and carbon dioxide out of it. So, after joining a diving club, the diver sees his surgeon, gets his plugs installed and has no further diving problems. Whenever he feels like going underwater he just pays his electricity bill, connects the apparatus and drowns a little bit! The diver could now reach the absolute limits for diving - the depths could be as much as 5000 ft. But then the body chemistry could become upset - under the extreme pressure the metabolism would change delicate equilibrium with fatal results. Such changes of metabolism occur when one changes body temperature. Very roughly, one can say that each 1000 ft. is equivalent to a degree Fahrenheit body temperature change. The effect is much too complicated to be compensated by drugs, so I believe that the limit of diving is between 3000 and 7000 ft. Naturally, one day some crazy chap will make it 7043 ft.!

For the scuba diver, descent and ascent rates will be a big problem. (I once made a descent from 300 to 1000 ft. in two minutes, but I admit that I did not feel too great). Today, one understands that pressure changes cause specific problems that limit ascent and descent rates to between 100 and 300 ft. per hour at depth.

For the properly equipped sports scuba diver of the future, I would expect dive durations of six to eight hours, the one big problem being that of opening the energy gap. To heat a swimming diver, a suit must generate approximately 500 watts; with propulsion and rapid water velocity cooling him, the diver needs 1000 watts. Propulsion (if only we could think of a small torpedo that could be carried on the belt or between the legs) requires another 1000 watts. We want about eight hours duration - and here we have a tremendous battery problem. We can manage with 1000 lbs of lead acid battery, or with 200 lbs of silver zinc battery, or with 40 lbs of liquid oxygen and hydrogen plus a fuel cell, or with atomic power - if atomic power can be provided and if the small isotope battery is ever constructed.

A diving suit must give protection without hindrance; thermal, mechanical, optical and logistical protection. The materials of the future will be strong enough to make you laugh very loudly when a white shark tries to chew you, but the point is that the suits will be strong enough to be blown under pressure if you surface accidentally and need pressure for the prevention of a decompression accident. Then, naturally, the suits will allow efficient buoyancy control in conjunction with the breathing apparatus and electronic black box.

At present I am working on a suit which completely protects a diver from the hazards of drowning: if the diver loses consciousness, he will continue to breathe and automatically drift to the surface instead of being lost at depth. Rescue will then be easy. Such dry suits will even beat the wet suit for comfort.

Electronics: Today, Hewlett Packard markets a pocket computer which has forty thousand transistors, taking care of all basic mathematical functions of trigonometry and so on. Very soon somebody will make such a thing for divers and your dive will become almost automatically controlled. The box will provide a continuous dive plan, including return to base, control of vital body functions, supervision of overall safety and precautions, computation of optimal procedures in case of failure and trouble, control of breathing apparatus, automatic control of ascent in case of unconsciousness, communication to other divers and base, and navigation relative to base, divers, target, surface and bottom. Some of this data will be received by the diver via the apparatus.

A laser beam projected on to the faceplate will give a three-dimensional holographic display of the navigational situation, including one's own path underwater, the positions and movements of other divers and the position of base, surface, bottom and target.

Maybe ultra violet light will penetrate depth and dirty water, and provide visibility. The navigation system will be provided by means of ultra sonics, maybe electro-magnetic waves, and probably inertia systems as those in submarines and jumbo and fighter planes, only much less precise and costly.

The future belongs to a variety of breathing apparatus, and although nothing will ever beat the simple aqualung, we will have miniature compressors silently filling our cylinders over-night, and a small scuba set fitted with 8 lbs of air will weigh approximately 20 lbs, including the air vaporizer.

In the closed-circuit deep-diving apparatus, CO_2 and other contaminants will be frozen out of the circuit; no filters will be needed, and buoyancy will be easily controlled.

So, what I predict is this. The oceans are not dead. In 2001 I hope still to have the choice of jumping nude into the water and playing with the mask, fins and snorkel I bought in 1960, but the miliatary divers will swim around silently with an extra-corporal blood circuit, and some enthusiasts will breath liquid far down at 3000 feet. Naturally, it will be great to be able to move hundreds of feet up and down, with propulsion, naviagation, communications and, last but not least, liquid gases.

Living Underwater: The earth is becoming somewhat crowded; there is a tremendous energy-gap, and there is a contamination problem. In the 19th century, people had great problems in protecting themselves from contamination inside the house - outside, nature was healthy. Today it is different. Last week I was in New York - I am told that breathing air in a New York street is equivalent to smoking 40 cigarettes a day, and that one cigarette shortens life by 15 minutes; one day in New York therefore shortens my life by half a day. However, I was in a hotel which advertised filtered air, so I spent as much time as possible in my hotel room so that I was safe from contamination!

I believe that the future belongs to three-dimensional structures, towns which are completely closed shells. Inside such shells one would be able to keep the air conditioned and to filter out all contaminants.

A sociologist told me that the ideal city has one million inhabitants. Such a city could be designed as a cube, as a sphere, or other three-dimensional shape. A 0.6 x 0.6 x 0.6 mile cube would give each person - child or adult - one thousand cubic metres of space, which amounts to about twice the volume of a complete six-roomed apartment, including a garage. In such a cube people would live and work and communicate.

