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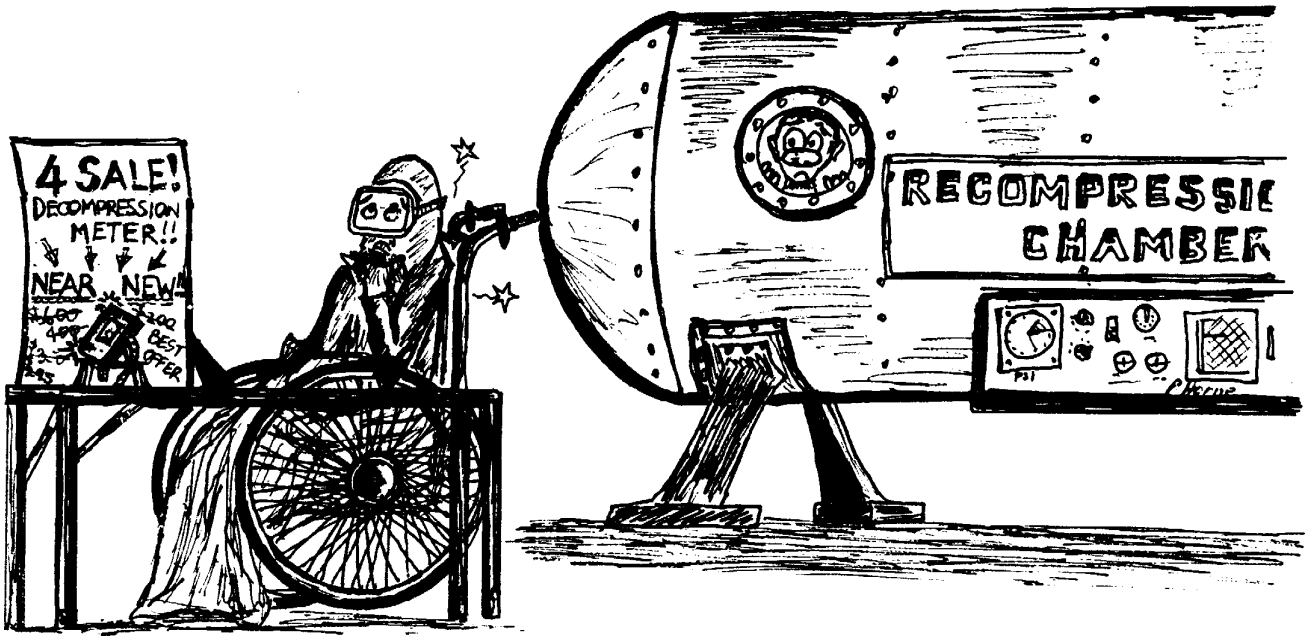

CONTENTS

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To provide information on underwater and hyperbaric medicine.
To publish a journal.
To convene members of the Society annually at a scientific conference.

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EDITORIAL

There are many things which are unattainable at this time and appear likely to remain so. They include elucidating the Meaning of Life, the discovery of an Elixir of Eternal Youth (especially one with a Guaranteed Health option !), learning how to circumvent Murphy's Law and acquiring a Decompression Meter which allows the wearer to venture underwater repeatedly for as long, and as deep as he or she may desire without the need to spend time decompressing or the risk of decompression sickness (DCS). It is the last named unattainable that seems to be attracting the smart money and brilliant minds in the recreational diving industry, which seems likely to crack and allow a breatkthrough. For this optimistic appraisal credit must be given to a belief that microchips and sophisticated computer programs can, and do, lead to the solving of all problems, which are tackled in a resolute manner. Divers seem to have a belief in the magic of science rather than an appreciation that Nature's Laws are what govern all life processes.

Alas for such hopes. Even if divers of the future prove to be better at obeying all the good advice they are given than are the divers of today, there can never be a complete guarantee that a dive cannot result in some decompression-related problem even when following any of the decompression tables blessed by navies and other authorities. Even the most recent tables come with a "No Guarantee" tag, though this is by convention in the traditional unwritten small print. This is a result of the unfortunate (in this context) fact that every living person differs unpredictably in some unknowable physiological ways from every other, and indeed varies from day to day. Such problems naturally can be engineered out of machines so that they only suffer decompression problems if they have developed some physical fault. However for humans it is an innate problem. Of course careful calculations give a vital and reasonable guide to the avoidance of DCS but it is human nature, or at least the nature of many divers, to seek out tables offering longer and deeper dives with less decompression time debit. It would be interesting to discover what such divers understand about DCS which allows them to believe that the use of a different piece of printed advice will alter the way in which their bodies will out-gas.

Father Brown, a creation of GK Chesterton, once said "It isn't that they can't see the solution. It is that they can't see the problem" and this is true of much of the work being done to produce and market machines which offer divers longer underwater times with a lesser need for decompression. As the present Tables have a failure rate only an incurable optimist will expect an increased exposure time to result in a decreased nitrogen uptake. Some day, no doubt, some clever pharmacist will discover a drug which postpones out-gassing by increasing the gas binding by the tissues, in effect removing the so called "fast tissues" and allowing what would today be regarded as supersaturation. This will be hailed as a great discovery. One thing is certain, such an "advance" would soon be followed by a rash of medical papers recording new diving illnesses. All this is by the way of indicating the importance of the work of Carl Edmonds and Tim Anderson (page 119) and warnings of Des Gorman and David Parsons (page 119). As a bonus there is the paper by Gorman, Browning, Parsons and Traugott on the important subject of arterial gas embolism (page 101).

Diver education has been very greatly improved over recent years. The public requires that their instructors be skilled at teaching and the major instructor organisations have become aware of the value, both for commercial and legal reasons, of high standards. However at present there is no regulation or control of the medical element in the drive to increase diving safety. While the discipline of diving medicine is the more healthy for containing persons having differing interests and opinions, readers are advised to conbsider the implications of that sage advice, Caveat Emptor,and read the waming notice below.

WARNING NOTICE

A notice advertising a "Medical Diving Conference" to be held in the Maldives in 1988 has been circulated to many doctors. There is some valid concern about certain statements prominently printed in this circular which are either misleading or untrue.

It is stated that the meeting is "Sponsored by SYNTEX Australia, Ltd." but this is completely false. According to a spokesperson in the advertising department of SYNTEX in Sydney "the only involvement by SYNTEX has been to supply (sell) a list of doctors' names and addresses to the travel agent. SYNTEX neither authorised nor was aware of their being named as a sponsor until contacted". While this reflects poorly on their sense of responsibility in a commercial matter it does confirm the inaccuracy of the circular.

It is stated that "Tuition is by the Professional Association of Diving Instructors International". This is misleading as PADI (Australia) has neither been approached to support, nor had any prior knowledge of, this conference. The statement should read that "there will be a PADI certificated instructor available", which is a rather different matter. The circular states that "You have been specially selected by SYNTEX Australia Ltd., to attend the first ever Australian Medical Diving Conference". This statement is patently untrue on several counts.

It states that "the conference you will be attending consists of both practical and theoretical diving tuition and medical diving lectures. Also paper presentations". No details of the medical program are given and none are known to the travel agent who is circulating this material. A claim is made that the Taxation Department will be certain to allow 50 % of the cost of attending the conference, a statement open to doubt as no course content has been published to demonstrate the value of this course as a valid source of information on diving medicine. It certainly would not be accepted, on the evidence available, as the basis for an attendee to claim to be qualified to conduct "diving medicals".

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INSTRUCTIONS TO AUTHORS

Contributions should be typed in double spacing, with wide margins, on one side of the paper. Figures, graphs and photographs should be on separate sheets of paper, clearly marked with the appropriate figure numbers and captions. Figures and graphs should be in a form suitable for direct photographic reproduction. Photographs should be glossy black and white prints at least 150 mm by 200 mm. The author's name and address should accompany any contribution even if it is not for publication.

The preferred format for contributions is the Vancouver style (*Br Med J* 1982; 284: 1766-1770 [12th June]). In this Uniform Requirements for Manuscripts Submitted to Biomedical Journals references appear in the text as superscript numbers.[1-2] The references are numbered in order of quoting. The format of references at the end of the paper is that used by *The Lancet*, *The British Medical Journal* and *The Medical Journal of Australia*. Page numbers should be inclusive. Examples of the format for journals and books are given below.

1 Anderson T. RAN medical officers' training. *SPUMS J* 1985; 15(2): 19-22.

2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: JL Publications, 1985.

Abbreviations do not mean the same to all readers. To avoid confusion they should only be used after they have appeared in brackets after the complete expression, eg. decompression sickness (DCS) can thereafter be referred to as DCS.

Measurements should be in SI units. Non-SI measurements can follow in brackets if desired.

PROJECT STICKYBEAK

This project is an ongoing investigation seeking to document all types and severities of diving-related incidents. Information. all of which is treated as being CONFIDENTIAL in regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organization to increase diving safety through better awareness of critical factors. Information may be sent (in confidence) to:

Dr D Walker
PO Box 120
NARRABEEN NSW 2101

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This contains further papers presented at the ANZICS-SPUMS Meeting in Rockhampton in October 1983.

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This contains papers from the 1985 Annual Scientific Meeting in Bandos and from the New Zealand Chapter of SPUMS Meeting in November 1985, including an account of the formation of the New Zealand Chapter.

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SPUMS ANNUAL SCIENTIFIC CONFERENCE 1986

The following paper covers the topics discussed by Dr Des Gorman during this conference

THE DISTRIBUTION OF ARTERIAL GAS EMBOLI IN THE PIAL CIRCULATION

DF Gorman,[a] DM Browning,[b] DW Parsons,[a] FM Traugott[c]

INTRODUCTION

A. The natural history of cerebral arterial gas embolism

Once introduced into a large artery, gas emboli will distribute according to their buoyancy, such that the cerebral circulation is embolised in humans and experimental animals placed in a head-up (upright) position.[1-3] A head-down (inverted) position protects the cephalic circulations.[3] Because of their upright posture during ascent, divers and submariners with arterial gas embolism (AGE) usually present with neurological symptoms and signs consistent with cerebral arterial gas embolism (CAGE).[4-8]

Before any study of CAGE treatment can be undertaken, the natural history of gas emboli in the cerebral circulation must be documented. Although it has not been shown directly, it is assumed that the gas emboli that arise during decompression occur as a result of pulmonary over-inflation and direct embolism of the pulmonary veins.[7,9,10] Such emboli may be coated with surfactants,[11] and this may alter subsequent events in the cerebral circulation.

From available studies it would appear that regardless of the form in which gas is introduced into the arterial circulation, coalescence of small emboli will create cylindrical gas columns.[11,12] It also appears that the larger the embolus, the more likely that it will lodge in a small arteriole and block blood flow.[12-15] The conventional pathophysiological model of CAGE is based on the physical blockage of a cerebral arteriole by gas,[9,10,12-15] and assumes that the observed regional brain ischaemia,[12,17-20] platelet accumulation,[21] thrombi formation,[21-25] and increased blood-brain barrier (BBB) permeability[15,19,20,26-30] are secondary to this blockage.

However, researchers using cranial windows to observe pial gas embolism[13,14,16] have obscured the natural history of the emboli by compressing their experimental animals in a recompression chamber (RCC). The potential for the results of these studies to be misleading is demonstrated by work with several animal models not incorporating a cranial window.[3,12,18,31] In these models gas emboli have been shown to spontaneously redistribute from the cerebral arteries to the jugular veins,[3,12,18,31] to the right ventricle, and to the pulmonary arteries.[12,31] It is not known what proportion of gas emboli entering the cerebral arterioles undergo such redistribution, nor is it known what effect they may have after redistributing. It is even possible that gas emboli may only lodge in cerebral arterioles temporarily, eventually redistributing to the venous circulation. The last possibility is a plausible explanation of the large number of human patients with CAGE that experience some resolution of symptoms and signs prior to any treatment.[32,33]

Almost all of the animal models of CAGE on which the conventional model is based, have involved the direct injection of gas into a carotid artery.[13,14,16] With the single exception of carotid artery surgery,[34] these vessels are not the usual source of arterial gas emboli. Also, it is known that the carotid artery gas infusion techniques employed by these researchers can avoid embolism of the brain stem circulation.[3,12-14,18,24] Gas embolism of the brain stem causes cardiac dysrrhythmias,[3,9,18,31,35-42] respiratory depression,[3,12-14,18,31,43] and an increase in arterial blood pressure[3,9,12-14,18,31,37-39,43] that exceeds the limits of cerebrovascular autoregulation.[42,44,45] The result is a significant increase in cerebral blood flow (CBF),[42,44,45] which will have a major influence on the passage of gas emboli through the cerebral circulation. This casts further doubt on the animal model data from which the conventional pathophysiological model of CAGE is derived. It follows that the natural history of gas emboli in the cerebral circulation is yet to be described.

B. The factors that could influence the passage of gas emboli through the cerebral circulation

The arrest of a region of the cerebral circulation, as a consequence of a gas embolus lodging in a small arteriole, will only occur if the forces that oppose embolus movement exceed the local cerebral perfusion pressure (CPP).[9,13]

The forces that oppose embolus movement increase as the length of the embolus increases.[14,19,22,31] The length of a gas embolus is a function of both its volume and the diameter of the vessel it occupies.

Local CPP is an interaction of mean arterial blood pressure (MABP), the level of cerebrovascular resistance (CVR), and the intra-cranial pressure (ICP).[46-50] The interaction of these factors is made more complex by the variation of CVR with MABP, such that CBF remains constant over a range of arterial pressures.[51-53] This latter phenomenon is called cerebrovascular autoregulation.

The infusion of gas into the carotid or vertebral arteries can inhibit this autoregulation, so that increases in MABP are accompanied by increases in CBF.[42,44,45] Accordingly,

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b Springwood Medical Centre, New South Wales
c Department of Anaesthesia, Royal North Shore Hospital.

CPP will vary with MABP, and the progress of gas emboli through the cerebral circulation will be directly influenced by the MABP.

The infusion of gas into the carotid or vertebral arteries, or into the aorta or pulmonary veins can cause a transient, but significant increase in MABP.[3,9,12-14,18,31,37-39,43] Embolism of the brain stem circulation appears necessary for the typical hypertensive response to gas emboli.[3,12-14,18,24] Because autoregulation of CBF is lost,[42,44,45] the transient hypertension that accompanies AGE will itself promote spontaneous redistribution of emboli from cerebral arterioles to the venous circulation.[3,12,18,31]

C. Aims of the studies

A series of studies were conducted with a rabbit animal model of CAGE to describe the natural history of gas emboli in the systemic circulation, and in particular in the cerebral circulation. The studies also aimed to identify the factors involved in the passage to these emboli through the cerebral vessels.

Methods

New Zealand (NZ) White Rabbits, of either sex, weighing between 4 and 6 kg were used in all experiments. This species was chosen because the behaviour of their pial arterioles has been shown to parallel that of intra-parencyhmal brain vessels of similar size[54] because the behaviour of their pial arterioles is not affected by being exposed in an open-brain preparation,[54] and because the modulation of cerebral vessel reactivity by changes in blood pressure persists in this species despite halothane anaesthesia.[55,56]

Pilot studies demonstrated that halothane was the only available anaesthetic that enabled both a steady-state of anaesthesia and prolonged survival after CAGE. The minimum alveolar concentration (MAC) for halothane in the NZ White Rabbit is well established.[55,56] The pilot studies also demonstrated that accurate measurements of pial arteriole diameter after gas embolism required exposure of the brain as an open-brain preparation.

Tracheal preparation
Following induction of anaesthesia in a perspex anaesthetic box, a tracheostomy was created. This was intubated with a size 3 or 4 cuffed endotracheal tube.

The cuff was inflated with isotonic saline. The tube was sutured into position, and connected to a respiratory circuit that included gas cylinders, pressure regulators, calibrated rotameter flow meters, and a Fluotec Mark 2 halothane vaporiser.

Fresh gas was delivered to the circuit at 4 L/minute to prevent re-breathing. The arterial and cerebral venous oxygen and carbon dioxide tensions were regularly monitored.

The halothane vaporiser was set at 1.5 % (11.62 + 0.01 (SD) mm Hg vapour pressure).

Jugular Venous Preparation
The left jugular vein draining the cranial contents was isolated by dissection, cannulated, and connected to a heparinized loop that included a graduated air trap (with gas collected over water). The loop was reintroduced into the jugular vein at its distal end with a second cannula. A 3-way tap was incorporated into the loop to permit both intravenous infusion and collection of venous blood samples. These were analysed for oxygen and carbon dioxide levels with a Radiometer ABL 30 blood-gas analyser using appropriate temperature corrections.

Body Temperature Maintenance
The rabbits' rectal temperature was maintained between 37.5°C and 37.8°C with a variable-output heat pad.

Electrocardiogram Recording
Electrodes were implanted in the rabbit's chest and limbs to provide a continuous ECG record on a Neotrace 8-channel recorder.

Femoral Artery Preparation
The right femoral artery was isolated by dissection, cannulated, and connected via a 3-way tap to a Bell & Howell pressure transducer (with pressure displayed on a chart recorder), and to an infusion line. Infusate was warmed in a heated coil bath to 37.5°C. Arterial blood samples were analysed for oxygen and carbon dioxide levels with a Radiometer ABL 30 blood gas analyser using appropriate temperature corrections. Venous drainage from the right leg was occluded by ligature.

Cranial Preparation
A parieto-occipital craniotomy of approximately 2 x 3 cm was created with a high-speed drill. Removal of the dura enabled observation of the brain and pial vessels with a Zeiss dissecting microscope. The microscope had a magnification range of 125 to 800 times, and had both video (Sony DXC150P) and still camera (Contax 35 mm) attachments. A tape dam with a central channel was built at the posterior aspect of the craniotomy to allow and control the outflow of cerebrospinal fluid (CSF).

The exposed brain was also bathed with warmed (37.5°C) and humidified gas mixtures, that were designed to replicate brain tissue partial pressures of oxygen (PO_2) and partial pressures of carbon dioxide (PCO_2) under the various experimental conditions. These mixtures were applied as a 1.5 L/minute diffuse jet into the craniotomy.

Gas Infusion Technique
Gas was introduced into the femoral artery as microbubbles of less than 200 μm diameter. This was achieved by infusing gas at a controlled rate (0.2 ml/sec) through an orifice of 0.025 ml internal diameter.[57] Three ml of normal saline were then infused at 0.1 ml/second to clear all gas from the arterial line.

Measurement of Pial Arteriole Diameter
A pial arteriole with an external diameter between 50 and

200 μm was selected for measurement. The pilot studies demonstrated that this size of arteriole trapped gas emboli.

