to Sydney. He couldn't even swim, let alone dive. So he had a crash course of swimming lessons before coming to Sydney. Now, they say, he's almost out'Shaneing Gould.

(*Daily Telegraph*, 3 May 1974)

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SOCIETY NOTES

The Extraordinary General Meeting voted overwhelmingly to pass the proposed change in the Constitution of SPUMS. There is no longer any bar to candidates on ground of domicile. Does this effect YOUR willingness to stand for the Committee?

To assist candidates for the Diploma, a list of Basic Reading books is now being prepared and will be published when available. Preparatory study will be essential to enable full benefit to be obtained from the course at SUM and Prince Henry Hospital Hyperbaric Unit. No Know, No Pass.

The intention to publish this Newsletter quarterly is dependent on articles being presented on time. No Write, No Read.

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MICRO AIR EMBOLISM AND LUNG ANATOMY
(Address to "Oceans 2000" October 1973)
Prof. Dennis Walder

I want to say something about micro air embolism. In Great Britain we have come to classify decompression sickness as being of two types Type 1, otherwise known as "the bends" in which there is pain in a limb but no constitutional upset. The pain may be very severe but the subject is not ill, doesn't look ill, and doesn't feel ill. Type II may be present in one of several ways as a disturbance of the central nervous system with paralysis and/or loss of sensation, usually in the limbs as a disturbance of the cardiovascular system with pain in the chest similar to that which occurs following a coronary thrombosis or as a disturbance of the respiratory system with difficulty in breathing and an associated blue appearance. This latter condition is known as the "chokes". In all these type II forms of decompression sickness there is a constitutional upset. The subject looks ill, feels ill and unless something is done quickly he may suffer permanent damage, or even die.

As you know, classically decompression sickness is thought to be due to the presence of bubbles in the body. It is said that these bubbles arise because the decompression procedure has been too fast and sore of the body's tissues have been left with an excessive amount of gas which has come out of solution in the form of bubbles. The longer and deeper the dive the more gas will be taken into the tissues and the greater will be the danger of decompression sickness. Conversely, short shallow dives in which very little gas enters the tissues wall be safe, a fact recognised by the existence of no-stop decompression schedules.

Occasionally, however, a diver who has only carried out a short dive at shallow depth,

say for a few minutes at 30 feet, will develop the signs and symptoms of decompression sickness. This can cause consternation and disbelief unless the mechanism by which this condition can cause consternation about is appreciated.

The suggested explanation is that when a diver is at depth a small pocket of air becomes trapped in his lungs and on the subsequent decompression when this pocket expands it bursts into his circulation as a stream of bubbles which give rise to the signs and symptoms Type II decompression sickness. This could be called micro air embolism because it is different from the situation which occurs when a man carries out a free ascent and inadvertently keeps his glottis closed. In this case the air in his lungs is trapped and when it expands it builds up a pressure till it finally bursts through into the circulation to give rise to a massive air embolism. The two situations are quite different as the former can occur in a diver who is returning normally to the surface after a shallow dive and the signs and symptoms are indistinguishable from Type II decompression sickness.

When we examined the lung radiographs of men who suffered from such unexpected Type II decompression sickness we sometimes saw something very interesting. The men had cysts in their lungs. The condition obviously required close investigation. It transpired that small animals undertaking short shallow simulated dives never suffered from cysts in the lung and this gave us a clue. The structure of man’s lung is different from that of an animal.

In both man and animals’ the lungs have a branching system of airways which, like the branches of a tree, conduct the breathed gases to little air sacs which are arranged on the branches like the leaves of a tree. It is at the air sac that the exchange of gases with the blood takes place. The whole lung structure is covered over by a membrane which is called the pleura. Man differs from animals in that here and there the pleura dips down between the groups of air sacs to form partitions. These partitions contain some loose material which enables their two sides to slip one against the other during small movements of the lung. At the extremity of each partition runs a branch of the thin walled pulmonary vein.

When a diver is at depth all the air sacs are filled with gas at the same pressure as the breathing gas. As the pressure of the breathing gas is reduced during the subsequent decompression the gas in the air sacs will normally vent freely through the airways. If, however, some branch airway becomes blocked by, for instance, a viscid blot of sputum the gas in the air sac served by that airway will not be able to vent and will therefore distend. After a few feet of ascent there will be some air sacs that are distending and some that are remaining the same size because they are able to vent in the normal way. As a result of this a shearing force will be set up along the partition and eventually the tissues will rupture and tear. The tear will involve both distended and non-distended air sacs as well as the thin-wailed vein. As a result of this, air will enter the vein and be conducted to the heart, from which it will be pumped round the circulation to give rise to the signs and symptoms of Type II decompression sickness.

Experiments with isolated lungs using various differential pressures between adjacent groups of air sacs have shown that this is a reasonable explanation of what takes place. Pressure differentials of as little as 2 psi, the equivalent in diving depth change of as little as 4 feet, can result in lung damage and micro air embolism.

There is an important practical implication of these findings. It means that if you go diving when you have, or have recently had, a cold, influenza or an attack of bronchitis, which has left you with some viscid mucus in your chest, then you are in danger of getting an unexpected attack of severe Type II decompression sickness. All the men I have known who have suffered from an unexpected attack of Type II decompression sickness have been found on enquiry to have had a cold in the week or ten days prior to the dive and had presumably returned to diving before their lung mucus had had time to return to normal.

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Brief Profile

Prof. Dennis Walder is the leading expert in Britain on bends and compressed air problems. He is Chairman of the Society for Underwater Technology diving technology committee, President of the Undersea Medical Society and of the European Undersea Bio-Medical Society and Chairman of the Medical Research Council Decompression Sickness Panel. He is based on the Department of Surgery, University of Newcastle upon Tyne.

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NINETY SECOND DEEP SCUBA RESCUE

GD Harpur, MD

(NAUI News January 1974)

It is proposed in this study to review the problems posed by the unconscious diver and to present solutions available to the diver attempting rescue.

The unconscious diver has ceased to breathe. There may be several reasons - depletion of supply, contaminated supply, equipment malfunction, the little appreciated danger of cold, or other medical problems. He will have lost his regulator, and his oxygen stores rapidly deplete. Unless he, or a rescuer is able to replenish these, he will die.

Illustration No. 1 shows a comparison of oxygen consumption with elapsed time in a non-breathing subject whose heart is still beating. Depending on the reason for his loss of consciousness he is left with a certain amount of time until he will have sustained irreversible damage to his brain and only a little longer before he will die. At the point in time when his arterial pO_2 drops below the level of 40 mmHg, (normal level = 80-90), his consciousness will be very much impaired. The time remaining after this until his pO_2 arterial drops to levels which will result in a permanent alteration in the diver's central nervous system is approximately 90 seconds. This does not mean that anyone found on the bottom known to have been down in excess of this time should be abandoned or handled differently, but attempts to point out that we should aim at developing a rescue technique which will take less than 90 seconds.

ILLUSTRATION NO. 1 (page 18A)