The first advantage - elimination of the energy gap: whatever energy the industry in the centre would use would heat the apartments. Secondly, people could commute easily with elevators and mechanical stairs - everyone could walk anywhere in 15 minutes. Thirdly, climate and contamination would be under control, with minimum technology needed. Fourthly, Nature would be within 10 minutes walking distance and could be kept intact for agriculture and recreation and joy - no more fences and houses anywhere - all would be one really nice big garden. Fifthly, it would be economical. Lastly, social contact would be optimal: one would have all the human contact one wanted, plus rooms for privacy for, naturally, to be happy, one needs a room in which to he alone - without acoustical or optical contact with the outside.

It has been proposed that we build such structures underwater. I am afraid this is not possible. It would be economic nonsense because of a simple physical law: a city underwater has to be of the same density and weight as the water it displaces.

If a structure is made of concrete, then 40 per cent of its volume would have to be of solid concrete; a room 21 ft wide would have concrete walls 3 ft thick. If made of steel, 13 per cent of the entire volume would have to be of steel: a room 25ft wide would need solid steel walls 1 foot thick. For buoyancy reasons alone, an underwater city would require several times more building material than a sky-scraper structure on land, which makes a nonsense of the whole idea. If one thinks of heavier materials, of the costs and quality of this magnitude, it is impossible

Floating cities - Yes! If one needs cooling, the water under a city would inevitably carry away heat energy. In a hot clinmate this is wanted, not so in a cold climate. So, certainly, Miami 2001 will float, while Oslo 2001 will be on solid ground where minimum heat is lost.

To Swiss people, the thought of a three-dimensional city filling a little valley with lots of people, all with their little bank accounts in the country, appeals very much. Actually, such structures have already been tried. The Egyptians once made big three-dimensional structures, but the architects only dared to move the dead inside. In Babylon, an architect tried, and when he failed because of an error in the calculations, found the greatest excuse I have ever heard. Since Babylon all architects have had their splendid excuses.

If underwater cities are impracticable, perhaps there is even less need for humans to acquire gills and cold blood and become homo aquaticus. Cousteau has predicted that selection and mutation will create new forms of human beings, capable of living fish-like underwater, for instance. But the human and his domestic animals have escaped the selection process from Nature - it is known, for example, that Eskimos are not hairier than Africans.

When trying to predict the future of mankind, we can certainly analyse some trends. One will try to permit the maximum number of individuals the experience of life. That means a big crowd! This crowd will certainly have to live in three-dimensional structures; the cities of the future. There is no need for humans to fly like birds as homoaeronauticus, or swim under the sea as homo aquaticus; if a city has 500 levels,

its three dimensions are enough. The factors limiting life will therefore be the energy-gap; waste of prime materials and contamination.

Other Trends: Each individual will seek maximum 'life fulfilment' which means a maximum of brain stimuli. Brain stumuli can be realities making an impression on nerves which then act on the brain, for instance, London Tower, or drugs, or electrical impulses - a computer could be connected to the brain to make it 'see' the Tower. But humans want not only a maximum of stimuli on the brain but also a maximum variety of stimuli. The brain wants to be able to choose its stimuli; it wants the freedom to choose and change. But the concept of homo aquaticus is profoundly opposed to these needs; aquaticus would have a very dull and one-sided animal life.

Another strong argument against aquaticus is the difficulty of maintaining body heat. The human metabolism is set at a very precise constant temperature, and homo aquaticus would need highly complicated chemical mechanisms to compensate for such temperature effects. The metabolism would need to be at least three times more complicated than it is already, and would probably be three times more prone to illness and malfunction. Nature dislikes such impracticable constructions.

If humans really want the maximum number of individuals surviving for the maximum time, then something else is needed.

Humans will not live in the sea, but they must become smaller - very much smaller. People only 3 ft tall could be as intelligent, sensitive, beautiful and as sexy to each other as people who are 6 ft tall. If humans could stop growing when eight years old, they would be easier to feed and to transport; less energy and prime materials would be used up, and more people could find a place on earth. Muscular effort could be implemented with tools. It could already be done today with hormones.

Finally, if by 2001 all the big fish have been shot, then at least a 3in fish, will be a great experience for a 3in diver!

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Brief Profile

Hannes Keller from Switzerland was the first man successfully to dive to the record depth of 1000 feet, and he did it as far back as 1962. Sadly, this achievement was marred by the tragic death of his diving companion, Peter Small.

Since then, he has concentrated on the technical development of safe and efficient underwater equipment for industrial deep diving, principally producing a professional suit and new chamber systems. He has also produced two-man and five-man portable recompression chambers.

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A FATAL DIVE

Dr FM Cave

This diver ‘V’ spent the day before he died getting his hookah and other gear ready. He may have had some beer before tea late that afternoon but certainly did not go out after his tea.

He left his home at about 5.15 am in company of two others, ‘A’ and ‘B’. The boat was launched about 7.15 am and they arrived at the dive site off an island an hour later. Diving was commenced without delay using two hoses from the hookah equipment in the boat. ‘V’ dived to the bottom and was followed by ‘B’ using the other mouthpiece, the latter waiting till he saw ‘V’ reach the bottom. ‘B’ glimpsed ‘V’ occasionally for about two minutes, lost sight of him for about two minutes then saw him swim past about 20 ft away, coming from behind him on a 45 degree angle and swimming level within a couple of feet of the bottom. He then swam out of ‘B’s sight. ‘B’ continued looking at coral until about two minutes later when he saw ‘V’s speargun standing vertically, spear down, with the point of the spear resting on the bottom. He swam over to the gun and found also a flipper on the bottom nearby, so he picked up both articles and surfaced. At the surface he dropped them and peeled off his own flippers.