The segment of the arteriole where the measurements were performed was fixed at the intersection of the microscope occular cross hairs. The microscope was locked in position except for the vertical focus control. Measurements were only performed at optimal focus, such that the distance from the segment being studied to the lens remained constant.

The external arteriole diameter was calculated as the mean of 6 measurements performed on 3 successive photographs and 3 successive still frames of the video sequence. All photographs and video sequences were recorded at 500 times magnification. The diameter was measured with calibrated metal calipers (measurement error <1%). Because of the large diameter range of arterioles studied (50200 N-m), changes in diameter were recorded as fractional changes referenced to the original diameter.

The type of photographic film and video tape, exposure time, and shutter speeds, were unchanged throughout the course of the experiments.

Procedure

Twenty seven rabbits were anaesthetised with either air and halothane (10 rabbits) or oxygen and halothane (17 rabbits). Throughout all of the experiments the rabbits remained within their physiological range (when breathing air and oxygen) for both the arterial PO_2 and PCO_2.

The 27 rabbits were divided into 5 groups; each of 5 rabbits, with the exception of Group Five which had 7 rabbits.

Group One was used to observe the distribution of arterial gas emboli, and the consequent cardiovascular and respiratory effects.

Group Two was used to determine the effect of posture on the distribution of arterial gas emboli.

Groups Three and Four were used to determine the effect of altered gas solubilities on AGE.

Group Five was used to determine the effect of lowered surface tension pressure on embolus distribution, both because gas foamed in detergent has been used by previous researchers to stabilise emboli,[13,14] and because gas emboli arising during decompression may become coated with pulmonary surfactants.[31]

Group One

Five rabbits breathing air were each bound in an upright posture to a tray that was fixed at 45° to the horizontal. Five ml of normal saline were infused into the femoral artery in a manner identical to that described for gas infusions. This was followed by repeated 5 ml air infusions, as microbubbles, given at 2 minute intervals until a gas embolus became trapped in a pial arteriole which was under observation on the brain surface. If the embolus was only trapped temporarily, and subsequently redistributed from the pial arteriole, further gas infusions into the femoral artery were performed. Infusions were repeated until another embolus became trapped. Only when an embolus remained trapped after MABP had returned to pre-infusion levels was local pial circulatory arrest assumed. The size (length and diameter) of emboli that became trapped was recorded, together with systolic and diastolic blood pressure, MABP, heart rate, ECG, and respiratory rate. Air was allowed to escape from the jugular venous loop into the graduated air trap. The volume of air collected was recorded.

Group Two

Five rabbits breathing air were bound in an inverted posture to a tray that was fixed at 45° to the horizontal. Saline and gas infusions were performed as in Group One. The procedure was abandoned if either pial circulatory arrest was caused by trapped gas emboli, or if the total amount of gas infused into the femoral artery exceeded 15 ml. If pial gas embolism did not occur in these animals, they were changed to an upright posture, again at 45° to the horizontal. If after 5 minutes pial embolism had not occurred, further 5 ml air infusions, as microbubbles, were made into the femoral artery in an identical manner to that in Group One. The rabbits were monitored as described for Group One.

Group Three

These 5 rabbits were subjected to an identical procedure to that described for Group One, with the single exception that they breathed oxygen throughout the experiment.

Group Four

These 5 rabbits were subjected to an identical procedure to that described for Group One, with two exceptions. First, they breathed oxygen throughout the experiment, and second, they had oxygen rather than air microbubbles infused into their femoral arteries.

Group Five

These 7 rabbits were subjected to an identical procedure to that described for Group One, with two exceptions. First, they breathed oxygen throughout the experiments, and second, they had an air foam rather than natural air infused into their femoral arteries. The air was either foamed in a 3% Teepol solution (5 rabbits) or in a homogenized lung preparation (2 rabbits). The latter was prepared by homogenizing lungs extracted from healthy rabbits, and used within 20 minutes of the death of the donor rabbit.

Results

General Observations

Gas introduced as microbubbles into the femoral artery of an upright rabbit caused pial gas embolism in the field of view, with cylindrical columns of gas entering the arterioles. The size distribution of these emboli is displayed in Figure 1 (page 104). The proximal blood-gas interface pulsed with each cardiac systole. The pulsations were damped by the gas and were not observed at the distal interface. No

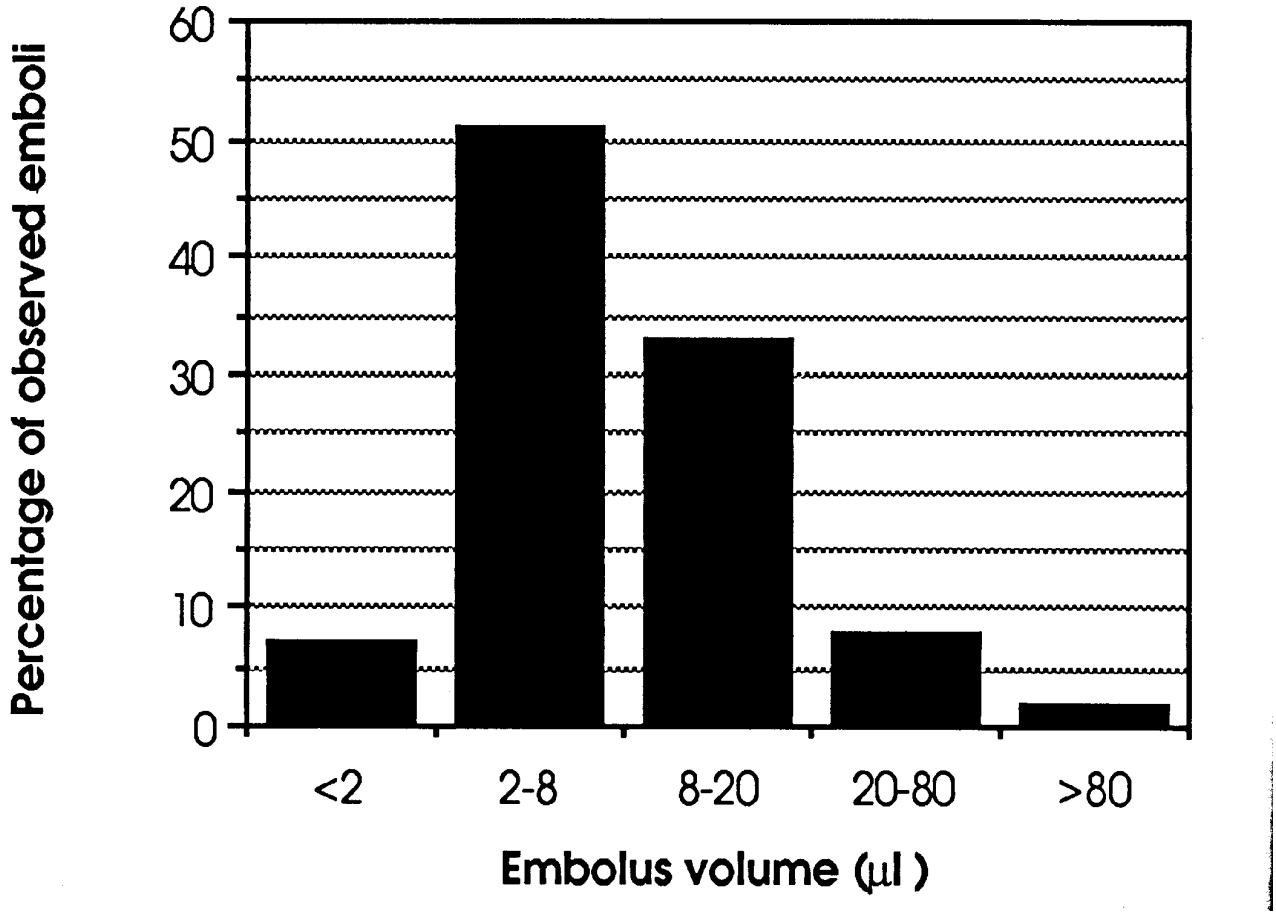

Figure 1. The size-distribution of gas emboli observed in the pial vessels of the 10 rabbits in Groups One and Two. There were 62 gas emboli.

discrete microbubbles were observed. Regardless of infusate volume, no gas entered the pial vessels of inverted rabbits.

More than 80 per cent of the gas emboli that entered the pial arterioles distributed without interruption to the veins, and gas was often seen in the large veins (>200 µm diameter). In upright rabbits, gas escaped from the jugular vein cannula within 30 seconds of gas introduction into the femoral artery. The gas was collected in the air traps as a blood foam. Gas accumulated in these air traps even when the jugular vein was ligated distal to the trap, a technique that prevented the retrograde passage of air emboli. Some gas emboli became trapped in the pial arterioles either temporarily, or permanently to cause local circulatory arrest (see Figure 2 on page 116).

The progress of a gas embolus through the pial circulation was related to the volume of the embolus (Figure 3). The larger the embolus the more likely it would lodge in a pial arteriole to block blood flow (Table 1) (Pearson ratio = 6.68; likelihood ratio — 6.73: $p < 0.01$). Emboli became trapped in arterioles of 50 to 200 µm diameter, most frequently in those vessels with external diameters of less than 100 µm (Figure 4). If an embolus entered an arteriole of this size such that the length of the embolus exceeded 5000 µm, then local circulatory arrest was inevitable. Conversely, if the length of the embolus was less than 5000 µm, it progressed to the venous circulation without interruption. Emboli of intermediate length (500-5000 µm) often became trapped, but this was usually temporary, redistributing to the veins within 3 minutes. Those intermediate length emboli that did not redistribute spontaneously were almost always trapped in arterioles with external diameters of less than 75 µm.

The spontaneous redistribution of emboli only occurred during the period of hypertension that followed gas embolism. If the embolus remained trapped in the arteriole after the blood pressure resumed normal or below normal values, then subsequent spontaneous redistribution did not occur. Redistribution always occurred, if within 10 minutes of embolus arrest, a forceful infusion of saline into the femoral artery was used to create a step increase in arterial pressure of greater than 150 mmHg. Conversely, redistribution never occurred, regardless of the increase in arterial pressure if the forceful fluid infusion occurred more than 15 minutes after embolus arrest. This inability to redistribute gas emboli from the pial arterioles was a consequence of arteriole wall collapse, and occlusion of the arteriole lumen.

Spontaneous redistribution was followed in 10 of the 27 rabbits by spontaneous re-embolism. In these rabbits, the additional gas emboli entered the pial arterioles within 5 minutes of the original emboli having redistributed. This

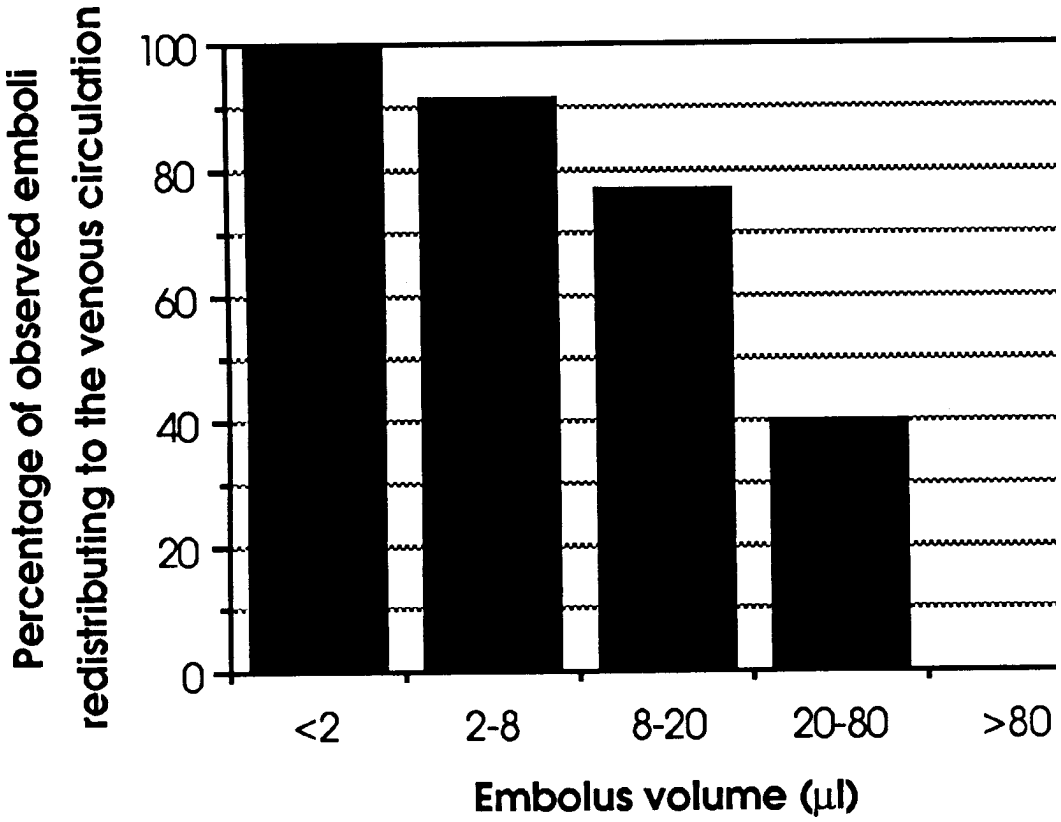

Figure 3. The relationship between the volume of gas emboli in the pial arterial circulation and the degree of redistribution of these emboli to the venous circulation in Groups One and Two (10 rabbits with 62 gas emboli).

occurred without any further gas infusion into the femoral artery, and without any other manipulation of the rabbit.

Respiration

Pial gas embolism was often accompanied by brief (< 30 seconds) periods of apnoea. If sufficient gas entered the arterioles to cause pial circulatory arrest there was invariably associated respiratory arrest. Spontaneous respiration resumed in those rabbits who became normotensive after embolism. No effect on respiration was observed if the infusion of gas into the femoral artery did not cause pial gas embolism. Regardless of infusate volume, no respiratory abnormalities were observed in inverted rabbits.

Circulation

Pial gas embolism was associated with cardiac bradyarrhythmias, and occasionally with cardiac arrest. Cardiac arrest always occurred within several minutes of respiratory arrest. A typical bradyarrhythmia is displayed in Figure 5.

If an embolis lodged in a pial arteriole, blocked blood flow, and did not redistribute to the venous circulation, then the consistent finding for all rabbits was progressive systemic arterial hypotension, and eventual death. The mean survival time for rabbits with such pial circulatory arrest was only 26 minutes ± 6.9 (SD).

TABLE 1

	EMBOLUS VOLUME µl	NUMBER OF EMBOLI	NUMBER OF EMBOLI PASSING TO VEINS	PERCENTAGE OF EMBOLI PASSING TO VEINS
	< 8	36	33	91.7
	> 8	26	17	65.4
TOTALS		62	50	80.6

The relationship between the volume of gas emboli in the pial arterial circulation and the redistribution of these emboli to the venous circulation in Groups One and Two (10 rabbits and 62 emboli).

Pearson ratio = 6.68; likelihood ratio = 6.73; $p < 0.01$; (p type 1 error = 0.009)

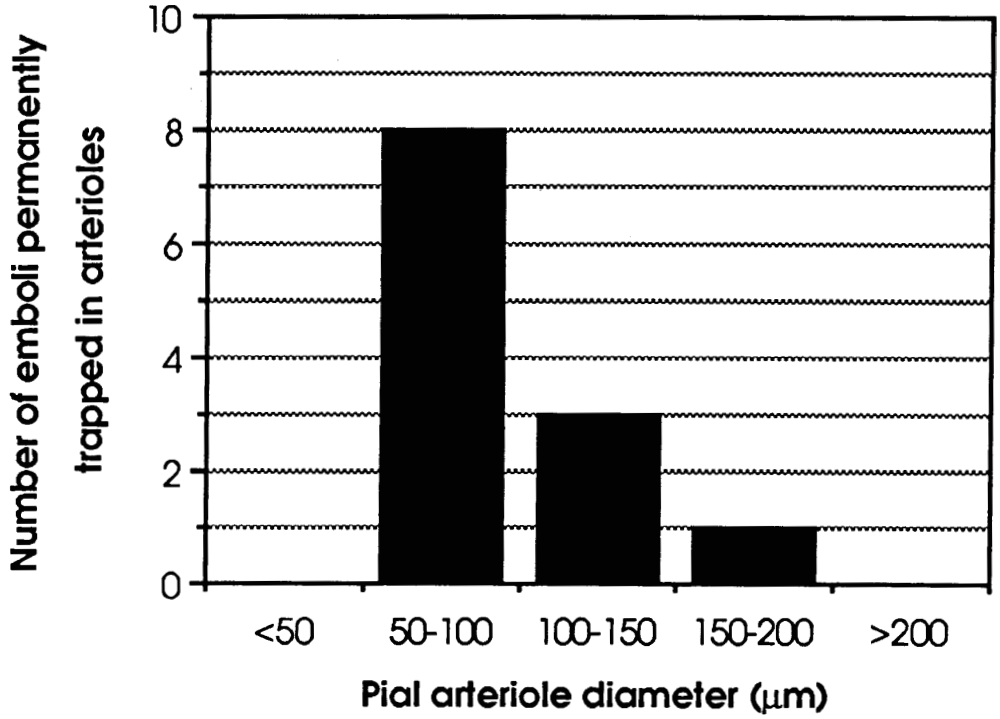

Figure 4. The relationship between the size (diameter) of pial arterioles and the number of emboli lodging in them to block blood flow in Groups One and Two (10 rabbits). n_1 = 62 gas emboli; n_2 = 12 gas emboli lodging in arterioles to block blood flow; n_1/n_2 = 0.194.

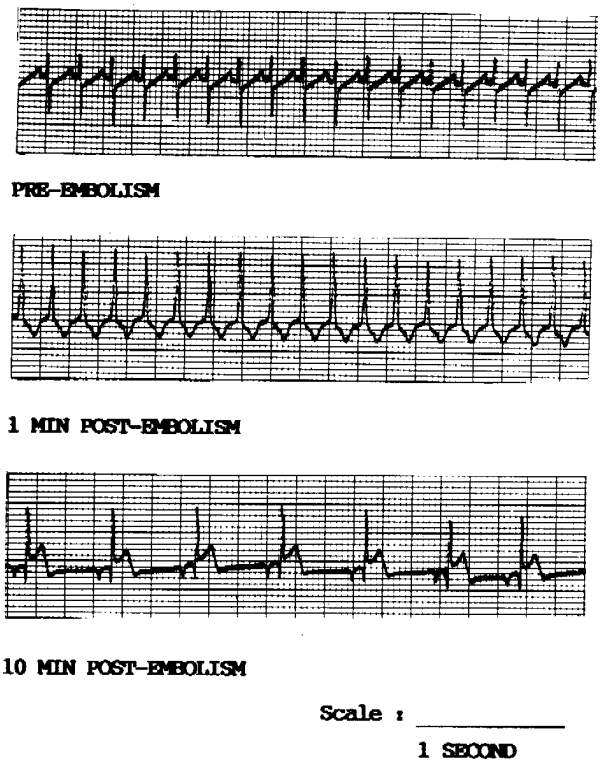

Figure 5. Cardiac bradyarrhythmia associated with pial arterial gas embolism. Scales: Vertical; 0.5 mv/major division. Horizontal; 5 major divisions/second.