‘A’ had remained in the boat with the motor. He estimated that ‘V’ was submerged for about 8-10 minutes before he saw him swim across and check the boat anchor and then swim to the end of the line. Soon after ‘V’ came up and called, apparently in distress though the engine’s noise prevented his words being heard. ‘V’ went under again for a moment, then resurfaced and called again. He probably had his mouthpiece out and his mask on his forehead the first time he surfaced and it was certainly out when he went under the second time. ‘A’ pulled him to the boat with the hose. He probably became unconscious about 8-10 ft from the boat. ‘A’ could not lift him into the boat unaided (he weighed about 12 stone). His eyes were half open and turned back. There was clear froth spurting from his nose and mouth but no unusual smell or vomit. He was pale.

‘B’ swam over and tried to help lift him into the boat from the water but had to come aboard himself before ‘V’ could be lifted in. The purge button of the mouthpiece was tried and found to work normally. Mouth-to-mouth resuscitation was tried. There was some difficulty in getting the boat’s engine started before they could take ‘V’ ashore to the island’s resort. There a nurse confirmed that death had occurred.

I interviewed the victim’s father and the two companions on the dive, also examining the equipment, establishing the following facts:-

- ‘V’ was in very good health, physical and mental. He had been diving for at least six years and started to use scuba about six years ago. He bought the hookah new about two years before this accident.

- ‘V’ had told ‘B’ “never hold your breath while using hookah, always breath normally, don’t rise faster than your bubbles”, he had taught him to clear his mask and to remove and replace his mouthpiece underwater.

- ‘B’ does not remember being instructed in free ascent but may have been told “if you have to ditch, breath out before coming up”. I am told that ‘V’ had attended lectures on these subjects given by myself. Such is the only indication we have as to the extent of ‘V’s knowledge.

‘B’ had been shown how to use hookah equipment in a swimming pool but had never previously dived in the sea either with snorkel or hookah. He is intelligent, healthy

and in his early twenties. ‘V’ was 29 years old. ‘A’, the man left in the boat, had never dived.

Equipment was ordinary petrol driven compressor with air intake from a flexible pipe about 10 foot long placed over the front windscreen of the boat. The boat engine’s exhaust was a two foot pipe extending vertically from the engine to reach any breeze.

The hookah supplied air through a hose that I tested and found to be very difficult to kink, resisting kinking unless used two hands to twist it. This went to the weight belt where was an adaptor, thence by a short tube to the mouth piece. The mouthpieces were tested after the accident and found to work satisfactorily. The only abnormality found on taking the mouthpieces apart was a twisted coil spring at the valve; this was one used by ‘B’. They were See-bee with tilt type up stream valves with a non-return valve. There was a small amount of verdigris in ‘V’s valve.

Both divers had their weights on the same belt as the hookah adaptor was on, and neither carried a snorkel. Though unwise, such factors did not appear to bear on the accident. ‘B’ had no difficulty with air supply at any time, though the same source supplied both divers.

Conditions on the day were a calm sea, light north-east wind, the boat was on the south-east side of a small island, and underwater visibility was good. The sea bottom could be seen from standing in the boat. Neither diver wore a depth gauge. Water depth will have been not more than 20 feet at the time of diving. The equipment was recovered the day following the incident and showed no damage marks.

The post mortem report was: brain swollen and the overlying cerebral vessels contain air. The lungs are distended and pale and there is underlying air in the pulmonary veins. The heart shows two areas of fresh haemorrhage from capillaries over the external surface and there is air in the coronary veins. Cause of death - Air Embolism.

Conclusion: It seems almost certain that ‘V’ ascended to the surface without exhaling. Why he did so cannot be known. There is no evidence of any natural illness, there is no evidence to suggest that his mouthpiece valve stuck, no sharks had been seen in the area and he would be too used to seeing sea snakes to become alarmed by their presence.

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RISKY JOBS

The Metropolitan Life Insurance Co. recently completed a study of hazardous occupations and came up with some interesting statistics. They found that probably the most dangerous job possible is sponge diving, though only 25 persons make a living in this occupation in the USA because artificial sponges are taking over the market. Following sponge divers in the high risk list are aerialists, motor cyclists and auto racers, lumberjacks, bank guards and, interestingly enough, deep sea and Great Lakes fishermen.

Skindiver Magazine, November 1974

A REVIEW OF AIR EMBOLISM AMONG SCUBA DIVERS IN THE MONTEREY PENINSULAR

Takashi Hattori, MD

The controversy over whether the novice scuba diver should be taught “free ascent” before receiving his diving certificate will always remain. Personally I feel that the diver should not attempt a free ascent until his or her third ocean dive and then only after practice under supervision in a pool. A second, though sometimes impossible condition is that no free ascent should be taught where a recompression chamber is not available nearby with an MD and operating personnel at immediate readiness.

Besides emphasis on the need to expel air as one ascends, the fact that severe and forced expiration before starting the ascent should be avoided also needs to be explained. Last summer I had two cases of air embolism which could only be explained on the basis that initial forced expiration closed off some of the lower lobe terminal bronchioles which then remained closed resulting in over expansion and rupture of alveoli and subsequent embolism.

From 1971 through 1974 a total of ten cases of air embolism or possible air embolism were treated on the Monterey Peninsular, California. Of these, three were embolized during the practice of “free ascent” as a part of their last ocean dive to qualify for the novice certificate. Known cases were 1971 - one, 1972 - two, 1973 - two and 1974 - five cases.

Cases incurring Air Embolism while practicing FREE ASCENT

Case 1 (1972) Male, age 21

On first ocean dive, panicked during free ascent from 25 feet. Did not exhale for the last 15 feet of ascent. Became unconscious within 2 minutes of surfacing. Compressed to 165 feet approx. 20 minutes after losing consciousness, regaining it after 10 minutes at 165 feet. Flown to San Diego on Table 4 (in 1-man chamber). Treatment was finished up on Table 6 with O_2 from 60 feet at San Diego. Victim was blind for three days after treatment but sight returned spontaneously. Psychometric examination at UC Medical School showed no neurologic deficit.

Case 2 (1974) Male, age 30

Complained of weakness after second “free ascent” from 25 feet. Buddies in class removed his weight belt and tank and entire class started for shore. His wife looked back to see how he was and found him floating unconscious and drowning. He was brought ashore, and then to hospital by ambulance.

He required 1 hour of intensive resuscitation from severe drowning before I could place him in our chamber. He was taken down to 165 feet while barely conscious and then transported to San Diego on Table 4. Treatment was completed on modified Table 6 on O_2. Recovery was complete.

Case 3 (1974) Female, age 25

Complained of chest pain after free ascent practice from 25 feet. She was brought to shore where she also developed dizziness. Examination at hospital revealed no nystagmus but there was loss of vibration sense in the left ankle. There was no muscle weakness and the remainder of the neurologic examination was negative. Chest pain had disappeared.

Since our chamber had not been returned from San Diego, she was placed in Trendelenberg position 10°, IV Saline was started and 100% Oxygen given while being flown to San Diego at 500 feet altitude. She entered their chamber approximately 3 hours after the accident.

It is interesting to note that her headache was slightly worse on reaching San Diego and she developed severe nystagmus on sitting up to enter the recompression chamber. Vertigo, nystagmus, loss of vibration sense, and headache all cleared within 10 minutes after recompression to 165 feet. She remained asymptomatic after leaving the chamber.

Other causes of Air Embolism

Case 4 (1971) Male, age 28

On his third ocean dive, with an instructor, he was down at 75 feet when he came to a rock about 25-30 feet high. He was excited and thinks he forgot to let his breath out as he came up over the rock. He became hemiplegic as he passed over the rock. His instructor saw that he was "in trouble" and assisted him to the surface and then to the shore. He was disorientated, unable to talk and unable to move his right extremities so was taken to the Pacific Grove Fire House. There he was found to be orientated as to time and place, able to talk and had recovered ability to move all extremities. However, there was residual weakness in his right arm and leg. Tongue did not deviate. Right biceps and knee showed hyperactive reflexes. Babinski was negative.

He was taken to 100 feet in our single-lock chamber and treated on Table 1A. He was completely asymptomatic at the end of treatment.

Case 5 (1972) Male, age 19

0n his first ocean dive with the class he was down at 60 feet with his instructor, who later described how he took his eyes off the victim for a couple of minutes and when he looked back he saw the victim on the bottom with his mouthpiece out. The instructor panicked and pulled him to the surface without forcing air from the victim's lungs. Haemoptysis was observed on surfacing. It took 30 minutes to get him to shore from the dive site, during which time he was unconscious and remained so when placed in our chamber. He was transported by air to San Diego on Table 4. Death occurred after the small chamber had been placed within the larger San Diego one, before pressure was equalised.

Case 6 (1973) Male, age 28

This uncertificated diver with 2 years diving experience was found unconscious in 30 feet of water. He was pulled to the surface by his buddy without forcing air out of his lungs and reached the hospital approximately 30 minutes after the accident. Pupils were barely reactive, there were no deep tendon reflexes, he was breathing spontaneously and blood pH was 6.8 with arterial O2 saturation 48% on arrival there. He required 1 hour of intensive resuscitation measures before I thought he could survive the usual three hours before he would reach the San Diego chamber for treatment. Such therapy was chosen because air embolism could not be excluded by the case history obtained. He was air transported on Table 4. He was treated on Table 6 three times in three days at San Diego Naval Hospital, being discharged apparently completely recovered 6 days after the accident.

Case 7 (1973) Male, age 16

Regulator trouble was encountered at 90 feet depth, necessitating an emergency ascent. Intermittent loss of consciousness occurred over the next 20 minutes. The patient received 500cc IV saline, 20 mgm decadron, and oxygen while awaiting the arrival of our chamber by air transport from Monterey to Eureka. He was then placed in this on Table 4 schedule and taken to San Diego, treatment being completed there on Table 6 on O_2. Recovery was complete.

The portable one-man hyperbaric chamber is single lock, air or oxygen capable, manufactured in Italy, 800 lbs, 6'4" length, 28' diameter (32" at hatch). It is kept at the Pacific Grove Fire Department for emergency use by the Marine Rescue Patrol. It can be transported by helicopter, the one used being a USOG H-3 (large). It can be placed within the large chamber at Catalina through the 29" door by dint of great luck, much angulation and less than 1/64th inch "clearance". An article about the operation of our Rescue Patrol and some of the problems we have encountered will be printed at a later date.

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Drugs from the sea

Mr Richard Helms, former director of the US CIA told the Senate committee on Intelligence that included among poisons stockpiled there was lethal shellfish toxin.