The infusion of air microbubbles into an upright rabbit's femoral artery always caused a significant increase in MABP ($t_9 = 8.15, p < 0.001$). However, the accompanying increase in pulse pressure was not significant ($t_9 = 1.07$). A typical blood pressure recording with infusion of gas into a femoral artery of a rabbit is shown in Figure 6. Similar, prolonged changes in blood pressure were never demonstrated in those rabbits where saline was infused into the femoral artery using an infusion technique identical to that used for gas. A typical blood pressure recording following such a saline infusion is also shown in Figure 6.

The increase in MABP did not differ significantly between those rabbits breathing air, where air microbubbles were infused into their femoral arteries, and those rabbits breathing oxygen, where oxygen microbubbles were infused into their femoral arteries ($t_{13} = 0.6$) (Table 2). The MABP returned to pre-infusion levels within 6 minutes of embolism in those rabbits breathing air who were embolised with air microbubbles (mean: 3.15 mins+ 2.2 (SD)). The MABP returned to pre-infusion levels within 2.5 minutes of embolism in those rabbits breathing oxygen who were embolised with oxygen microbubbles (mean: 1.85 mins+ 0.46 (SD)). The difference between the recovery times in these 2 groups was not significant. ($t_{13} = 1.28$).

Gas microbubbles infusion into the femoral artery of inverted rabbits also caused a significant increase in MABP ($t_4 = 7.95, p < 0.01$). However, this increase in MABP was significantly less than that seen in upright rabbits ($t_{13} = 3.68$, $p < 0.01$; One way ANOVA $F = 8.37$, $p < 0.005$). Also, in 3 of the 5 inverted rabbits the blood pressure did

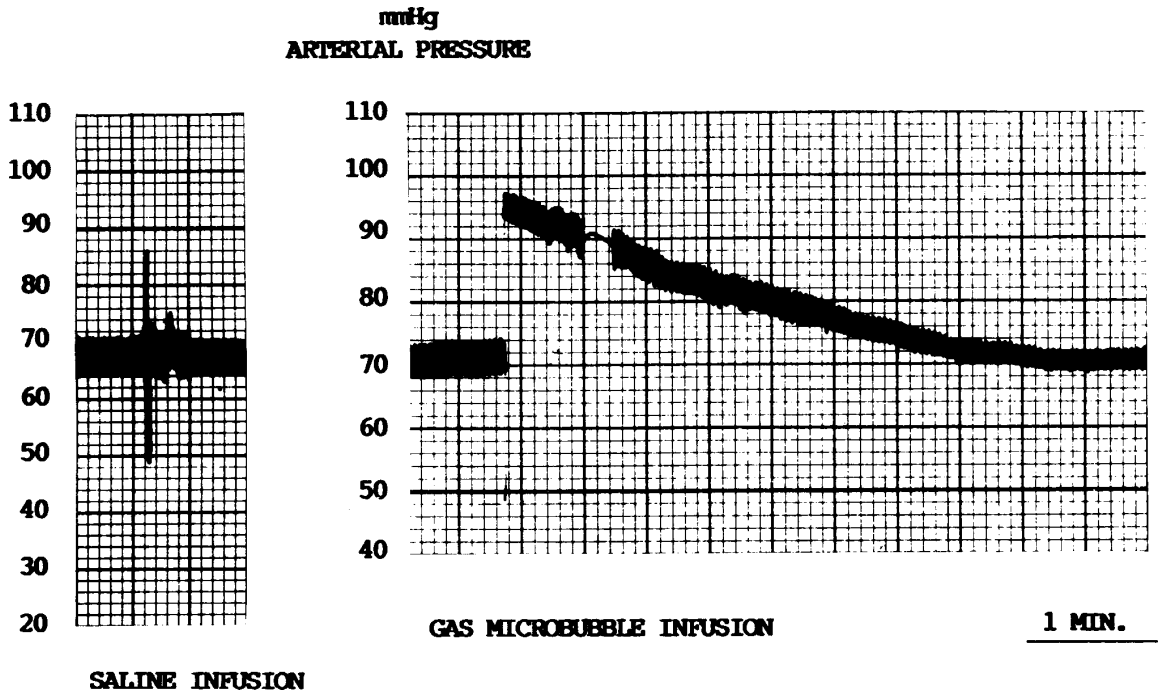

Figure 6. The effect of femoral artery gas microbubble infusion, and saline infusion on the arterial blood pressure of an anaesthetised rabbit.

TABLE 2

Increase in Mean Arterial Blood Pressure (mmHg)

	Air Emboli Head-up N=10	Air Emboli Head-down N=5	Oxygen Emboli Head-up N=5
Mean ± Standard Deviation	41.1 ± 16.0	14.0 ± 4.0	45.2 ± 13.4
Range	20 - 66	12 - 21	22 - 57

Blood pressure increases with infusion of gas microbubbles into the femoral artery of rabbits.

not return to the pre-infusion level, remaining stable at the increased level (Table 2).

The arterial hypertension that accompanied AGE was often associated with pial arterial and venous haemorrhage.

Prior to embolism, the resting MABP in upright rabbits breathing air (mean: 69.8 mmHg ± 9.1 (SD)), was the same as that recorded in upright rabbits breathing oxygen (mean: 73.6 mmHg ± 6.62 (SD)) ($t_{13} = 0.82$). However, prior to embolism, the resting MABP in inverted rabbits breathing air (mean: 84.2 mmHg ± 14.2 (SD)), was significantly greater than in upright rabbits also breathing air ($t_{13} = 2.41$, $p < 0.05$).

Pial Vessel Responses

Pial gas embolism caused an increase in pial arteriole diameter in all rabbits (mean increase: 42 % ± 28.13 (SD)). When the embolus progressed without interruption through to the venous circulation, the vessel immediately returned to its original size.

Observations on 16 of the 27 rabbits, demonstrated that the pial vasodilation that followed the infusion of gas into the femoral artery could occur without pial gas embolism (mean increase: 17 % ± 7.75 (SD)). The arteriole diameter increased concurrently with the increase in blood pressure in these 16 rabbits. Similarly, if pial gas embolism did not subsequently occur, the arteriole returned to its pre-infusion size with the fall in blood pressure.

This increase in arteriole diameter seen prior to embolism did not occur to the same extent in all the pial vessels of a given rabbit. Occasionally the arteriole wall was observed to pulse, with pulse cycle periods in a vessel segment of about 20 seconds.

Vasodilation was preceded by a transient, but measurable reduction in arteriole diameter in 7 of the 27 rabbits (mean decrease: 18.3 % + 14.5 (SD)). The reduction in arteriole diameter occurred prior to (3 animals), or without (4

animals) pial gas embolism, and was seen immediately after the infusion of gas into the femoral artery in these rabbits.

Groups One, Three, Four and Five

The gas infusion volumes necessary to cause pial circulatory arrest in rabbits in Groups One, Three, Four and Five are displayed in Table 3. A one way ANOVA ($F = 17.46$, $p < 0.005$) demonstrated that the volumes differed significantly between the groups. Further analyses with unpaired t-tests demonstrated that the gas volume necessary to cause pial circulatory arrest in Group Four (Oxygen ventilation/ Oxygen microbubbles) rabbits was significantly greater than in Group Three (Oxygen ventilation/Air microbubbles) rabbits ($t_4 = 3.96$, $p < 0.05$). In addition, the gas volume necessary to cause pial circulatory arrest in Group Three rabbits was significantly greater than in either Group One (Air ventilation/Air microbubbles) or Five (Oxygen ventilation/Air foam microbubbles) rabbits ($t_4 = 3.29$, $p < 0.05$; $t_4 = 2.88$, $p < 0.05$).

The mean volume of gas collected in the left jugular vein air-traps in upright rabbits (Group One and Two) was 24.7% $\pm$ 2.5 (SD) of the volume infused into the right femoral artery.

Group Two

Regardless of infusate volume, the infusion of air microbubbles into the femoral artery of inverted rabbits never caused pial gas embolism. Similarly, under these conditions gas was never collected in the left jugular vein air-traps. This was not due to mechanical problems with their infusion lines, because both pial gas emboli, and jugular venous gas emboli were produced in these rabbits when they were subsequently changed to an upright posture.

Discussion

Although the results of the experiments reported here deal with pial gas embolism in rabbits, the conclusions drawn can be extrapolated more generally to CAGE for two reasons. Firstly, the responses of rabbit pial arterioles to changes in blood pressure and PCO_2 are not affected by being exposed (54), nor are they abolished by a constant level of halothane anaesthesia at the MAC for this species (55,56). Secondly, in this species, pial vessel responses parallel those of intro-parenchymal vessels of similar size (54).

Respiratory and Circulatory Effects of AGE

The infusion of air, of air foamed in either detergent or a lung extract, or of microbubbles of oxygen into the femoral artery of rabbits caused respiratory depression. This varied from a brief apnoea to lethal respiratory arrest. Respiratory depression was never produced in inverted rabbits, supporting the association of respiratory depression with CAGE reported elsewhere.[3,12-14,18,31,43]

TABLE 3

	NUMBER OF RABBITS	EXPERIMENTAL CONDITION	MEAN VOLUME OF GAS TO EFFECT ARREST (ml)
GROUP ONE	5	Air/Air microbubbles	10.0 + 1.6
GROUP THREE	5	O_2/Air microbubbles	30.0 + 12.2
GROUP FOUR	5	O_2/O_2 microbubbles	60.0 + 19.0
GROUP FIVE	7	O_2/Air^2 microbubbles	9.0 + 1.6

Mean (+ Standard Deviation) volumes of gas infused into the femoral artery of rabbits to effect pial circulatory arrest under varied experimental conditions ($F = 17.46$, $p < 0.005$).

NOTE: Air foamed in 3% teepol or lung preparation.

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Group Two

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Gas microbubble infusion into the femoral artery was also associated with cardiac bradyarrhythmias and occasionally with cardiac arrest, but only if pial gas emboli were produced. The role of coronary artery embolism was not measured, but it is accepted by other authors that gas emboli can effect heart function by entering the brain stem circulation, the coronary circulation, the heart chambers, or can affect cardiac function indirectly by enhancing the release of catecholamines into the systemic circulation.3[,9,18,31,35,36,37-39,40,42]

The infusion of oxygen or air microbubbles into the femoral artery of upright rabbits caused the MABP to increase. This increase was significantly different from the increases seen either after the infusion of saline into upright rabbits, or the infusion of gas microbubbles into the femoral artery of inverted rabbits. Similar increases in MABP have been demonstrated by other researchers, with infusion of gas into either the carotid or vertebral arteries, or into the aorta or pulmonary veins.3,9,12-14,18,31,37-40 Embolism of the brain stem circulation appears necessary for the typical hypertensive response to gas emboli.3,12-14,18,24,58-62 This conclusion is based on the observation that this characteristic increase in MABP can be prevented by bypassing the brain stem by either

using a slow infusion carotid artery, or by isolation of the vertebrobasilar system. The transient nature of the hypertensive response to gas emboli demonstrated in this study has also been seen elsewhere.3,12-14,18,38,39,43

In inverted rabbits, embolism of the brain stem circulation can not explain the small, but often prolonged increase in MABP that followed gas microbubble infusion into the femoral artery. This increase in MABP was identical to that reported by another researcher who introduced gas into a pulmonary vein of inverted dogs.[3] It was also consistent with other studies where gas emboli were injected into peripheral arteries.[63,64] These increases in blood pressure may be a consequence of emboli becoming trapped and so physically causing an increase in peripheral vascular resistance.[3]

Pial gas embolism was often associated with pial arterial and venous haemorrhage. Haemorrhage concurrent with CAGE has already been reported.

The increase in MABP recorded after the infusion of oxygen microbubbles into the femoral artery of rabbits being ventilated with oxygen, was not significantly different from the increase seen after the infusion of air microbubbles into rabbits ventilated with air. It follows that blood pressure changes could not explain the greater volume of oxygen microbubbles needed to cause pial circulatory arrest.

The diameter of the affected arterioles increased after pial gas embolism. Similar diameter increases have been reported previously, and attributed to either local acidosis, or to endothelial damage causing local vasoparalysis. In this study, if the emboli did not become trapped, the vessel immediately returned to its pre-infusion size. This rapid reversal is not consistent with the time course of a vasoparalytic state resulting from either the local hydrogen ion concentration, or endothelial damage. More significantly, arteriole dilation was recorded after gas microbubble infusion when pial gas embolism was not observed, demonstrating that the blood vessel dilation was not entirely due to local, gas-induced, phenomena.

The concurrence of arteriole dilation and an increase in MABP after gas infusion demonstrated that AGE in this model was associated with a loss of cerebrovascular autoregulation. This was confirmed by the observation that if gas embolism did not occur, the arterioles returned to their pre-infusion size as the MABP returned to normal levels.

One significant observation in this study was that AGE, and not the nature of the anaesthesia or the surgical preparation of these rabbits, was responsible for the demonstrated loss of cerebrovascular autoregulation. Seven rabbits exhibited a reduction in arteriole diameter with the initial increase in blood pressure. That is, they demonstrated normal cerebrovascular autoregulation. Further increases in the blood pressure of these 7 rabbits caused the vessels to dilate, which suggests that the loss of autoregulation was a consequence of the blood pressure exceeding the upper limit at which this regulation of CBF can exist.[52,53]

Other researchers have shown that rabbit pial vessels exhibit cerebrovascular autoregulation, and that creation of an open-brain preparation does not inhibit these responses. For example, both normal cerebrovascular autoregulation to reductions in blood pressure, and cerebral vasoreactivity to changes in PCO_2, have been demonstrated in rabbit pial arterioles despite an open-brain preparation.[54] Normal cerebrovascular autoregulation to increases in blood pressure has also been demonstrated with open brain preparations; a 5 to 6% decrease in diameter of rabbit and cat pial arteries of about 120 μm diameter has been reported in response to blood pressure increases within the physiological range.[46] Importantly, in the NZ White Rabbit, it can be assumed that the behaviour of pial arterioles is similar to that of intra-parenchymal vessels of similar size.[54]

Research with other species also demonstrates that exposed pial vessels remain reactive to changes in carbon dioxide, vasoactive metabolite, and to cation and anion concentration.[66-68] Pial arterioles of 50 to 300 μm diameter contribute to overall cerebrovascular resistance, with 15 to 21% of the drop of total CPP occurring across these vessels.[69] Furthermore, cerebrovascular autoregulation is largely independent of changes in ICP, CSF pressure, and cerebral venous pressure.[47,70,71]

Although halothane has been shown to inhibit cerebrovascular autoregulation in some species,[72-74] it clearly did not in this study as the rabbits demonstrated a normal response to the initial increase in blood pressure. Other researchers have also shown that cerebral vessel reactivity, and its modulation by changes in systemic blood pressure, persists in NZ White Rabbits despite halothane anaesthesia.[55,56] As in this study, these researchers maintained anaesthesia with a constant halothane vapour pressure, equivalent to the MAC for this species.[56]

A review of the available literature also supports the argument that AGE itself can disrupt cerebrovascular autoregulation. The most important finding is that cerebrovascular autoregulation is lost in animal models of AGE not involving either halothane anaesthesia or a craniotomy, where CBF rather than vessel diameter has been measured

The mechanics of normal cerebrovascular autoregulation remains controversial.[51-53] The mechanism by which AGE disrupts autoregulation was not addressed by this study. However, the simplistic explanation that the disruption was the consequence of exceeding the upper limit of blood pressure at which autoregulation can exist, has attraction.[52,53] Because the loss of autoregulation often occurred in the absence of pial gas emboli, it can be concluded that it was not entirely due to gas-induced endothelial damage, or to local acidosis as a consequence of circulatory arrest. The disruption of the normal autoregulation of CBF will influence the outcome of patients with CAGE because both CPP and CBF will passively follow MABP, such that rises and fails in MABP will respectively promote and

inhibit embolus passage to the venous circulation.

Arterial Gas Embolus Distribution

Gas microbubbles infused into the femoral artery of upright rabbits distributed against aortic flow to embolise the pial circulation. Conversely, pial gas emboli were never observed in inverted rabbits. These observations are consistent with other reports on the relationship of posture to the distribution of arterial gas emboli,[1-3] and with the frequent neurological involvement in divers and submariners with AGE.[4-8,32,40] This study also supports the use of an inverted posture in the treatment of AGE, to restrict embolism of the cerebral circulations.[65,75,76] It is even possible that posture may influence the redistribution of gas emboli that have already entered the brain vessels.[65,77,78]

Although the gas was infused into the femoral artery as microbubbles, only coalesced columns of gas were observed in the pial vessels. Such coalescence is predictable gas behaviour,[14,36,76] and was not prevented by foaming the gas in either a detergent or a homogenised lung preparation. Only cylindrical gas emboli became trapped in the pial vessels. This is consistent with previous reports, and supports the argument that the cylinder, and not the sphere, is the appropriate gas model for CAGE

The passage of a gas embolus through the pial circulation of this animal model was determined by the MABP, the volume, and the length of the embolus, and the diameter of the vessels. The redistribution of emboli from pial arterioles to the venous circulation only occurred while the MABP remained elevated above pre-infusion levels. Movement of emboli after this time only occurred if a step increase in arterial pressure was created by forceful infusion of saline into the femoral artery. On this evidence induced hypertension could become an important component of the treatment of patients with CAGE.[80] However, any therapeutically induced hypertension needs to be controlled to avoid either increasing the permeability of the BBB, or further disturbing cerebrovascular autoregulation.[52,81,82]

While an increase in arterial pressure induced shortly after the pial circulation was arrested by gas emboli was highly effective, the universal failure of similar increases induced more than 15 minutes after embolus arrest suggests that therapeutic gas embolus redistribution is only possible for a short time after embolism; before arteriolar collapse. The time scale of arteriolar collapse found in this animal model may not be the same in humans. Nevertheless, this phenomenon may explain the difference in morbidity with CAGE between those patients compressed in a RCC immediately after the onset of symptoms and signs[4,6-8,40,83] and those with a delay prior to compression[4,6-8,32,34,40,83,84]

Large emboli (> 5000 μm length; > 20 μm volume) usually became trapped in a pial arteriole, and caused local circulatory arrest. Conversely, small emboli (< 500 μm length; < 8 μm volume) usually did not become trapped; emboli passed through without interruption, to the pial veins. Many intermediate- sized emboli (500-5000 μm length; 8-20 μm volume) were temporarily trapped. Most of these emboli (> 75%) eventually passed through to the venous circulation during the period of hypertension that followed embolism.