(*The Australian*, 19 September 1975)

The new Physiology (1)

According to the director of the Sydney Human Performance Laboratory, the Parramatta players "had performed beyond the limit of human endurance". It just shows the value of training!

(*The Australian*, 29 August 1975)

The new Physiology (2)

It as reported that Mr Frank Braun, 1968 president of the South African Olympic Committee, once gave as the reason for the absence of blacks in South African swimming team, that "Some sports the African is not suited for. In swimming the water closes their pores and they cannot get rid of carbon dioxide, so they tire quickly". An interesting observation!

[illegible]

The new Physiology (3)

Prof. Leston L Havena, Professor of Psychiatry at Massachusetts mental health centre at Harvard Medical School has said that most people are psychologically dead by the time they are 30. "They go on breathing for a lot longer than that but from a psychological point of view it would be hard to find signs of life." A Canberra team of academics and researchers has worked for nine months to reveal that human beings are limited in their capacity to learn. They observed that what a team decides is the solution of a problem may be different from the solution that any single member of the group might have arrived at. All things considered, it is lucky that the ages of the SPUMS committee are to be kept strictly secret!

(rewritten from the *Daily Telegraph*, of 23 July and 14 October 1975)

..DOPPLER ULTRASOUND FOR DETECTION OF "BENDS"

Robert L. Meckelnburg, MD

Since the Doppler ultrasound was first introduced for bubble detection in cardiovascular work by the Japanese worker, Taratka in 1975, many different applications have been made of this technique. One of the earliest attempts at the use of the Doppler ultrasonic flow meter for the objective detection of circulating bubbles in the blood stream of larger arteries and veins was done by Spencer and Campbell in 1968. Since that time refinements in electronics and instrumentation, coupled with improved clinical application have produced a technique of monitoring the vascular systems for the presence of inert gas bubbles that hold much promise for the study and prevention of dysbarism.

Basically the technique of Doppler ultrasonic detection is applied transcutaneously by means of a piezo-electric crystal of five to ten megahertz applied at usually ten milliwatts per square centimetre of the body surface. Utilizing a pair of focused quartz crystals in a contoured probe placed over the pulmonary arteries and veins just to the left of the sternum at the third to fourth interspace, one can monitor the entire return of the vascular system. Hence, bubbles arising in any portion of the body, and gaining entrance into the vasculature will be sooner or later passed in front of the view of the doppler transmitter and result in reflections of the ultrasound beam at the gas-water interface. With the improvement in electronics, the use of larger crystals with reduced background-to-signal distortion have allowed greater flexibility in placement of the ultrasound probe over the chest wall. The radiated area encompassed by the quartz crystals then becomes sufficiently large, so that all of the significant blood flow is examined upon its return to the heart. The presence or absence of venous gas emboli can be determined with a fair degree of accuracy. The arterial system is not monitored, since it has been determined that only under the most unusual circumstances do gas emboli ever appear on the arterial side of the vascular system. To date, a fairly consistent pattern of detection of venous gas emboli prior to the onset of any symptoms of the bends, has been recognized by most investigators using the Doppler ultrasound technique. Since many venous gas emboli can be detected by the Doppler ultrasound technique and no symptomatology whatsoever developed in the subject, it is quite obvious that the body has an extremely large tolerance of gas emboli before it produces any clinical signs or symptoms.

The application of the Doppler ultrasound, of course, would be mostly aptly applied directly on a diver during his dive. With surface or self-monitoring, one could detect the immediate development of gas emboli in the blood vascular system, and thus be able to shorten or terminate the dive. Unfortunately, present equipment and technique do not allow monitoring of divers during their dive conveniently, except in the chamber stimulations. Thus in the actual field work, the best that can be accomplished at this point is surveying the diver after he has completed his dive, and returned to the surface vessel. Even with this modified technique, it is quite important to be able to detect those individuals who do manifest a significant amount of bubbling in their blood vascular system, and who, therefore, should be kept under surveillance for the possible onset of bends symptomatology. Also, those individuals wishing to perform a repeat dive excursion can be warned away from this endeavour if their Doppler ultrasound examination shows a significant number of bubbles in their blood vascular system from their first exposure.

Concomitantly, the ultrasound detector can also be utilized as a monitoring system for individuals undergoing therapeutic recompression and staged decompression for the treatment of their bends. The elimination of the venous gas emboli can be used

as a predictor of effectiveness of treatment, and delineate how much treatment must be administered to the individual to produce therapeutic recompression. Also, it has been shown that in many instances of simple bends, a shortened procedure both in time and depth of recompression can be utilized as effectively as more prolonged methods. Typical Doppler ultrasound equipment can be made quite compact and portable, and easily utilized in the field. This makes the application on shipboard quite feasible, and limits it only by personnel and time.

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A FOUR YEAR SURVEY OF MORTALITY IN BRITISH DIVERS

GW Crockford and Mrs DF Dyer

(Paper given at Underwater Association (UK) Conference, November 1974)

In July 1970 there were 250 divers on the National Divers Register which had been started by Mrs DF Dyer. As far as can be judged at that time all 250 were obtaining the whole or a substantial part of their income from commercial diving and were making or intending to make a career of diving. An attempt is now being made to trace what has happened to these men over the past four years in terms of death and retirement in order to determine the fatal accident rate of commercial divers. The fatal accident rate can be expressed in terms of deaths per 1000 man years of exposure and a comparison then made with other sectors of industry. It is assumed for the purposes of this study that a man is exposed to the hazards of diving as long as he is available for work and has not made a definite decision to retire from active diving, irrespective of the amount of diving he was doing over the four year period.