Less than 20 % of all the observed emboli lodged permanently in pial arterioles and blocked blood flow. This usually occurred in vessels less than 100 μm in diameter. The measured mean survival time of rabbits in whom emboli were large enough to cause pial circulatory arrest was only 26 minutes. This mortality contrasts with the observation that most humans with CAGE do not die, but rather experience some degree of improvement prior to any treatment.[32,33] When coupled with the finding that more than 80% of the emboli that were seen to enter the pial arteries in our study eventually redistributed to the venous circulation, it is clear that the conventional pathophysiological model of AGE, one depending on the blockage of arterioles by gas, is not supported by these data.

The frequent redistribution of gas emboli from pial arterioles to the venous circulation without any therapeutic manoeuvre can explain why most human patients with CAGE experience some degree of spontaneous recovery[32,33] However, even in those with complete recovery, many will subsequently relapse.[32] Our results suggest one mechanism to account for some of these relapses.

The patient's initial improvement is probably due to spontaneous redistribution of gas emboli from cerebral arteries to the venous circulation. However, in some patients, intermediate-sized emboli will remain in vessels less than 75 μm in diameter. Recovery of brain function will still be possible, because of the collateral pathways that exist at this level of the brain circulation.[85] The passage of gas emboli through the larger vessels will have disrupted the BBB[15,20,26-30,32,83] and stimulated the local accumulation of platelets.[24] As platelet thrombi form there will be a progressive fall in CBF.[21-25] The resulting reduction in flow through the collateral pathways will eventually cause a loss of function in the areas of brain tissue supplied by the embolised vessels, and the patient will relapse with similar symptoms to their original presentation. The small number of patients with CAGE that have fulminant, and occasionally lethal disease,[4,7,40,83] are well explained by the observed behaviour of large emboli.

It has already been argued[19] that the course of one group of patients with CAGE can only be adequately explained by re-embolism. In contrast, the mechanism of relapse detailed above is proposed only for those patients that relapse with similar symptoms and signs to their initial presentation .[8,86]

There is a simple explanation for the relationship between the size (length in a given vessel) of an embolus and its eventual distribution. This can be derived from the La Place Equation, and is based on the difference in arteriole diameter, and hence surface tension pressure, at the extremes of the embolus. As the embolus length increases, the difference in diameter, and hence the nett surface tension

pressure opposing embolus movement also increases.

A simplified schematic is presented in Figure 7. In this schematic, Øp and Ød are measured contact angles, and rp and rd are the proximal and distal embolus radii respectively. The driving force will be the nett CPP (Δ CPP), and will be opposed by the nett surface tension pressure. The La Place equation, Equation 1, defines the condition for movement of an embolus:

$$\Delta CPP > \frac{2\gamma \cos Ød}{rd} - \frac{2\gamma \cos Øp}{rp} \quad \text{(Equation 1)}$$

where: y is the plasma surface tension.

Figure 7 is a simplified schematic, and Equation 1 does not account for the irregular arteriole dilation associated with gas embolism. Similarly, it ignores the pulsatile nature of the proximal interface of the gas embolus. Nevertheless, the principle established above remains valid.

did not allow for spontaneous embolus redistribution, and so have probably overestimated the success of the regimens tested.

Some rabbits not only demonstrated spontaneous redistribution of gas emboli from the pial arterioles, but also spontaneous re-embolism. The subsequent emboli entered the pial circulation without further infusion of gas, and without manipulation of the rabbit. It follows that re-embolism of the brain circulation can occur whenever gas emboli exist in the arterial circulation, and does not require continued embolus production. This phenomenon can explain the progress of those patients with CAGE who, after an initial improvement, relapse with a different set of focal symptoms and signs.[4,86]

More oxygen microbubbles had to be infused into rabbits ventilated with oxygen to cause pial circulatory arrest, than either the volume of oxygen microbubbles used in rabbits ventilated with air, or the volume of air microbubbles

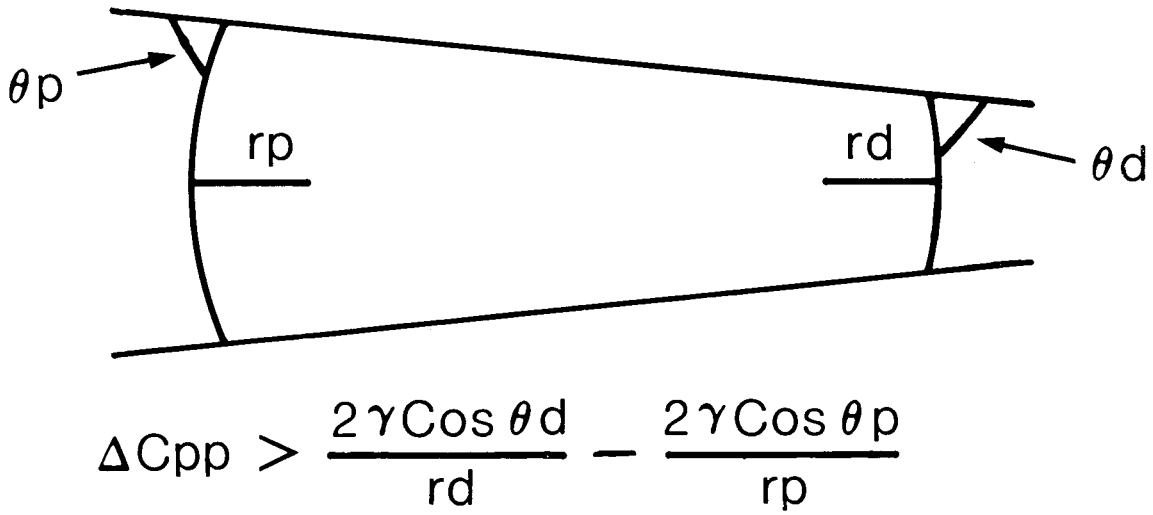

Figure 7. Simplified schematic of pial arterial gas embolism demonstrating a state of embolus progression.

The large volumes of gas collected in the jugular vein air-traps in this study, were similar to those reported by another group of researchers who also used jugular vein air traps.[3] Clearly, redistribution of gas emboli from pial arteries, and presumably other cerebral arteries, to the venous circulation can occur without compression of subjects in a RCC. Other authors have also reported the spontaneous redistribution of gas emboli from cerebral arteries to veins and to the right ventricle and pulmonary arteries.[12,31] Significantly, the earlier this redistribution occurs, the quicker animals with experimental CAGE improve.

The spontaneous redistribution of gas emboli seen in this study, and the similar observations in other animal models of CAGE, not only casts doubt on the conventional model of CAGE, but also on the validity of findings in several animal model studies of CAGE treatment.[13,14,16] These studies demonstrated clearance of gas emboli from pial arterioles during compression to 6 Bars. However, they

used in rabbits ventilated with air to achieve the same result. This difference can not be explained on the basis of desaturation, but does support previous studies of oxygen gas embolism.[12,18] It also supports the suggestion that oxygen be breathed by submariners during an escape from a stricken submarine, where the pressure exposure would be brief, and overt central nervous system oxygen toxicity unlikely.[87,88]

Multiple infusions of gas into the femoral artery, and large volumes of gas in comparison to those used in previous studies,[13,14,16,18,21-29,31,33,37-39, 58,60-62] were necessary to cause circulatory arrest in a pial arteriole in this study. The reasons for the relatively large gas volumes include how the end-point of circulatory arrest was chosen, and the site of gas infusion.

Since previous animal model studies of CAGE in which the pial circulation was observed have not allowed for any spontaneous redistribution of emboli,[13,14,16] a condition of

pial circulatory arrest may not have been created. It is also possible that the subsequent embolus redistribution seen by these researchers may have occurred regardless of the treatment they administered to their animals.

Most animal model studies of CAGE have used carotid artery gas infusions.[13,14,16,18,21-29,31,33,58,60-62] Emboli from such infusions can bypass the brain stem circulation, and so avoid triggering an increase in blood pressure.3,12-14,18,24,58-62 It is clear from this study, that the transient, but significant increases in blood pressure that accompany AGE are critical determinants of the passage of an embolus through the cerebral vessels. Carotid artery cannulation itself may alter cerebral perfusion, and so distort the balance of forces that determine embolus distribution.

Other approaches to the study of CAGE have involved either the measurement of neurophysiological parameters, or studies of subsequent cerebral histopathology.[18,58,60-62,89] Despite using gas volumes which were much smaller than those necessary to cause pial circulatory arrest in this study, these authors have reported significant brain pathology. It follows that such pathology can be associated with CAGE even though cerebral circulatory arrest, due to gas emboli, has not occurred. The likely explanation is the decrease in CBF that occurs after CAGE.[21-25] Once stimulated, the processes that account for this fall in CBF would not require the continued presence of a gas embolus, and would eventually cause the CBF to become inadequate for normal neuron function.[21-25]

Summary and Conclusions

It follows from these data and arguments that an alternative pathophysiological model of CAGE should be used. This model must be able to account for a small number of very large emboli becoming trapped in cerebral arterioles and blocking blood flow. This is the rare situation of fulminant, and sometimes lethal CAGE. The model must also attribute much of the brain pathology that results from CAGE to the effects of a transient gas-endothelial interaction (accumulation of platelets, formation of platelet thrombi, disruption of the BBB) rather than to the direct mechanical effects of gas emboli themselves. Finally, the alternative pathophysiological model must be able to explain both the improvement seen in most patients prior to any treatment, and any subsequent relapse.

The aims of treating patients with CAGE must remain the removal of gas emboli from cerebral arterioles, and the restoration of CBF. Although a study of the former is only applicable to the small number of emboli large enough to become entrapped, such emboli probably underlie fulminant disease, and contribute significantly to subsequent relapses in brain function in those patients that survive the initial episode. Because available data on this subject are very limited, and will have overestimated treatment efficacy, it is clear that such a study is needed.

The data presented here also demonstrate that any study of therapeutic embolus redistribution must be able to measure spontaneous embolus passage to the venous circulation. Furthermore, it is essential that all treatment trials begin from a point of established cerebral circulatory arrest.

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KEY WORDS

Arterial Gas Embolism, Cerebral Arterial Gas Embolism, Gas Embolism

Figure 2 appears, in colour, on page 116.

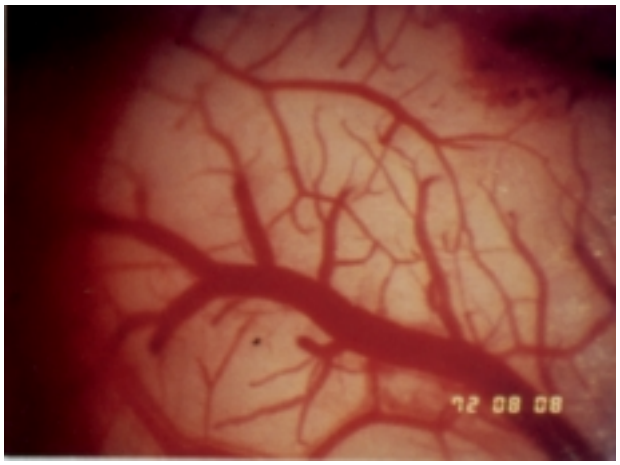

Figure 2A Photomicrograph (x500) of rabbit brain surface before arterial gas embolism.

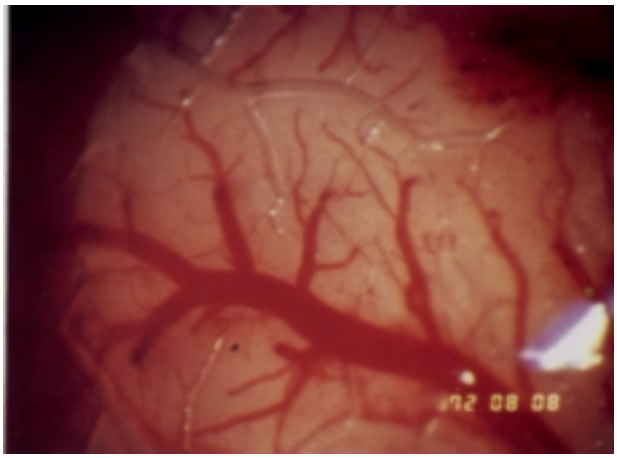

Figure 2B Photomicrograph (x500) of rabbit brain surface after arterial gas embolism.

SPUMS ANNUAL SCIENTIFIC MEETING 1987

SCOMBROID POISONING

John Knight

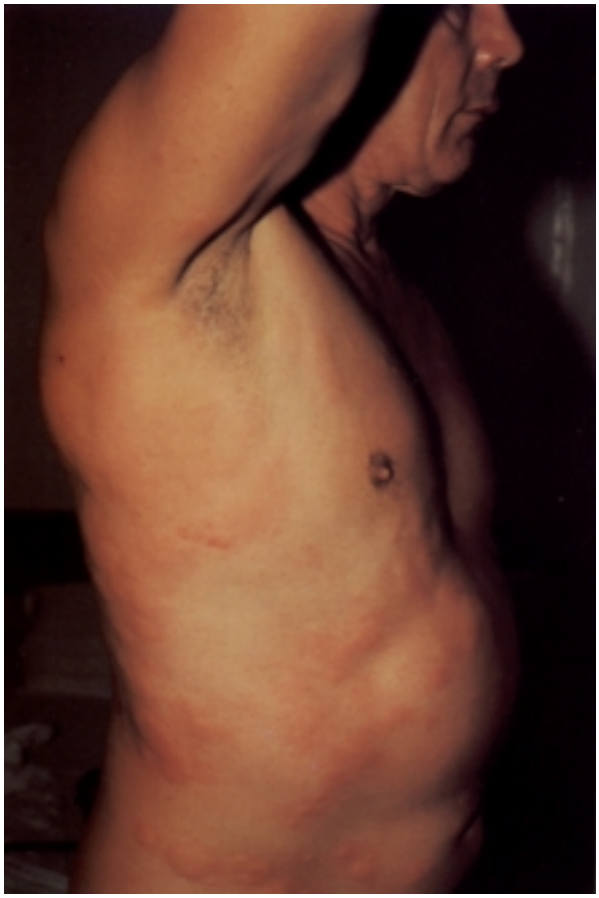

Figure 1. Side view of the author at 0100 on 25 April 1985.

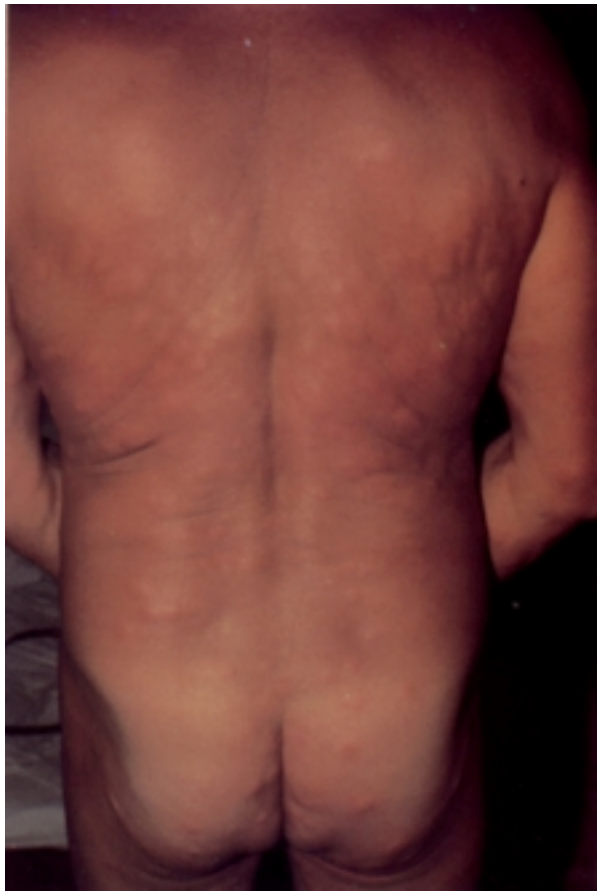

Figure 2. Back view of the author at 0100 on 27 April 1985.

The Scombroid fishes, which include mackerel and tuna, can cause a particularly dramatic and unpleasant form of poisoning. It is a series of histamine reactions. This paper is a personal recollection of what it does to the patient and the effects of treatment combined with a short review of what is known about it.

Case History

During the 1985 SPUMS meeting in the Maldives most members had their fill of the local fish and by the final night refused the barbecued fish. I had some about 2000 and was given a larger than usual helping. The fish tasted excellent. The rest of the evening was spent at the bar, leaving for my room about 2330. On the way I started to itch on my arms and body and thought that I had been bitten by mosquitoes. When I undressed I had a number of blotchy spots on my skin which itched. However it was not too difficult to get off to sleep.

About 0100 I woke itching and scratching. My face felt swollen and my mouth felt rather like the after effects of dental analgesia. However I could still smile but in repose I looked as if the end of the world was nigh. The rash had spread widely and obviously was not mosquito bites (Figures 1 and 2). It was then that the penny dropped. I had eaten some tuna and now had an obvious histamine rash. I had scombroid poisoning. I took a promethazine (Phenergan) tablet with two betamethasone (Celestone) tablets and managed to get back to sleep.

At 0500 when we had to get up I was covered in blotches. My face and body were both involved and I was itching madly. Carl Edmonds took some photographs of me at this stage. More promethazine and betamethasone made me feel a bit better but by the time we were waiting at the airport I was having difficulty standing. By the time we got to Singapore all I wanted to do was collapse into bed.

Next morning the "maps" were even more pronounced, promethazine was not controlling the itching and there were no more betamethasone tablets. At 1300 I still had a swollen face and many blotchy areas (Figure 3) so I sought local medical aid and was given more betamethasone tablets. I needed two every six hours to control the itching and stop the appearance of new blotches. After 24 hours on steroids my face was nearly normal and the blotches were beginning to disappear although there were plenty still visible (Figure 4). Over the next three days the dose was reduced to one betamethasone tablet every six hours and I had to stay on this dose for five days as symptoms recurred every time I reduced the dose by a tablet. Then I was able to reduce the dose by a tablet a day. When I had taken the last tablet, even though I had been on steroids for a fortnight, I still had some blotches.