The period covered by the study was from July 1970 to July 1974 inclusive, a period of four years one month or 1021 man years of exposure. Due to the difficulty of determining when a diver retires from active diving it was decided to assume that all 250 were exposed the whole time although it is known that 63 are not now diving. A further 43 men have not been traced and are also assumed to still be diving. Some information has been obtained on the rest of the group either by answers to questionnaires or word of mouth. As many of the fatal accidents reported to us were by word of mouth it was decided that a fatal accident would only be counted as confirmed if there were supporting newspaper reports, confirmation from the firm the man was working for, or from relatives or from research workers who are keeping medical and accident records of divers. Verbal or written reports of accidents from colleagues for which a confirmatory source has not been found to date are counted as “unconfirmed accidents”.

To aid in the task of matching divers to prospective employers the register was divided into a group of 100 widely experienced divers who were prepared to travel overseas and a group of 150 who were more specialised in their underwater skills.

The results of the study to date are given in Table 1 and it must be emphasised that these figures are only provisional until all 250 men have been traced.

TABLE 1

FATAL ACCIDENTS AMONG 250 DIVERS

	Group 1 (100)	**Group 2 (150)**
Deaths in the UK	5 (confirmed)	7 (confirmed)
Deaths outside the UK	3 (unconfirmed)	1 (unconfirmed)

The distribution of the UK deaths was 7 in association with oil and gas industry in the North Sea, 1 crawfishing, 1 inland, 1 it is believed in a harbour or estuary and 2 in association with salvage and inspection.

Expressing the results in terms of 1000 man years of exposure gives a risk rate for

the UK of 11.7. If the overseas deaths are included this rate could be 15.6.

The relationship these figures bear to other industries is illustrated in Table 2.

TABLE 2
Fatal Accident Rate per 1000 man years of exposure (UK)

Manufacturing	0.04	- 0.05
Coal Mining	0.3	- 0.4
Deep-Sea Trawling	2.0	- 4.0
Deep-Sea Trawling 1968	9.0	
Commercial diving	11.7	- 15.6 (?)

These figures illustrate a number of important aspects of the hazardous nature of diving:

1. The minimal confirmed fatal accident rate is higher than that of deep-sea trawler fishing in 1968 when the loss of 3 ships pushed the rate up to 9 and resulted in a full Governmental enquiry into trawler safety.

2. Unconfirmed deaths indicate that the true rate may prove even higher.

3. Commercial divers are at risk and are losing their lives in a wide range of diving activities in the UK and are very much at risk when working overseas.

4. Safety legislation that deals with a restricted aspect of commercial diving is of limited value to the profession as a whole or to individual divers who move between different types of work, eg. North Sea, reservoirs, harbours.

5. As the number of man hours spent by the group in the North Sea or other areas is not available the figures do not indicate that the North Sea is any more dangerous than any other areas of diving activity.

In summary it can be said that commercial diving is an extremely hazardous profession. Comprehensive legislation at both the National and International level is required if the fatal accident rate is to be reduced to a level comparable to that of other dangerous industries. Legislation covering one particular type of diving, such as offshore installations, will have a limited impact on the hazardous nature of the profession.

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UNSUITABLE SPORTS

The Education Gazette lists most sports as suitable for schools. Predictably, it does not approve of boxing, karate, tae-kwong-do, rifle shooting, clay pigeon shooting, spearfishing and flying. But just when you are thinking the list shows admirable concern for physical safety you come across another of the forbidden sports - contract bridge.

(Sydney Morning Herald, 20 July 1974)

IDLE THOUGHTS: "FILL ME UP, DOC!"

It soon becomes apparent to newcomers to diving that their problems are considerably more complex than the mere satisfaction of their keen interest in ensuring a dependable source of air to breathe. The constraints that result from the operation of the "Gas Laws" acquire a degree o£ reality when pain in ears or sinuses reinforces the text book advice regarding the need to "equalise" on descent. Unfortunately the warning discomfort that should accompany the barotrauma of ascent is too diffident a teacher for safety, pulmonary damage occurring silently and on occasion leading to the entry of air into the arterial circulation and thereby by ill luck to a vital area of the brain. As to the other problems that plague man underwater, even the non-diving public know about "the bends" and "narcosis", thanks to film and TV presentations of diving dramas. The possibility of oxygen toxicity and of bone changes is now in the awareness of many scuba divers who will in all likelihood never get within cooee of being at risk, and some degree of awareness of the dangers of hypothermia and that the use of special gas mixtures in place of air merely postpones the onset of deepdiving problems but does not obviate them is now accepted fact to many more than the small elite most closely involved in such problems. It is to minimise such adverse factors that diving routines have been designed and taught and the diving depth/time tables devised. Additionally, great thought has been given to the concoction of special gas mixtures for use in place God's good clean air for deep dives, always recognising that one buys exemption from one problem by accepting some alternative disadvantage charting a course between the competing risks of decompression sickness, inert gas narcosis, hypoxia, oxygen toxicity, HPNS, gas density, carbon dioxide problems and the joker in the pack, human variability, keeps many a mathematician happily and usefully employed with his computer, tinkering with the pros and cons of the advice to give that man-that-never-will-be, the diver whose tissue reactions fit the theories. Nevertheless, off the peg though they may be, no diver who values his health can afford to disregard the tables. Unfortunately the theoretical basis of such advice lacks adequate factual feed back from the users, for divers are notoriously poor at giving accurate details of dive profiles where symptoms have occurred. They tend to select out and report only those symptoms they think justify complaining about, the resultant lack of reporting of minor symptoms giving rise to a misleading belief in the safety of present procedures. In a "state of the art" where it is still under dispute whether the initial or final portion of the ascent is most critical regarding the prevention of the onset of decompression sickness, the lack of accurate information concerning whether the divers are truly symptoms free if they follow calculated diving patterns is becoming recognised as being a brake on progress. How nice it would be if one could do without air filled spaces, eliminating all barotrauma, and avoid the use of any (so ill named) "inert gases" and thereby obviate most of the remaining problems. To reduce the problems of diving to the avoidance of hypothermia and the oxygen-in/carbon dioxide-out balance would be an immense gain. Man would gain vertical freedom of the sea also. Naturally it was only a matter of time before someone came along determined to put theory to the test and starting with small mammals began the long slog of redesigning Homo Sapiens into the mould of a Homo Pisces.

One of the first to make a scientific attempt to solve the problems and to actually "aquetise" a mammal was Dr Johannes Kylstra. Naturally our furry friends provided the volunteers for this first great step towards joining the fishes rather than merely visiting them, and their survival underwater under hyperbaric conditions led to further experiments and the use of more complicated compounds than the initial simple isotonic solutions of salts. The degree of progress made will encourage further

investigations and offer the expectation that one day man (?) will enter into an exciting and totally unchartable dimension of the environment. The only certainty is that the unexpected will occur and that hitherto non-existent problems will arise to cause disability, diseases, and deaths. But think of the chances for undying fame for those who are credited with first describing the eponymous troubles of this new men!

No amount of wishing and flooding can convert lungs into gills, their respective anatomical structures dictating inverse relationships between tissues and the respiratory fluid. While the tidal ebb and flow of a relatively low density gas with good diffusing properties and minimal eddy turbulence at normal respiratory rates through the Bronchial tree provides for good gas exchange function at low energy cost, problems escalate immediately gas density or respiratory rate is significantly increased. Normal respiration is by moderate expansion of the thoracic cage and then elastic recoil, this being about the neutral point of thoracic size. The accessory muscles of respiration are “designed” for bursts of action and not for prolonged use, consequently they tire easily and are unsuitable for indefinite ventilation of the tract by dense fluids, be they gaseous or liquid. The energy cost can rise such that the oxygen requirements of continued forced respiration equal or exceed the respiratory intake. The supply is limited because range of safe oxygen partial pressures is narrow and at high ambient pressures a minor change in gas composition in the mixture causes marked partial pressure change, with values both above and below the allowable range being dangerous. Mechanical assistance to thoracic movements would therefore become essential. So far no solution has been found for the problem of efficiently removing carbon dioxide from the alveolar capillaries by way of intra-alveolar liquid, though “clearing” the liquid outside the body of such carbon dioxide as has managed to cross over would be relatively simple. The efficiency of the pulmonary system for fluid based respiration would be further reduced by the likely circulatory preference for any areas of collapse or consolidation that resulted from such unphysiological demands on its functioning. Gills, by way of contrast, rely on a through flow of respiratory fluid over the gas exchange tissues. Such a mechanism would appear to be the most rational one when using liquid, a conclusion reached by marine life some time ago. It is therefore hoped that no benefactor of mankind will finance human trials of this approach to producing a barotrauma-proof man.

Even though the pulmonary tract may be considered as unsuitable for liquid based respiratory exchanges it may still be advantageous to fill all the body spaces with some liquid. It will have to be one that is not only inert but can never produce any foreign-body, auto-immune or neoplastic changes. There are enough graves already occupied by victims of perfect drugs and inert inserts. It is essential that rapid and complete drainage with unimpaired return of function be possible, for fame and fortune would be dear indeed if one had to visit landbound friends in a kind of goldfish bowl! Still, as all of us can claim to have logged abut nine months of “fluid time” before our first breath, it is not innately against nature to “mothball” our lungs for a time.

This still leaves the problem of what alternatives there are (besides the one of forgetting the whole matter), what other routes can be utilised. Any straightforward gas exchange through the skin is definitely a non-starter because ambient pressure would make oxygen toxic to the tissues of the exchange area even supposing that adequate absorption was theoretically possible, which is extremely doubtful. There would still then remain the problem of removing the carbon dioxide, almost certainly impossible through any skin area though drugs could be considered to utilise renal excretion, a last (sic!) resort. It therefore appears to be inevitable that some liquid be used. The most obvious and at hand is, of course, blood.

The use of some adaptation of a heart-lung machine at first sight appears attractive, for the extra-corporal circulation will require mechanical assistance and there is already considerable expertise available in its use. It is known that such machines cause damaging changes to the blood, both the cellular and non-cellular constituents being altered. As a major artery and vein would be necessary for such a machine the preparatory surgery would pose some problems for the diver even if not daunting to the surgeon. It is accepted that the engineering and monitoring hardware made necessary by any of the postulated changes can be designed, the problems arising from the attempts to make-over H. Sap. into a more fashionable underwater version. Almost certainly a multitude of parameters will need to be checked because the normal body functions depend on such a large number of little understood and inter-related checks and balances and feedbacks and cerebral cortical over-rides that a computer will be necessary. As one example, the dive reflex reduction of peripheral circulation is removed by the action of breathing: but there won't be any "breathing". Over the ages mankind has probably got buried in its psyche quite a firm desire to breathe. It will really be quite a game of spillikins, with such problems as the straws and life and health as the stakes. Care for a game?