Compared with ciguatera the course of the disease was short and, once adequate steroid treatment was started, painfree. The reversal of temperature sensation with ciguatera lasts for months and drinking alcohol can restart the symptoms. Mercifully neither occur with scombroid poisoning.

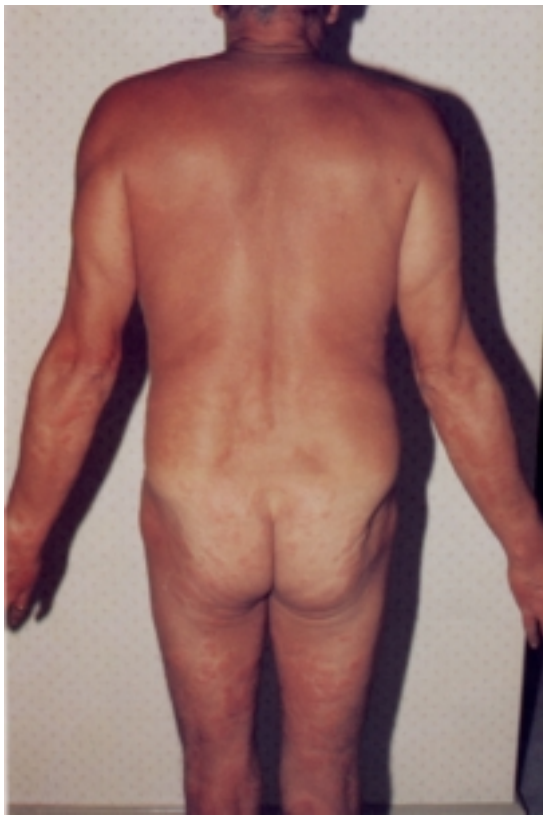

Figure 3. Back view of the author at 1300 on 26 April 1985.

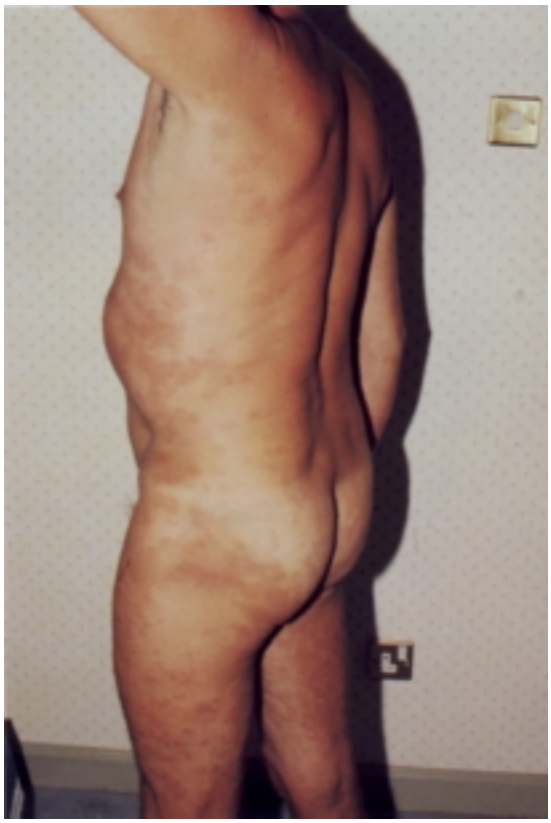

Figure 4. Side view of the author at 1400 on 27 April 1985.

Causation

Not much has been written recently about scombroid poisoning. I have been able to trace six papers or letters to the editor in journals since 1975[1-6] and there are a few words about it in "Australian Animal Toxins"[7] and "Diving and Subaquatic Medicine".[8] The most information I came across was published in 1961.[9] The relevant chapter was written by Kimata and dealt mostly with work done in Japan.

50 years ago Igarashi found large amounts of histamine in the muscle of the chub mackerel inspite of treatment with antibacterial solutions. He thought that the histamine was due to autolysis. Kimata was unable to reproduce these results. He considered that it was bacterial action which caused the histamine build up. The maximum histamine production, both in rate and amount occurred at 20°C in dark meat fishes, such as mackerel, horse mackerel, bonito and tuna, which were the only ones to produce histamine with bacterial spoilage. Oddly enough little histamine was produced at 37°C and less surprisingly none was produced when the fish were kept at 0°C. There was no evidence of a link between the freshness of the fish and histamine production.

The amino acid histidine appears to be necessary for the formation of histamine which is not only found in the muscle of fish. Kimata stated that histidine production was least around 17°C. It was three times as much at both 6°C and at 35°C.

It appears that the fish become poisonous when they are left at room temperature or exposed to the sun, for several hours. Kimata's work suggests that bacterial action turns the histidine in their muscles into histamine.

However it seems to me that other amines must also be formed as the effects of histamine are short lived. Anyone who has been stung by stinging nettles knows that symptoms seldom last for more than an hour or two. So histamine is probably not the only poison produced in the fish. Kawabata gave the name saurine to substances having the same action as histamine which he though were present with the histamine. Besides saurine a scombrotoxin, which also has histamine like effects, has been postulated. From the fact that my symptoms lasted for many days, I am of the opinion that there was either a histamine provoking poison present in my body or a long lasting, slowly broken down, congener of histamine producing the same effects.

It is obvious that the site of the histidine degradation and the size of the dose of poison influences the onset of the symptoms as a number of people ate the fish that night but only one had symptoms.

Confirmation of the diagnosis

If any of the fish is left when the patient develops symptoms the diagnosis can be confirmed by laboratory testing showing a high histamine content in the muscle. The critical histamine level to produce symptoms is 100 mg/ 100 g of fish muscle.

Histamine producing bacteria

Proteus morganii has been shown to produce both histamine and saurine when innoculated into raw fresh tuna flesh. This organism is thought to be the main cause of scombroid poisoning. Other histamine producing bacteria include *Salmonellae, Shigella dysenteriae, Clostridum perfringens, Escherichia coli* and *Aerobacter aerogenes*. The identifications date from between 1910 and 1939 and come from Europe as well as Japan.

Histamine producing bacteria were found on the surface of all freshly caught fish in the 1950s in Japan. The bacteria were presumed to come from the fishing gear, nets and boxes. This was in the days before nylon nets became common.

Conclusions

Anyone who has eaten fish of the tuna and mackerel family and then develops symptoms of histamine release has probably developed scombroid poisoning. Treatment with antihistamines is helpful in reducing the itch but probably will not influence the progress of the illness very much. Steroids in large doses are needed to control the symptoms and the rash.

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DECOMPRESSION METERS PHILOSOPHICAL AND OTHER OBJECTIONS

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The use of decompression meters (DCMs) is not new, and has involved a wide range of apparatus, from mechanical to electronic, and both diver-worn and remote. The Canadian Defence and Civil Institute of Environmental Medicine surface-based decompression computer represents one extreme of this development and has proved useful. However, the active marketing of a new range (not "new-generation" as is claimed) of diver-worn DCMs requires that the case against such devices be stated again.

Multi-level Diving

A major advantage claimed for DCMs is that they account for the multi-level nature of most recreational diving. Consequently, a DCM will "permit" a longer exposure to pressure, for a given multi-level dive, than that allowed by the traditional use of the same decompression schedule (which assumes that the entire exposure was at the maximum depth).

The number of cases of Decompression Sickness (DCS) presenting for treatment in Australia and New Zealand has increased since 1980 and has shown an alarming predominance of nervous symptom involvement. These episodes of neurological DCS often arise after dives that either were conducted in accordance with conventional tables (with and without fudging), or were within no-decompression limits (despite being multi-level).

Based on current treatment rates it is anticipated that in 1987 between 300 and 400 divers will be treated for DCS in Australasia. While this does not establish that the disease rate (eg. DCS/1000 diving hours) has increased, it is clear that the diving practice of the recreational diving community needs to become more conservative. This recommendation for safer diving is not consistent with the increased exposure possible with DCM-controlled multi-level diving.

Measurement of Exposure

While the marketing information released with each new batch of DCMs declares the arrival of a "new generation" of devices, this is simply not true. All devices that have been sold, and are about to be sold, measure depth and time, and not tissue nitrogen tensions. What does change with each new model is how the information is manipulated and presented. The expected body-tissue nitrogen tensions are calculated from this input, using one or more mathematical models. In general, these models are perfusion-based and do not account for the diffusion limits of intracellular fluid. Whatever the basis of calculation, it is important to understand that the kinetics of inert gas uptake and elimination have not been accurately described. Not surprisingly then, the accuracy of calculated tissue nitrogen tensions using these available mathematical models of decompression is quite poor.

This intrinsic inaccuracy of decompression models, and hence of DCMs, will remain until a DCM can directly measure an individual's tissue nitrogen tension (eg. using transcutaneous or implanted electrodes). Such a DCM would only then be a "new generation" device.

Electronic Reliability

An absolutely reliable electronic instrument has not and never will be built. Trials with all available DCMs have shown a real, although often small, failure rate (including total display loss). Obviously electronic diver-worn DCMs can never be used in isolation. Divers using DCMs should always carry and use a hard copy of suitable decompression tables.

Summary

Although DCMs are simple to use and account for multi-level diving, it is not possible to support or advocate total reliance on them. They may have a useful role in diving, but only in conjunction with a careful dive plan and concurrent use of a hard copy of decompression tables.

ASSESSMENT OF THE ORCA EDGE DIVE COMPUTER

Carl Edmonds and Tim Anderson

INTRODUCTION

The Royal Australian Navy School of Underwater Medicine first became interested in decompression meters used by divers during 1972. Many patients sought treatment for decompression sickness, following the use of the SOS decompression meter. A study of this meter showed that it indicated shorter decompression times than required by the US Navy decompression tables when used for repetitive dives, and for dives in excess of 60ft.[1] The Farrallon Multi-Tissue Decomputer was also studied[2] but was unacceptable because of its unreliability. The DECO-BRAIN suffered a similar fate when tested, approximately two years ago.

The senior author was involved in the treatment of a diver in 1986 who used an Orca EDGE for two dives to 87ft, after which she developed decompression sickness. It appeared that the meter had allowed a dive combination that would not be permitted by the US Navy tables. There were several possible explanations of this decompression incident: a chance occurrence because of the fallibility of the decompression tables, a misreading of the meter, a fault within the meter itself, or the meter programme permitted unsafe diving profiles.

It was against this background that it was decided to test the EDGE decompression meter's no-decompression repetitive dives and compare these with the established decompression tables.

PROMOTIONAL MATERIAL

The literature which accompanies the Orca EDGE meter makes the following statements:

> The dive computer is a compact submersible computer which gives information needed to plan dives and avoid bends. It is also a precision depth gauge, dive timer and surface interval timer. It takes care of repetitive as well as single dives.
>
> The programme provides a safer dive than the US Navy tables, while providing more dive time for Multi-Level dives. It makes allowance for altitude exposure, and is functional over a wide range of water temperatures.
>
> The US Navy tables were designed for single depth dives. Divers are required to use the maximum depth of any dive profile to calculate the decompression, as if the entire dive had occurred at that maximum depth. Many divers feel that they are penalised and limited by this procedure since they do not spend all the time at the deepest depth.
>
> The EDGE accounts for the absorption of less nitrogen at shallower depths, and typically divers using the EDGE get double the time allowed by the US Navy tables.
>
> Divers have sought new procedures for interpreting the US Navy dive tables to allow longer bottom times. The interpretations that gained the most acceptance in the diving community are Multi-Level dive procedures.
>
> Some diving authorities indicate that these or similar procedures have been tested and used in commercial or oil field diving, although little published data is available. Many hyperbaric physicians feel that the concept is valid and safe, if specific precautions are followed.
>
> The decompression calculations used in the meter are based on Multi-Level diving techniques adapted from the US Navy no-decompression tables, and the shorter no-decompression limits developed by Dr Merrill Spencer.
>
> The dive computer calculates divers' tissue and decompression status of 12 different tissue groups ranging from half times of 5 minutes to 480 minutes. At the present time, the EDGE is the best solution to decompression problems, providing long bottom times along with excellent safety.

Other claims made on behalf of the meter are worth of note.

The manufacturers claimed to have carried out a study to examine the effects of Multi-Level dives allowed by the EDGE decompression meter on human subjects. The results were said to have shown that the profiles tested were safe to all the divers exposed. The work referred to was presumably that supplied by the manufacturers in a paper entitled "Doppler Evaluation of Multi-Level Dive Profiles" by Carl E Huggins.[4] Doubts expressed by the same author, regarding the safety of multi-level diving were not reflected in the promotional material, even though this paper is quoted in the references.

It is stated in the manual that the EDGE is not a guarantee of avoiding the bends. It is claimed that the experience from thousands of dives indicates that the EDGE is a better bet than the US Navy tables. It is said that until August 1984, no cases of bends had been reported. The manual has been modified since then, but this quotation remains despite the manufacturers being aware of such cases.

A comparison of the no-decompression limits with both the US Navy tables and the Royal Navy tables at the end of the manual infer that the EDGE is more conservative or "safer" than the US Navy tables at all depths from 30-140ft. On the same page, a comparison of selected depths in metres shows the EDGE to be equal or more conservative than the Royal Navy no-decompression limits at all depths.

In the Questions and Answers section, the manufacturers suggest that it is a good idea for sport divers to add extra safety factors, eg. not getting closer than 5 or 10 minutes to no-decompression limits. This would presumably exclude all no-decompression dives in excess of 120ft, although the manufacturers do not draw this conclusion from their advice.

The brochure stresses the importance of dive planning, wearing back-up depth and time measuring devices and regular confirmations of the calibrations. There is a very clear disclaimer, without limitation, exonerating both the seller and the manufacturer from any liability for personal injury resulting from the use of EDGE.

Popular skindiving magazines, both in articles and by advertising, have supported the promotion of the EDGE. In 1985, Murphy[6] stated that the computer programme is based on entirely new technology. The article claims that "those who use on swear by it and the instrument's safety record is impressive". It quotes Dr Bruce Bassett as describing the EDGE as a "revolutionary electronic device that may change the destiny of divers". These authors would not contest the claim, but would point out that it is ambiguous.

There is some difference of opinion in the claims made for the EDGE software. Murphy states that "one thing that has not changed over the years is the software computer programme contained in the EDGE". In the documentation obtained from the manufacturer, it was clearly stated that those instruments shipped after 1 September 1986 have improved software called "Div 4", which gives a modified rate of ascent indicator plus two other small changes. The ascent rates now recommended are:

> 60ft (18m) per minute in the >120 ft (36m) range
> 40ft (12m) per minute in the 60-120ft (18-36m) range
> 20ft (6m) per minute in the 0-60ft (0-18m) range.

The temperature reading was converted to Fahrenheit rather than Celsius, and the limits of the slowest tissues were increased slightly.

In 1987, the Australian diving newsletter *In Depth*, in articles by Harper and Latimer, extolled the virtues of the meters and a report in *Undercurrent* [7] is complimentary to the EDGE. Like the other magazines, the support is made without recording any specific testing of dive profiles.

METHODS

The depth function of the meters was compared to the gauge readings on the main therapeutic recompression chamber. Agreement was within the claimed errors of the gauges. The clock function of the meters was checked against the Telecom time signal and found to be accurate (no errors were detected over a three hour period).

For all the following tests, the meter's depth readings and timings were used by the operators controlling the chamber, with the chamber's gauge and a chronometer as a check. No discrepancies in these functions were observed.

Four EDGE "dive computers" were available for assessment. These were:

Number 1452. This equipment had been used for many months, had shown no mechanical problems, and was regarded with high esteem by its owner. It had been used during a dive in which a diver had developed decompression sickness and required recompression therapy.

Numbers 4167 and 4170 were new and supplied direct from a potential Australian distributor.

Number 0085. This was an Orca EDGE decompression meter simulator. It allows a simulation of dives, without the necessity of a compression chamber and was found to accurately simulate the readings observed on the meters tested.

As an additional check, the three dive meters were subjected to the same hyperbaric exposure, and comparisons were made between the readings of maximum depth, dive time and no-decompression time, surface interval and the scrolling depth and time allowed for repetitive dives. As well as this, in the first series of dives, one of the meters was placed in an ice water bath before the hyperbaric exposure.

All the dives were carried out in a compression chamber, with direct observation by two or more researchers.

It was decided to restrict the dives to no-decompression exposures, and always to commence the ascent prior to the expiry of the no-decompression, as shown on the meter.

The descent rates were kept at 18m (60ft) per minute and the ascent rates were in accordance with the new, modified ascent recommended by the manufacturer as described above.

The depths chosen in the first series were constant, ie. there was no variation in the depth between the first and subsequent dives. The three depths chosen for testing were: 17m (56ft), 31m (102ft), and 43m (141ft), ie. depths halfway between table depths used in the RNPL/BSAC tables, in an attempt to reduce the bias in either direction.

The second series included repetitive dives to different depths.

The third series involved multi-level diving, ie. staying at different depths during the same dive. This duplicated the repetitive dive profile that caused decompression sickness in a diver, and led to the initiation of the project.

NONE of the dives tested required decompression according to the meter.

RESULTS

DIVE PARAMETERS

Dive parameters, including depths, maximum depth, durations, surface intervals and temperatures, were recorded accurately. No significant discrepancies between meters were observed.

SINGLE DEPTH DIVE

The no-decompression times permitted by the meters were compared to those depicted in the manual and seen during the "scrolling" of the meter on the surface. Only small discrepancies were noted.

Although the manual states that at 60ft the no-decompression time is 53 minutes, in practice it is 54. At 90ft it is stated to be 24 minutes, in practice it is over 25. At 120ft the no-decompression time is stated to be 11 minutes, whereas in practice it was 12. At 140ft the no-decompression time was stated to be seven minutes, whereas in practice it was nine. At 150ft, the no-decompression time was said to be seven minutes; in practice it was over eight.

REPETITIVE DIVES TO THE SAME DEPTH

Ten repetitive dive combinations were performed without requiring decompression according to the EDGE meter. These are reproduced as tables 1, 2 and 3.

In all the repetitive dive series performed above, decompression was omitted with the use of the EDGE meter, compared to the US Navy and RNPL/BSAC tables. However, in comparing the omitted decompression from both US Navy and RNPL/BSAC tables, two minutes extra decompression could be credited to the EDGE for each dive to make allowance for the slower ascent rate at the shallower depths. If this is done, the results are as follows:

In the repetitive dive series to 17m (56ft) there was omitted decompression of between 10 and 46 minutes (US Navy) and between 66 and 302 minutes (RNPL/BSAC).