An alternative approach would be either to leave the blood to do its own thing and let the respiratory liquid remain separate, or the blood could be removed and stored and the liquid utilise the vasculature made vacant. In the former case the liquid could make gas exchanges through specially prepared skin areas, either skin folds or highly vascular skin surfaces, such being de-facto new gills, or the liquid could be circulated through the peritoneal cavity. The membranes are here resistant to infection to a high degree, require no preparation and offer a considerable surface. Or one could implant exchange membranes subcutaneously with plug attachment to allow ready conversion from land to water modes of life. The replacement of the blood would require much further investigation and possibly prove too ambitious and costly a venture at the present time, for the blood has very many functions in addition to that of the oxygen/carbon dioxide equation. One shouldn't be too ambitious with first steps.

This idle chatter may have started you thinking of the points, so obvious to you, that I have missed. So please write in your suggestions. For myself, I think I'll follow the advice of the "Beatles" and get a yellow submarine.

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SPUMS MEETING AT PRINCE HENRY'S, MELBOURNE
ON FRIDAY OCTOBER 31ST 1975

With the President, Carl Edmonds, in the chair the meeting started within minutes of the advertised time of 9.30 am. The first speaker was John Knight who spoke on "Oxygen Toxicity". He managed to baffle his audience with science and fielded all the questions with aplomb. Gavin Dawson then spoke about the clinical indications for the use of hyperbaric oxygen in the treatment of carbon monoxide poisoning and gas gangrene. He briefly referred to less clear cut indications such as Raynaud's phenomenon, with which he has had the occasional success. His review of the results in his 90 odd patients treated in the single man Vickers oxygen chamber was interesting and informative.

During the tea break the audience went in relays to watch Bill Refish being a patient under 2.5 ATA of oxygen. It must be a noisy experience being shut in that perspex cylinder as a constant wind whistle came over the intercom.

Gordon Donnan described the radiological aspects of dysbaric osteo-necrosis and gave a most interesting clinical history as follows. In the early 1950s a man was diving in about 200 feet in Lake Eildon when his air hose broke. Naturally enough he surfaced in a hurry. Soon after surfacing he got pain in his limbs and collapsed semi-conscious. He was taken home and remained semi-comatose for some three or four days. He had no treatment for his bends. Up till that time he had been quite well but after that episode he had pain in his shoulders and hips, not all the time just occasionally. Some years later he was deep diving on air in Bass Strait working on an oil rig when again he had to ascend rapidly. Now he has obvious dysbaric osteo-necrosis of both shoulders and one hip, which joint is almost completely destroyed. Geof Macfarlane then gave a fascinating resume of the work of divers in Bass Strait. The most hair-raising job he described was the man who sits on the sting and guides, through a microphone to the barge captain, the pipelaying barge onto the pipe already on the bottom. The sting is a huge tubular scaffold hinged to the barge through which the lifting cables attached to the already laid pipe pass. The taut cables lift the pipe from the sea bed and the barge is backed onto the pipe by winching in on its stern anchors. One mistake and the sting can be torn like paper although the sting is made of 20" steel piping. Just imagine sitting on top of the sting while the cables vibrate under the strain of lifting and visibility is about three feet. The whole process is rather like the old story of the soldier with TB meningitis having his course of intra-thecal streptomycin. His usual doctor was on leave and the substitute was having terrible trouble finding the vertebral canal. After many tries the soldiers said over his shoulder. "Sir, would it be easier if you held the needle steady and I backed onto it?". When Geof had finished everyone had a much clearer idea of what oil rigs look like at the various stages of their construction and the various jobs that the divers have to do and the precautions, such as recompression chamber and their back up facilities, needed to care for the divers.

Lunch was provided through the kindness of Prince Henry's Hospital and the those who could spare the afternoon went out to the Melbourne Metropolitan Board of Works medical lock (recompression chamber) at Braeside. Here we were met by Arthur Keech, medical officer to the MMBW, and Laurie Brennan of the chamber attendants. Considerable confusion ensued between tunnellers and divers. Increasing pressure is going down to divers and going up to tunnellers. Divers think in feet of seawater (but they should really think in metres) and tunnellers think in PSI Gauge. They will soon have to convert to Pascals. The therapeutic chamber makes any Navy chamber look small. After a very welcome afternoon tea during which tunnelling operations were clearly explained and the need for compressed air to stabilise sandy soil as well as to keep out water explained the party went off to visit a tunnel so to see the progress at work. However there was no visit to a compressed air working.

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The meeting attracted about 35 people to Prince Henry's Hospital. A pleasant mixture of SPUMS and other interested people. At least four had come interstate specifically for the meeting and a further contingent had remained in Melbourne following the AGM of the Australian Society of Anaesthetists to attend the meeting. A pleasant and well worth repeating feature of the meeting was the buffet dinner (subscription) held the night before at John and Gill Knight's home. The last guests left about 1 am. so they had enjoyed themselves.

As Carl said in his opening remarks "It is not only Sydney that has enough expertise to put on a meeting." The success of the first non-AGM scientific meeting should encourage other cities to try to outshine Melbourne. Your reporter hopes that the Editor will be able to persuade the speakers to forward their presentations for publication.

ANON.