TABLE ONE - REPETITIVE 17M (56 FT) DIVES

DIVE NUMBER	BOTTOM TIME	SURFACE INTERVAL	OMITTED DECOMPRESSION STOPS US NAVY	RNPL/BSAC*
1a	60 mins	60 mins	0	10 mins
2a	45 mins	11 mins	14 mins	90 mins
3a	8 mins		26 mins	90 mins
1b	60 mins	120 mins	0	10 mins
2b	56 mins		14 mins	60 mins
1c	60 mins	60 mins	0	10 mins
2c	45 mins	11 mins	14 mins	90 mins
3c	8 mins	120 mins	26 mins	90 mins
4c	41 mins		14 mins	120 mins

* BSAC 18m table was used until the maximum tabulated bottom time was exceeded, then RNPL 20m table was used.

TABLE TWO - REPETITIVE 31M (102 FT) DIVES

DIVE NUMBER	BOTTOM TIME	SURFACE INTERVAL	OMITTED DECOMPRESSION STOPS US NAVY	RNPL/BSAC*
1a	17 mins	300 mins	0	0
2a	17 mins		3 mins	10 mins
1b	17 mins	120 mins	0	0
2b	17 mins	277 mins	7 mins	15 mins
3b	17 mine		7 mins	115 mins
1c	17 mins	60 mins	0	
2c	17 mins	37 mins	23 mins	30 mins
3c	15 mins	134 mins	54 mins	105 mins
4c **	17 mins		34 mins	155 mins
1d	18 mins	30 mins	0	0
2d	16 mins	30 mins	23 mins	30 mins
3d	13 mins	30 mins	54 mins	105 mins
4d	11 mins	30 mins	54 mins	125 mins
5d	8 mins		54 mins	155 mins

* BSAC 32m table was used until the maximum tabulated bottom time was exceeded, then RNPL 35m table was used.

In the repetitive dive series to 31 m (102ft) there was omitted decompression of between -1 and 175 minutes (US Navy) and between 6 and 315 minutes (RNPL/ BSAC).

In the repetitive dive series to 43m (141 ft) there was omitted decompression of between 20 and 275 minutes (US Navy) and between 112 minutes and "off the page" (RNPL/ BSAC).

REPETITIVE DIVES TO DIFFERENT DEPTHS

Repetitive dive combinations were performed which did not require decompression, according the EDGE meter. These are displayed in table 4 (page 125). Omitted decompression in this series was considerable, and far in excess of that which could be credited because of the slower ascent rate of the EDGE.

Table 5 shows an empirically unacceptable repetitive diving combination, which can be performed without any decompression according to the EDGE meter. The combination of dives would have required 100 minutes of decompression (including ascent times) according to the US Navy tables and over five hours according to the Royal Navy tables.

To avoid the safety factors inherent in "rounding up" of

TABLE THREE - REPETITIVE 31 M (102 FT) DIVES

DIVE NUMBER	BOTTOM TIME	SURFACE INTERVAL	OMITTED DECOMPRESSION STOPS US NAVY	RNPL/BSAC*
1a	7 mins	60 mins	1 min	0
2a	7 mins	105 mins	9 mins	10 mins
3a	7 mins	165 mins	9 mins	25 mins
4a **	7 mins		9 mins	85 mins
1b	7 mins	30 mins	1 min	0
2b	7 mins	30 mins	9 mins	10 mins
3b	7 mins	30 mins	32 mins	25 mins
4b	7 mins	30 mins	57 mins	185 mins
5b	7 mins	30 mins	57 mins	105 mins
1c	8 mins	60 mins	1 min	0
2c	8 mins	60 mins	9 mins	15 mins
3c	8 mins	60 mins	21 mins	55 mins
4c	8 mins	60 mins	32 mins	105 mins
5c	8 mins	60 mins	57 mins	115 mins
6c	8 mins	60 rains	57 mins	160 mins
7c	8 mins	60 mins	57 mins	off tables
8c	8 mins	60 mins	57 min	off tables

* BSAC 44m table was used until the maximum tabulated bottom time was exceeded, then RNPL 45m table was used.
** Dive combination ‘a’ was repeated twice; the same results being obtained each time.

TABLE FIVE

First dive	15m (49 ft)	duration 75 minutes	surface interval 3 hours
Second dive	25m (82 ft)	duration 25 minutes	surface interval 2 hours
Third dive	35m (115 ft)	duration 10 minutes	surface interval 1 hour
Fourth dive	45m (148 ft)	duration 8 minutes	

TABLE SIX

REPETITIVE DIVES CHOSEN TO AVOID ANY SAFETY FACTORS FAVOURING THE TABLES COMPARED TO THE EDGE DUE TO THE ROUNDING-UP OF DEPTHS, DURATIONS OR SURFACE INTERVALS

DIVE NUMBER	DIVE DEPTH	BOTTOM TIME	SURFACE INTERVAL	OMITTED DECOMPRESSION STOPS US NAVY	RNPL/BSAC*
1a	70 ft	40 mins	67 mins	1 min 10 sec	3 min 15 sec
2a	110 ft	10 mins	32 mins	8 min 50 sec	4 min 15 sec
3a	70 ft	30 mins		19 min 10 sec	3 min 15 sec
			TOTAL:	29 min 10 sec	10 min 45 sec
1b	120 ft	15 mins	46 mins	2 mins	4 min 30 sec
2b	120 ft	10 mins	34 mins	8 mins	4 min 30 sec
3b	120 ft	15 mins	27 mins	32 mins	4 min 30 sec
4b	120 ft	5 mins		32 mins	4 min 30 sec
			TOTAL:	74 mins	18 mins

TABLE FOUR - REPETITIVE DIVES TO DIFFERENT DEPTHS

DIVE NO.	DIVE DEPTH ft	m	BOTTOM TIME	SURFACE INTERVAL	OMITTED DECOMPRESSION STOPS US NAVY	RNPL/BSAC*
1a	56	17	60 mins	30 mins	0	10 mins
2a	108	33	12 mins	102 mins	34 mins	155 mins
3a	56	17	38 mins		4 mins	off tables
1b	102	31	16 mins	219 mins	0	0
2b	122	37	10 mins	14 mins	4 mins	10 mins
3b	132	40	6 mins		26 mins	85 mins
1c	62	19	50 mins	30 mins		10 mins
2c	102	31	11 mins	180 mins	34 mins	155 mins
3c	102	31	13 mins	30 mins	23 mins	155 mins
4c	62	19	35 mins		33 mins	off tables
1d	55	17	60 mins	60 mins	0	10 mins
2d	115	35	10 mins	60 mins	30 mins	155 mins
3d	55	17	45 mins	60 mins	26 mins	off tables
4d	115	35	5 mins	60 mins	30 mins	off tables
5d	115	35	10 mins	60 mins	46 mins	off tables
6d	115	35	0 mins		46 mins	off tables
1e	49	15	75 mins	180 mins	0	10 mins
2e	82	25	25 mins	120 mins	18 mins	85 mins
3e	115	35	10 rains	60 mins	30 mins	off tables
4e	148	45	8 mins		57 mins	off tables

* BSAC tables were used until maximum tabulated bottom time was exceeded, then RNPL tables used.
"Off tables" indicates that bottom exceeds the bottom times permitted by the table.

depths, durations and surface intervals with the use of the US Navy tables, two repetitive dive series (Table 6, page 124) were carefully chosen so as to avoid such safety factors.

With these two exposures, the EDGE omitted decompression and ascent time of 19 and 56 minutes compared to the US Navy table.

MULTI-LEVEL DIVING

The acceptability of single multi-level dives could not be assessed in the absence of any tested authoritative standards for comparison, however, it is considered that repetitive multi-level diving would have at least similar problems to repetitive fixed level diving, using the meter.

A multi-level repetitive dive profile was performed without requiring decompression according to the EDGE meter. It is shown as table 7 on page 126.

Converting time at different depths using the US Navy residual nitrogen proposed by Graver[8] indicates a stop of 14 minutes at 10ft (3m) on the second dive. The EDGE ascent rate (4 mins from 90ft) constituted some decompression for each dive.

This was a reconstruction of a dive schedule in which a diver using the EDGE meter was "bent" and required recompression treatment. The dive was considered safe according to the meter, but unsafe according to the US Navy tables (omitted decompression of over 30 minutes). Using multi-level dive calculations by Graver,[8] there was an omitted decompression of 14 minutes by the residual nitrogen method.

DISCUSSION

This study compared the EDGE to the established tables, as stipulated in the diving manuals, to determine its relative safety. No judgement is made of its adherence to the theoretical principles on which it or the tables were originally based. The US Navy and Royal Navy/BSAC tables have been tested, and have an acknowledged decompression prevalence. The relevance of theories of Haldane and others, including the half times, number of tissues, Doppler data, etc. is conjectural and still requires clarification.

The results showed that the meters were less conservative than the tables, and would result in repetitive dives which proponents of the established decompression tables would consider unacceptable.

TABLE SEVEN - REPETITIVE MULTI-LEVEL DIVES

DIVE ONE

87ft (26.5m) for 15 minutes bottom time. Ascend to 60ft (18m) in one minute, and remain for 15 minutes. Ascend to surface in six minutes with a precautionary stop at 10ft (3m) for three minutes included in ascent time.

Surface interval of 90 minutes.

DIVE TWO

87ft (26.5m) for 15 minutes bottom time. Ascend to 60ft (18m) in one minute, and remain for 18 minutes. Ascend to surface in six minutes with a precautionary stop at 10ft (3m) for three minutes included in ascent time.

Apart from the observations that the EDGE allows diving protocols that appear both radical and dangerous, there are many promotional claims and theoretical arguments that are contentious.

SINGLE DIVES

On the surface, scrolling of the no-decompression times for the EDGE for any depth ("bottom time") is usually an underestimate of the actual time that is available to the diver using the meter, probably because less nitrogen is absorbed during the descent than while at maximum depth. The "bottom time" is, by convention, a summation of these times.

The "bottom time" recorded in the EDGE manual and used for favourable comparison with other dive tables' no-decompression times is misleading. It does not include the time taken to reach depth. Descent rate is conventionally accepted as 60ft or 18m per minute. To obtain the bottom times used in the manual, it appears that the manufacturers have presumed that the diver is instantaneously transported to that particular depth. The result is that the more gradual nitrogen load experienced with descent, when added to the actual time at the bottom, gives a greater "bottom time" for the EDGE than the manual or scrolling depicts.

The manufacturer's selection of depths to compare the EDGE with US Navy and the RNPL/BSAC tables resulted in the "rounded up" depths being used, thereby showing the EDGE in a more favourable light than if random depths were chosen, ie. if a depth of 18m or 60ft is chosen, then the EDGE looks more conservative than the US Navy tables, or the RNPL/BSAC tables. If, however, a depth of 141 ft or 43m is chosen, then the advantage of the EDGE decompression is immediately lost, as the decompression according to the US Navy tables has to be carried out as if the dive was at 150ft, and with the RNPL/BSAC tables as if the dive was at 44m. In these cases, the no-decompression limits are more conservative with the established tables than with the EDGE. Thus the depths chosen for comparison will have a great bearing on the apparent safety of one procedure compared to another.

The same anomaly is found with no-decompression durations, ie. with a no-decompression dive for 20 minutes, the US Navy will permit a dive to 100ft, the RNPL/BSAC table allows no-decompression to 30m, and with this duration the EDGE compares favourably with the other tables. If, however, a six minute maximum depth no-decompression time is chosen, then the EDGE would allow 160ft depth, whereas the US Navy allows only 140ft. If a maximum depth no-decompression dive of 24 minutes was chosen, the EDGE would compare less favourably and allow a greater depth than the RNPL/BSAC tables.

When one considers these three factors, and modifies the EDGE no-decompression limits accordingly, it is evident that the EDGE no-decompression fixed level diving is less conservative than both Bassett and Spencer tables, although both these are quoted in the manual.

Even without such corrections, the comparison of Spencer's no-decompression limits with those of the EDGE, does not really lend support to the claim that the EDGE is based on Spencer's figures. In the 30-80ft range, the EDGE allows the same or more time without imposing decompression requirements. Spencer's exposures do not exceed 130ft, but at that depth the EDGE allows almost twice as much time as Spencer. Although Spencer's work is quoted on many occasions in the manual, the manner in which the two are related is not clear.

SAFETY FACTORS WITH ESTABLISHED TABLES

With the use of diving tables, there is no possibility of the tables encompassing the vast numbers of combinations of depths and durations available with the EDGE meter. The tables use increments of water depth and time segments, thereby compelling the diver to "pigeon hole" his dive into one of the established depth/duration "boxes".

One of the most obvious safety factors is the "rounding up" of the depth and duration so as to decompress according to a greater depth and greater duration. Thus, if a diver descends to a depth of 17m (56ft) for a period of 62 minutes, he will decompress as if he has been to 18m for 66 minutes (RNPL/BSAC tables), 20m for 65 minutes (RN 1972 tables), or 60ft for 70 minutes (US Navy tables).

This rounding up results in a safety factor in favour of the established tables. In each case, as one approaches the designated depths and durations, the less safe the dive will be, as more inert gas is absorbed into the tissues for the same decompression obligation.

This safety factor contributes to the relatively acceptable results when divers use these tables. Attempts to use the maximum depth/duration to approach the no-decompression limits, have resulted in unacceptable incidences of decompression sickness.[9,10] As it calculates decompression requirements for the precise depth and time, this safety factor is omitted with the EDGE diving computer.

Although the slower ascent rate with EDGE may be of benefit in reducing the danger of pulmonary overload with venous gas emboli, it will also add to the nitrogen load in the tissues, when performing repetitive dives.

MULTI-LEVEL DIVING

Huggins' report[4] receives acknowledgment by the manufacturer as a theoretical basis of the meter's development. Four of the ten profiles Huggins used finished with significant stops at 25ft or 30ft (8 or 9m). These would act as decompression stops. Huggins states "[t]his study is only the first step in validating the Multi-Level diving procedures. More research needs to be conducted to increase sample size". An interest in the Multi-Level tables has been expressed by the US Navy, and perhaps trials by this group may clarify the issues.

Huggins' dive schedules could confuse the effect of repetitive dives with multi-level dives. It seems that there is little sound experimental evidence for any multi-level calculation system.

For a single multi-level dive in which the depth plateaus are gradually diminishing, ie. five minutes at 120ft, 20 minutes at 60ft and 30 minutes at 30ft, decompression would not be considered necessary by most authorities and was not required by the meter.

If, however, the opposite situation is produced, ie. the dive gets deeper as it progresses then the nitrogen load in the "slower" tissues is likely to contribute more than usual to bubbles which are subsequently developed in the "fast" or "medium" tissues during or following ascent. These multi-level tables have yet to be competently tested.

REPETITIVE DIVES

"Rounding up" of surface intervals with the US Navy tables also adds a safety factor over the EDGE, with repetitive diving. A dive to 60ft (18m) for 20 minutes would be interpreted as moving into repetitive group D according to the US Navy manual, and therefore a surface interval of, say, five hours, would be calculated in the US Navy diving tables to be equivalent to a surface interval of two hours and 39 minutes, ie. moving into group B. According to the RNPL/BSAC tables, it would be calculated as a four-hour surface interval. With the EDGE, it is evaluated strictly as a five-hour surface interval, ie. the EDGE loses the safety factor applied in both other tables.

Because of the loss of safety factors involved in "rounding up" depths, durations and surface intervals, the EDGE is likely to require much less decompression time with most arbitrarily chosen repetitive dive profiles. This must make it more dangerous to use than the tables, which incorporate these safety factors.

Even when dives are chosen specifically to avoid the safety factors inherent in the US Navy tables, the EDGE still allows much greater durations for repetitive dives. This is demonstrated by the dive series in Table Six.

The more radical nature of the EDGE can also be demonstrated by recalculating the 102 or 141 ft dive series of Tables Two and Three. The US Navy tables still require decompression stops, even when the next shallower depth, the next shorter bottom time, and the next longer surface interval are used. Because of this, the EDGE must be considered unsuitable for repetitive dives.

FUTURE METERS

It is considered that the programme of the decompression computer should incorporate:

1. A safety margin in the model equivalent to the "rounding up" of depths and durations to those designated in the established tables, eg. 64ft depth should be read by the computer as 70ft. This would ensure that the meter does not exceed the durations allowed by the tables, and thereby increase the likelihood of decompression sickness.

2. In repetitive diving, the meter should be at least as restrictive as the US Navy tables.

3. Once descent has been completed in the multi-level dives, no subsequent descents should be permitted from that or any other plateau depth, until multi-level diving is better researched.

CONCLUSIONS

Single fixed-depth no-decompression dives allowed by the EDGE are comparable to the established US Navy and RNPL/BSAC tables. In some instances, the bottom times are more conservative than the tables; at other times, they are more radical. The comparisons, as quoted in the Instruction Manual, give an impression of safety with the EDGE meter, which is somewhat misleading.

The acceptance of the EDGE in the use of a single multi-level dive, depends on one's philosophy or approach to these theoretical dive tables. The EDGE meter, used on certain multi-level single dives, may give greater durations without greater decompression stress, eg. when the dive is performed in such a way that the depth lessens as the dive progresses.

For repetitive dives with either single or different depths, and using either the US Navy tables or the Royal Navy tables as a minimal acceptable standard for decompression, the EDGE meter could not be classified as either safe or acceptable. This is so even when the "rounding up" and safety factors are not applied.

SUMMARY

The EDGE seems suitable for measuring and recording the various dive parameters, such as depth, times, temperature, etc. It seems suitable for some single fixed depth dives and on some single multi-level dives, if sufficient care is taken to ensure a sensible dive plan, eg. diving from deep to shallow.

Its use in any repetitive dive situation, with either fixed or multi-level dives, should be discouraged.

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DIVER NAVIGATION BY MEANS OF ACOUSTIC BEACONS

Harry Hollien

SUMMARY

Divers traditionally have difficulty navigating underwater. In air, they have vision plus all types of sensory cues to accomplish this task. However, when submerged, the diver's visual modality is sharply impaired and in a sense, he or she is left virtually blind. Ordinarily divers attempt to navigate by compass (dead reckoning) but research has demonstrated that this approach leads to unacceptable errors. Some other approach, then, needs to be developed. In this regard, we have carried out and reported a number of experiments focused on the abilities of divers to navigate by means of programmed acoustic signals. It has been found that sound which "moves" underwater (ie. via the UAPP or Underwater Auditory Phi Phenomena) greatly aids sound localization and, ultimately, navigation. Indeed, for diver retrieval this phenomena is so powerful that no subject in any of our experiments has ever swum to an area except that containing the signal source. Previously published data will be reviewed briefly and new data on the effects of experience and/or training on diver navigation by acoustic signal will be presented.

INTRODUCTION

Diver navigation and retrieval of personnel continues to be a very serious problem. At present, only a very few partially developed systems are available (explosives, dead reckoning, beacons, etc.) that will permit even the most limited (controlled) travel underwater. This situation results from the fact that, when a person is submerged, there are very few (to no) location markers and his or her vision is sharply limited. That is, in the normal situation (ie. in air), humans utilize their vision for observing markers, localizing objects and moving from place-to-place. Underwater, however, human vision is greatly limited, the diver quite often is functionally blind or close to being so. As stated, the consequences of this condition are quite serious; divers often are unable to locate objects or team members, swim to desired locations/targets and/or find their way "home". This latter problem can be a pretty grim one if the diver is saturated. Traditionally, the solution to the problem has been the use of an underwater compass with the diver navigating by "dead reckoning". However, Anderson[1] has reported an experiment wherein he states that "even for well-trained subjects ... the average performance accuracy ... was plus or minus 53 feet from the centerline of the measurement array or 3.98 degrees in compass error ... in an operational situation when a diver might be engaged in an underwater search task or in accurate placement of underwater sensors, this level of performance would be marginal." Indeed so. A navigational error of this magnitude would become crucial, and possibly fatal, for saturated divers or divers attempting to find a moving vehicle. To illustrate, if a saturated diver made an error in navigating back to the underwater habitat as large as that reported by Anderson, he could easily miss it, and

being saturated he would be unable to surface and reorientate himself. As a matter of fact, navigational errors of this size would prove undesirable under almost any underwater situation. Further, due to the nature of diving, the use of complex, bulky (and often unreliable) electronic systems for navigation has proved to be but minimally effective. Hence, we suggest that some other type of sensory mechanisms be substituted for vision to compensate for the cited deficit. Specifically, we propose that it is possible to utilize the diver's sound localization abilities as a substitute.

DIVER HEARING

Knowledge about underwater auditory function is both sparse and primitive. It would appear that a great deal of data must be obtained before very many basic hypotheses and postulates can be (inductively) generated. We have found that, when attempting to predict underwater hearing behaviours and mechanisms, immersing humans resulted in effects that biased our predictions. In other cases, existing variables (noise, reflective surfaces, stress, etc.) appeared to change human behaviour in significant ways. Perhaps the most important fact is that it is impossible to directly duplicate normal research techniques underwater. Rather, a great variety of life support systems must necessarily be attached to the diver with their concomitant, and shifting, effects on the responses made to heard stimuli. On the other hand, we believe that current technology permits systematic and appropriate research to be applied to these problems with the result that reasonable solutions can occur. A brief review of some of the more important findings in this area of inquiry would appear appropriate to demonstrate that some useful concepts are already available.

Initially, there was a substantial question about the sensitivity and nature of underwater auditory function in humans. However, it has been shown recently that the auditory capability of the submerged ear is not nearly as impaired as was thought. For example, when divers are submerged, their hearing is conductively reduced but they do not experience neurological impairments. That is, although there is a loss of sensitivity, a diver can detect a sinusoidal signal between 125 and 8000 Hz at 60-70 dB SPL.[2,9] This sensitivity to sound is within the normal range for conversational speech in air at a distance of one foot. Thus, although underwater hearing is accomplished primarily by "bone conduction", hearing function otherwise is normal as speech reception thresholds relate normally (ie. plus 15 dB) to standard thresholds for sinusoids and speech discrimination is normal once the sound can be heard.[6,10,11]

Divers' sound localization ability also has been found to be far superior to that which was originally predicted,[11-13] as has the possibility that divers can navigate by sound.[11,12,14,17] The combined results of these experiments result in the suggestion that pulsed low-frequency sinusoids or glides (up to 1 kHz) and broadband noise are superior to other signals for localization purposes and there is no distance effect.[18] Further, it was found that the sensation of acoustic "movement" can be a powerful localization cue.[11,18-11] It also has been observed that the difference in the minimal audible angle (MAA) in air and water is less than 10 degrees and the difference between absolute localization precision in air and in water is approximately ±5 degrees.[16,22] Finally, Thompson and Herman[23] found that the pitch discrimination of divers does not differ markedly from that of listeners in air. Thus, a diver is potentially capable of identifying subtle pitch, quality and distance differences in acoustic signals and should be able to utilize this information in order to determine the location of various underwater sound sources.

On the basis of the several sets of data discussed above, it appeared that divers might be capable of localizing sound sources underwater and of potentially utilizing this ability to navigate. For example, if they were able to "home" on a beacon, a substantial improvement in underwater safety would result. Accordingly, we consider it profitable to study human behaviours and capabilities related to this issue. Moreover, data from a related set of experiments convinced us that we had inadvertently uncovered a perceptual characteristic related to underwater hearing that could provide a powerful aid to diver navigation and retrieval. As a consequence, we were able to hypothesise that a line array of underwater projectors, energized in sequence to produce an apparent auditory "movement", would provide an effective localization signal. In theory, the array would produce an Underwater Auditory Phi Phenomenon (UAPP) similar to that produced (visually in air) by landing light systems for aircraft runways or on theatre marquees. The resulting pilot investigations suggested that the Phi characteristics were of such good potential, that we developed and have partially carried out (nine major experiments) an extensive research effort designed to study its effectiveness relative to diver retrieval and navigation. This paper reviews the already published information plus reports new data resulting from one of the cited experiments.

METHOD

While much of our earlier work on sound localization and underwater distance estimation abilities, plus the pilot studies assessing acoustic beacons and the UAPP approach, was carried out in the ocean, most of the current studies were conducted in a quiet lake on a military reservation (Camp Blanding). Lake Magnolia is almost 1.5 by 1.0 km in size and slopes gradually to a large central area with a depth of nearly 15 m. It proved to be an ideal site for the highly controlled, basic research that we found necessary to carry out initially. For the cited experiments, three J-11 transducers were positioned 3m apart in a linear array at a depth of 7m perpendicular to a straight line 150m experimental range as seen in Figures 1 and 2 (page 129). The acoustic signals used in the "training" investigation were chosen from among those evaluated in pilot work and earlier experiments.[17] Specifically they were:

1. A 500Hz square wave of 500 ms duration and with 25 ms rise/decay times.

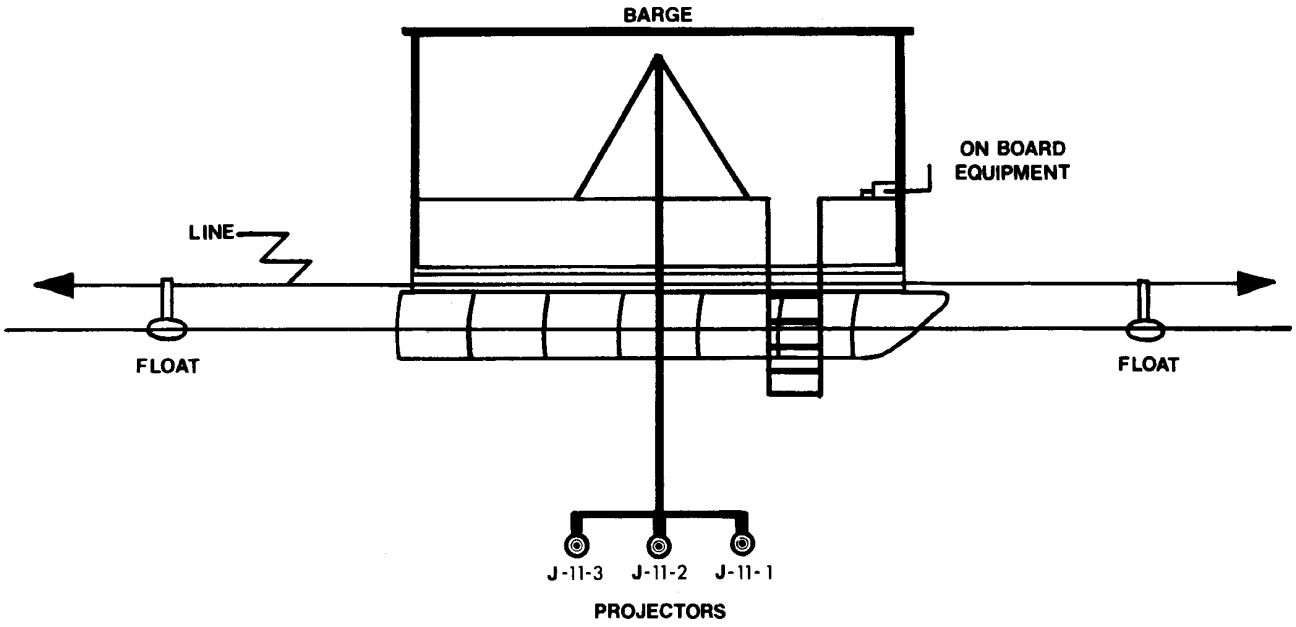

Figure 1. Schematic diagram of the barge, the transducer array and the plane (float to float) also included as part of the target.

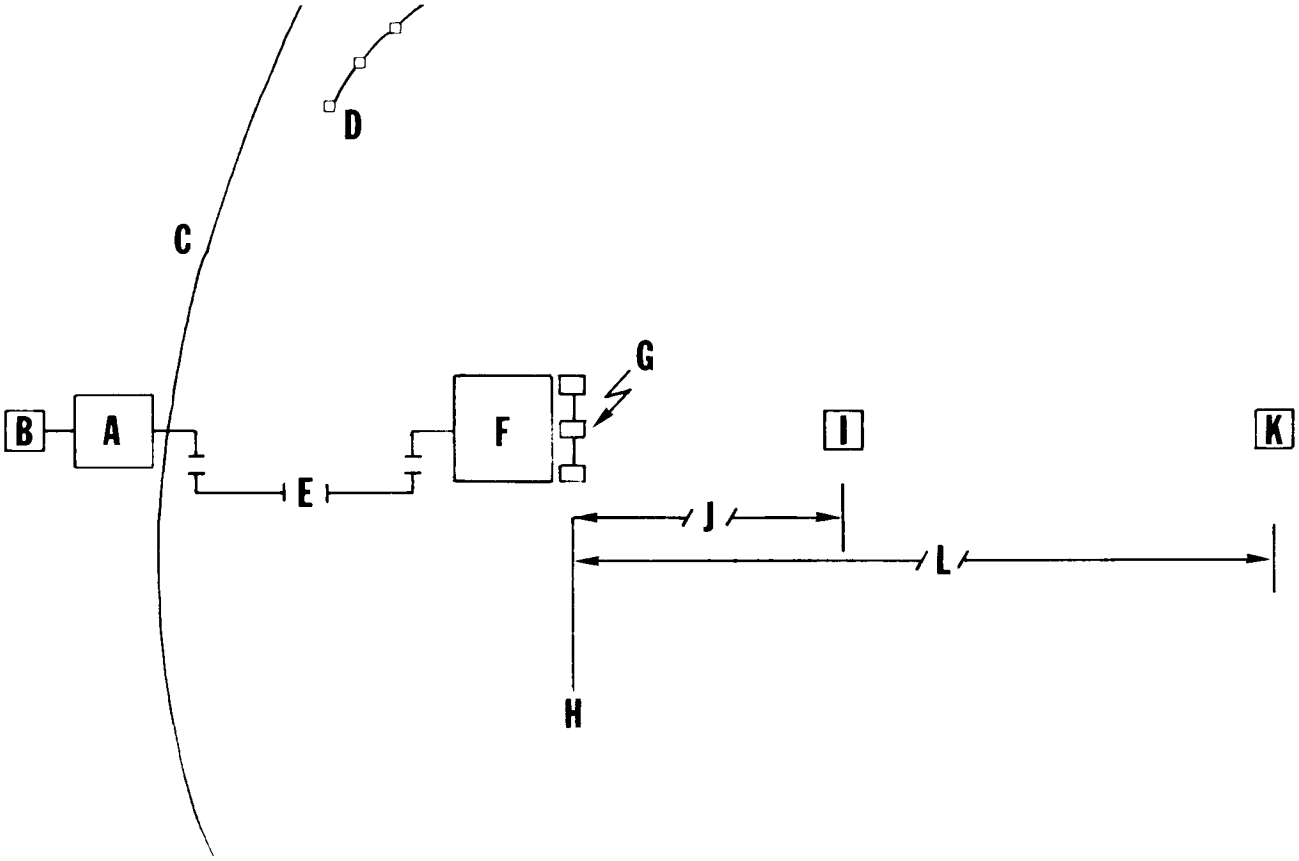

Figure 2. A schematic drawing of the range used in many of the UAPP experiments. A is the equipment van, B a generator and C is the shore. Line E carries the signal to the staging equipment on barge E. The J-11 s are placed 7m under the barge (see also Figure 1) and "hits" are counted when the diver passes line H (between the floats anyway). The range is depicted by L and the starting point by K (I and J refer to other experiments).

2. A 1.0 kHz square wave of 500 ms duration and with a 100 ms rise/decay.

3. A 0.2-2.0 kHz noise of 1 sec duration and with 50 ms rise/decay times.

Depending upon the particular study, the divers generally swam one or more trials involving:

1. a single beacon (SB) source,

2. a compass (C) dead-reckoning procedure (no acoustic signal),

3. a multiple beacon (UAPP) source, and/or

4. a procedure utilizing a compass in conjunction with one or more of the multiple beacon signals (MBC).

In all cases, the diver/subjects participated in two or more "visual" (V) swims in which they followed a line positioned along the bottom in a different part of the range. The visual trials ordinarily bracketed the other trials and the mean of the two swims was used to obtain base-line data relative to the time each diver would need to swim 150 m (see range D; Figure 2). As would be expected, the various multiple beacon (MB) conditions were counter-balanced across divers in order to minimize inadvertent learning effects.

Each diver (K) was transported by boat to the starting point located 150 m (or more) from the acoustic target (see again Figure 2). He descended to a depth of 7 m and was spun approximately three times by a buddy diver. He then indicated his preparedness to begin the trial by pulling several times on his safety line which was attached to a small buoy (he would tow the buoy during the entire trial). The safety line and buoy also served to maintain appropriate diver depth as the connecting line was 7m long and the diver was requested to keep a tight line between himself and the buoy. The diver was timed from his "ready"; signal to when he or she reached the acoustic target (G) or he swam past the vertical plane (H) of the transducers. The processes involved in these experiments can be understood also by consideration of Figure 3, at least from the diver's point of view. Subjects for these experiments were both male and female, trained and untrained divers drawn from the IASCP team and the University of Florida.

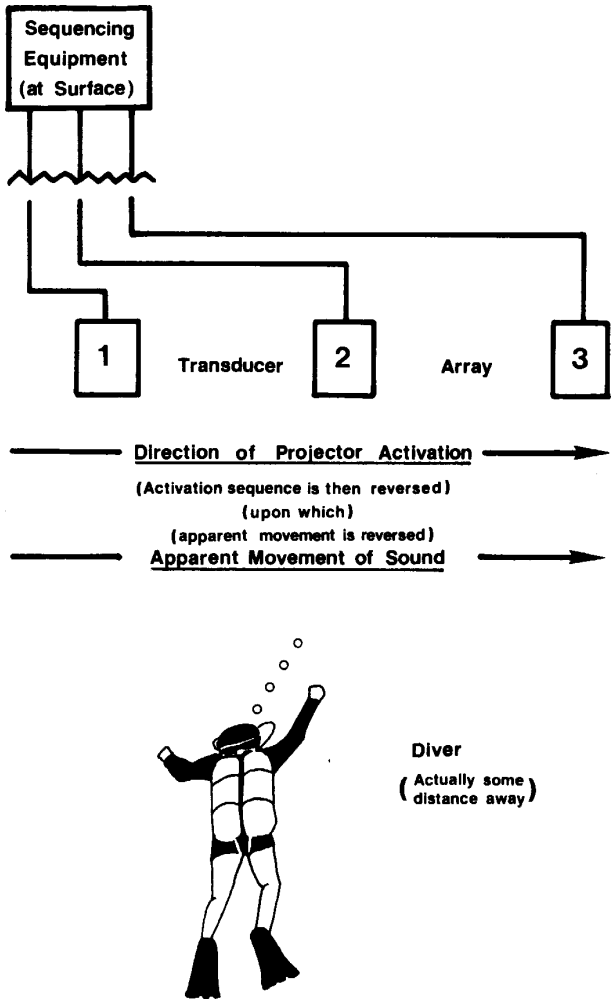

Figure 3. This drawing portrays the procedure by which the diver navigates. The sound attracts him to the target by first moving from left to right (from J-11-1 to J-11-3) and then from right to left (J-11-3 to J-11-1) and so on.

RESULTS

Basic Data

Table 1 provides basic data previously reported;[17] that is, it will serve as a summary for several of the earlier experiments. All values are proportions of the mean visual swim trials. It should be noted that values of less than 1.0 indicate a trial time which is faster than the visual swim and, conversely, trials with values greater than 1.0 required more time to complete than did the visual swim. As can be seen, the fastest trial (0.8) was shared by divers M-1 for the noise signal (MB3) alone (a hit) and M-7 for the compass swim (a miss), while the slowest time (5.7) was turned in by diver M5 in response to the single beacon (SB); this trial ended in a miss also. A hit was scored when the divers either reached the transducers (G in Figure 2) or bisected the transducer line between the buoys (H); a miss was scored when the diver stopped before reaching the buoy (even if only by a few feet) or missed the area between the buoys.

The primary conclusion that may be drawn from these and related data is that the divers were, in fact, able to effectively utilize the multiple beacon signals to navigate to the target. Indeed, while the times for some divers were occasionally high, no subject swam into any area other than that at, or adjacent to, the target. Furthermore, in many instances performance times are very close to those for the visual swims, ie. where the divers simply swam the 150 m distance following a line along the bottom. In short, it is clear that divers are able to "home" on the basis of heart stimuli.

It was expected after the pilot study, the multiple beacon

TABLE 1

Navigation scores for each diver and condition; values are proportions of visual swimtimes. Data for subject M-7 are incomplete as he did not return for the final set of trials.

CONDITION	M-1	M-2	M-3	M-4	M-5	M-6	M-7	MEAN	% HITS
C	1.7	1.5	1.2*	1.2*	1.7	1.5	0.8	1.37	29
SB	2.6*	2.4	2.7	2.7	5.7	1.9*	2.6	2.94	29
MB1	2.4	4.1	3.6	3.6	2.3	3.9	2.0*	3.13	14
MBC1	1.1'	2.2	3.1	1.1*	1.4*	1.6*	1.0*	1.64	71
MB2	2.6*	1.8*	5.2*	3.2	2.9	2.7*	—	3.07	67
MBC2	1.0'	2.9	3.5	1.3*	1.9	1.2*	—	1.97	50
MB3	0.8*	2.3*	3.9	1.1 *	2.4	1.9'	—	2.07	67
MBC3	0.9*	1.7	2.4*	1.1*	2.2*	1.4	0.9	1.15	57

MB1 = 500 Hz; 500 ms; 25 ms, MB2 = 1 = kHz; 500 ms; 100 ms, MB3 = N2k; 1 sec; 50 ms, * = hit

TABLE 1. Navigation scores for each diver and condition; values are proportions of visual swimtimes. Data for subject M7 are incomplete as he did not return for the final set of trials.

proved to be a more powerful cue than did the single beacon with the noise signal associated slightly better times and scores. Moreover, even though the times for the beacon swims often were slower than for dead-reckoning, accuracy was substantially greater and perhaps, most important, the beacons provided self-correcting information not available from a compass. Finally, since greater than expected variation was observed among diver performances, a learning function was suggested. That is, all divers showed improvement as the experiment progressed (no matter what the sequence of trials) with some showing greater improvement than others. Unfortunately, due to the structure of the early experiments, the nature of this training function could not be isolated. It was tested later and is reported below.

Training Effects

As stated, the results of the several previous experiments suggested that a much stronger learning function existed in the development of diver navigation by UAPP than was previously thought possible. Therefore, the effects of learning (or training) on the diver's ability to perform the cited tasks was studied. Only one MB signal was used in this experiment; it was the 0.2-2.0 kHz white noise of 1 second duration with a 100 ms rise/decay time and a 100 ms overlap. Nine certified divers served as subjects; four were experienced with underwater research on hearing and auditory localization whereas five were not. Training consisted of a lecture, a training trial with feedback and the multiple trials of the experiment itself. The diver's task was to navigate a 150 m course (using the cited beacon) either 10 times or until his performance plateaued. In addition to the acoustical trials, the diver also swam three 150 m visual trials (as swim speed controls). The diver's learning curve was assumed to have plateaued if he achieved consecutive trials in which:

1. Two were "hits" and arrival times were less than 1.5 of the mean visual swim time (VST);
2. three were hits and arrival was less-than or equal-to 1.5 of VST; or
3. three trials were within 3 m of the target and the times were less than 1.2 VST.

As with the earlier studies, a "hit" was defined as the diver navigating to the acoustical target, ie. arriving at one of the transducers, passing between them (the water was turbid and sometimes the subjects passed the target without seeing it) or passing between a J-11 and an outlying buoy.

The data from this experiment can be utilized to establish several relationships. First divers improved their performance as a function of continued trials and a diver's previous experience, or lack of it, with the underwater hearing research turning out not to be a factor in the determination of his or her performance. Thus, since the functions of all diver/subjects were similar, we would suggest that it should be just as easy to train naive divers to navigate acoustically, as it would be to train divers with previous exposure. Second, three measures of performance were utilized (time, angle and a time/angle composite). Of these, only the angle metric was found to dramatically measure change in performance. Specifically, swim times stabilized very quickly, ie. from 1.1 to 1.5 of visual swim by the second trial. A similar observation could be made for accuracy; indeed, there were only 4 per cent misses, and very close ones at that, in all the trials after the fourth and all divers arrived at one of the transducers 87 per cent of the time from the fifth trial on. In other words, divers improved in their navigational accuracy while maintaining fairly constant swimming rates and, as may be seen from observation of Figure 4 (page 132), most of the improvement in accuracy occurred within the first few trials. By that time, subjects had reduced the angle metric to about 1.5 degrees, which corresponds to an error of less than 4 m at

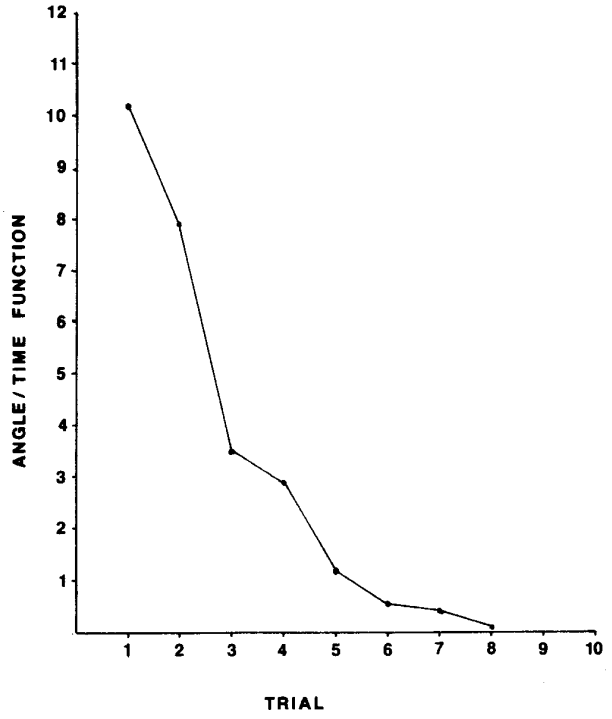

Figure 4. Graphed data depicting the function by which divers learn to navigate by UAPP. As may be seen, most learning takes place within the first few trials.

the transducers. Moreover, a particular feature of the multiple beacon approach is that it appears to be self correcting.

CONCLUSIONS

An integrated and systematic programme of research has been undertaken at the University of Florida in order to develop an operational system for acoustic diver navigation. As can be seen from the cited data, the research that has been completed to date has demonstrated that divers not only can localize sound underwater reasonably well but also can use this ability to navigate. To be specific, it was found that (1) when a multiple sound source was used to produce the Underwater Auditory Phi Phenomenon (UAPP), the divers are able to navigate to the target almost as well as if they had a visual line to follow and (2) even untrained divers could learn to "home" acoustically and do so very quickly. Finally, substantial progress has been made toward optimizing the parameters of the acoustic signals for the purpose of diver navigation and research in this regard is to be published soon.

Finally, two features of this approach should be stressed. First, when a UAPP signal constituted the underwater beacon, not a single diver ever swam to a sector other than that which contained the sound source. This relationship has been found to hold for all trials with in all experiments. Second, the procedure clearly is self-correcting, at least when the task is to bring the diver to a fixed source. We are now developing experiments which are designed to study the possibility that UAPP information may be employed to permit a diver to navigate freely underwater.

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Professor Harry Hollien, PhD is the Founding Director of the Institute for Advanced Study of the Communication Processes, University of Florida, Gainesville, Florida 32611, USA. His address is ASB-50, IASCP, University of Florida, Gainesville, Florida 32611, USA.

A SPUMS MEMBER IS HONOURED

We reproduce below the citation of the Craig Hoffman Memorial Award presented at The Undersea and Hyperbaric Medical Society (UHMS) meeting in Baltimore, Maryland, USA, in May 1987.

The Undersea and Hyperbaric Medical Society takes great pleasure in presenting

THE CRAIG HOFFMAN MEMORIAL AWARD

to

CARL EDMONDS

This award is conferred upon the recipient for significant contribution to diving safety. Dr Carl Edmonds has for over 20 years been a leader in the Australian diving safety community. His contributions to worldwide diving safety have benefited those involved in military, commercial, scientific and sport diving.

Dr Carl Edmonds accepting the Craig Hoffman Memorial Award.

Dr Edmonds' accomplishments cover the gamut of diving. His contributions to the field - marine animal injuries, his work developing the civilian diving medical courses in Australia and the development of in-water decompression techniques have all played major roles in diving safety.

Additionally, his worldwide involvement with the undersea medical community has provided a means of disseminating this works in diving safety for the benefit of all.

SPUMS congratulates Dr Edmonds, known to the diving world in Australia simply as "Carl", on being the first Australian to be given an UHMS International Award.

DIVING AND SAFETY

POLICY OF THE VICTORIAN ASTHMA FOUNDATION

Persons with asthma are at increased risk of potentially fatal lung complications from undersea diving. Diving itself may induce asthma attacks and asthma related diving deaths have been clearly documented. The exercise associated with diving, the changes in body temperature, the inhalation of dry gas mixtures and the potential for inhalation of saline may all play a role in triggering an attack of asthma in the hyperreactive airways of asthmatic subjects. Additionally, the occurrence of an asthma attack may lead to panic reactions with mishandling of equipment and errors of judgement.

The expansion of lung gas trapped behind narrowed airways in asthma during ascent from depth and decompression results in increased risk of tearing or rupture of the lung surface leading to pneumothorax (potentially of tension type), mediastinal emphysema and air embolism.

Similar risks of pulmonary barotrauma also apply to patients with lung cysts, emphysema, chronic airflow obstruction associated with chronic bronchitis or with smoking and lung scarring from any cause.

The Asthma Foundation of Victoria recommends the following policy:-

1. Any person contemplating undersea diving should have:-

   (i) medical examination by a doctor experienced in underwater and diving medicine (Guidelines for examination are given in References 2, 3 below)
   (ii) a full-plate chest x-ray
   (iii) properly performed spirometry
   (iv) advice regarding the risks of diving with asthma or other lung diseases.

2. Persons currently suffering from asthma or from symptoms of wheeze or chest tightness should be advised not to dive.

3. Persons who have a past history of asthma or wheezing attacks should not dive unless:

   (i) they have normal spirometry and a normal chest x-ray; and
   (ii) they have been demonstrated to have bronchial reactivity within the normal range. Bronchial reactivity is currently measured by broncho-provocation tests using methacholine or histamine.[7] Hyperosmolar saline inhalation challenge is currently undergoing evaluation as a further test of bronchial hyper-reactivity[8-9] and, if large population studies demonstrate it to have a high diagnostic sensitivity for asthma, may have particular relevance to the assessment of asthma in potential divers. Exercise testing provocation,[10] if positive, is useful but a substantial proportion (20-25%) of patients with clinically significant asthma do not have exercise hyper-reactivity. Provocation tests are best carried out in respiratory function laboratories experienced in their use and having established ranges for normality.

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LETTERS TO THE EDITOR

39 Oswald Street
ROCKHAMPTON QLD 4700

Dear Sir,

As retiring President of SPUMS I wish to convey my sincere thanks to the past Executive Committee who have served with me. Their expertise has made my two years as President enjoyable.

I would also like to take this opportunity to thank the organisers and participants involved with the 1987 Annual Scientific Meeting held in Honiara. l am sure that all who were present would agree that it was one of the best SPUMS conferences to date.

Yours sincerely

CJ Acott

39 Oswald Street
ROCKHAMPTON QLD 4700

Dear Sir,

Due to the failure of financial arrangements for both the June Conference on Heron Island and negotiations for another date, it has been decided to change the venue for next year's Annual Scientific Meeting.

It will now be held in Fiji in June 1988.

Guest Speakers (in alphabetical order) will be
Dr William Runciman
Dr Robert Thomas
Dr John Williamson.

Members who wish to present a paper should contact
Dr CJ Acott
39 Oswald Street
ROCKHAMPTON QLD 4700

Yours sincerely

CJ Acott

BOOK REVIEWS

The Diving Emergency Handbook, 3rd (revised) edition.
John Lippmann and Stan Bugg.
JL Publications, PO Box 381, CARNEGIE VIC 3163, Australia.
RRP $10.00

We reviewed this book when the first edition was published in 1985 and recommended it strongly. Since then a second edition has been produced, in 1986, for the USA market where it is sold as the DAN Emergency Handbook, with changes suggested by the Divers Alert Network (DAN) organisation. Sales in Australia have been such that reprinting was necessary and the authors have taken the opportunity to revise and enlarge the book and make it useful for most of the English speaking world by giving the emergency numbers for Canada, New Zealand, UK, and USA as well as for Australia.

Major changes have been required by the setting up of the Australian Diver Emergency Service (DES). Every diver in Australia should know the DES toll free number (008 088 200) and when the book is opened it is the first thing that catches the eye.

The text is in three sections, an index of signs and symptoms with their possible causes, a list of diving ailments with appropriate first aid, and an appendix covering many things. The pages dealing with arterial gas embolism, severe bleeding, the blue ringed octopus, decompression sickness, nitrogen narcosis, pulmonary barotrauma, and stings have been extensively revised to reflect current therapies and arrangements for hyperbaric treatment.

In the appendices the procedures for omitted decompression have been reduced to those for the USN tables and the use of 100 % oxygen at the surface, which is what our reviewer would do if he found himself in such a situation. The decompression table section features "Dr Bruce Bassett's Revised Bottom Times "No-decompression" Decompression Tables Arranged For Repetitive Diving by John Knight and John Lippmann". This table with its yellow highlighting is extremely easy to use and should be more widely used for the reasons given on page 46. The section on the administration of oxygen has been revised and is clear and easy to follow.

A useful feature, following the provision for the owner to enter useful telephone numbers, is the space for recording details of a diving accident. There are clear diagrams of the left-side-head-down position recommended for diving accident victims. As before there are clear flow charts of the first aid for diving accidents and the management of the unconscious person.

The revised edition has improved an excellent publication to the point where ever diver should buy a copy of the revised edition, even if he or she had a copy of the first edition. It is easily identified by the red REVISED across the [sentence unfinished in original Journal].

The Abalone Diver. Carl Edmonds.
National Safety Council of Australia, Victorian Division, 464 St Kilda Road, MELBOURNE VIC 3004, Australia.
1986. A Diving Medical Centre Publication. 209 pp.
Price $9.95.

This paperback is based on a survey conducted for the Fishing Industry Research Committee. Carl Edmonds was assisted by 18 other contributors. The book starts with a history of abalone diving and a description of the survey which covered 152 of the less than 300 active divers. The chapter on the profile of abalone divers and their diving habits is fascinating.

The survey revealed that the divers were critical of the absence of easily available technical and diving information and that they distrusted officialdom. Sensible and practical recommendations to improve the divers knowledge and the safety of their diving practices as well as for monitoring their health are found on pages 34 and 35.

Dysbaric osteonecrosis is well covered with a general review, which includes clear instructions for the taking of the necessary x-rays on page 44, and then a discussion of the incidence in the surveyed divers. This section closes with a discussion of joint replacement for dysbaric osteonecrosis.

Four chapters cover hearing loss, chronic ENT disorders and recommendations on how abalone divers should be cared for to prevent damage to nose and ears.

Eight chapters are devoted to the neurological and psychiatric sequelae of diving and provide interesting information. Chapter 17 reviews the divers treated at the Royal Australian Navy School of Underwater Medicine in an eighteen month period. It is of interest that neurological deficits could be detected, before treatment, in 68 (78%) of the 87 divers with decompression sickness. 84 of these had complete resolution of the acute signs and symptoms before being discharged from Naval care. Follow up arrangements were to review all patients one week and one month after the incident. Only 46 divers attended on both occasions, a drop out rate of 48%. Seven divers had developed neurological signs in the week and the three who were left with neurological signs after treatment were still abnormal. However at one month only 2 divers had detectable lesions. This clearly indicates the commonness of neurological injury with decompression sickness and its tendency to recur and then regress with time.

The final two chapters discuss miscellaneous medical diseases of abalone divers and make general recommendations for the better management of the divers' health.

This book should be compulsory reading for all doctors involved with abalone divers.

NEW ZEALAND CHAPTER OF SPUMS 1988 ANNUAL MEETING

Preliminary Notice
1 to 4 April 1988 at Furneaux Lodge in the Marlborough Sounds.

For further details write to the convenor of this meeting

Dr Mike Davis, PO Box 35, TAI TAPU, New Zealand

SPUMS ANNUAL SCIENTIFIC MEETING 1988.

Owing to circumstances beyond the control of the Executive Committee Heron Island is no longer the venue. The Annual Scientific Meeting will now be held in Fiji in June 1988.

Guest Speakers will be: Dr William Runciman, Dr Robert Thomas, and Dr John Williamson.

The conference organiser is
Dr CJ Acott
39 Oswald Street, ROCKHAMPTON QLD 4700

Members who wish to present a paper should contact Dr Acott and inform him of the title of their paper, how long the presentation will take and what sort of projector will be needed.

DIVER EMERGENCY SERVICE
008 088 200

The duty supervisor of the Intensive Care Unit at the Royal Adelaide Hospital will answer the telephone and when told that it is a diving emergency will contact the on-call diving doctor. The call will be diverted to the diving doctor who will offer the caller expert advice. Civilian and naval doctors experienced in the treatment of diving accidents from all over Australia will be taking part in DES.

The diving casualty should contact DES on 008 088 200. In most cases he will be advised to attend the local hospital unless he has easy access to one with a hyperbaric unit. That hospital will be contacted by DES with advice. The hospital will notify the nearest hyperbaric unit and arrange a hospital to hospital transfer. It will also notify the local ambulance service. If necessary the hyperbaric unit will alert the retrieval agency, such as the National Safety Council of Australia (Victorian Division) who have portable recompression chambers and aircraft to carry them. If specialist transfer is necessary the local ambulance service will arrange it with the retrieval agency.

The DES number 008 088 200 can only be used in Australia. For access to the same service from outside Australia ring ISD 61-8-223-2855.

THE MARINE STINGER HOTLINE
NEW NATIONAL NUMBER 008-079-909

The Marine Stinger Hotline is now toll free Australia wide. The old number was only available in Queensland. Arrangements have been made with Telecom to place a recorded message on the old number to direct callers to the new number.

For expert advice about the treatment of marine stingers dial 008 079 909.

THE SAFETY SAUSAGE

As anyone who has looked for a diver at sea knows, a diver on the surface is difficult to spot. There is now a modestly priced aid to diver location.

The Safety Sausage *(SPUMS J.* 1986; 16(2): 59), is a red plastic tube which can be inflated by the diver's regulator. It can either be held vertically in the water to indicate the diver's position to a boat or allowed to lie on the surface to aid recognition by aircraft. It is manufactured by TL Begg and Sons Ltd, PO Box 5216, Moray Place, Dunedin, New Zealand.

The Australian distributor of the Safety Sausage is RJ Knight Pty Ltd, 80 Wellington Parade, EAST MELBOURNE VIC 3002. They are available for members of SPUMS at $7.00 including postage. They will soon be available through dive shops.