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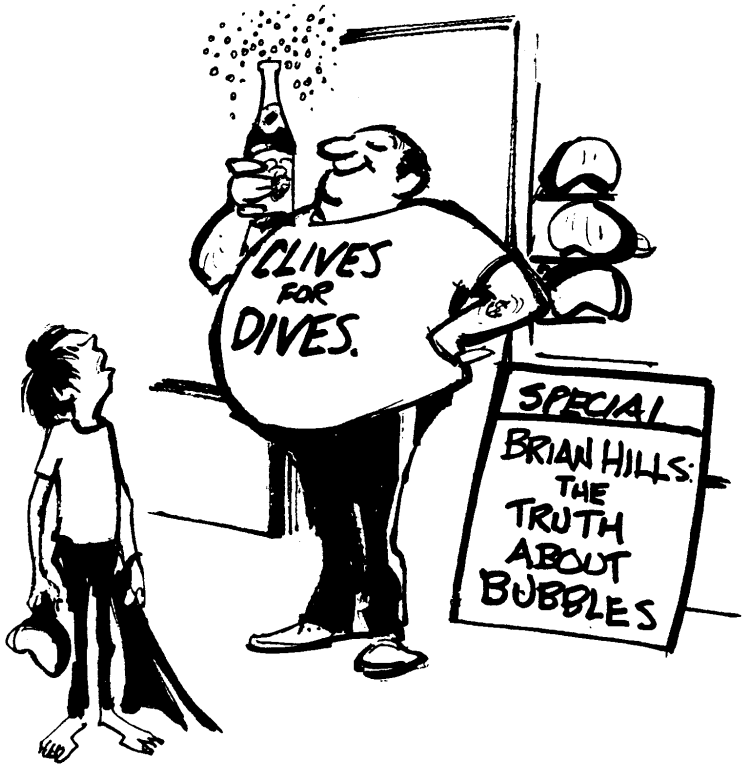

"IS UNCONTROLLED DECOMPRESSION YOUR PROBLEM, MATE?"

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EDITORIAL

Until recently everyone (nearly) thought that everything that needed to be known was known about decompression sickness (DCS). It was believed that it was the inevitable result of the sudden production of bubbles the moment one transgressed the dive profile laid down in the tables. The smart people were able to dive longer and deeper and ascend more rapidly, it was thought, if they chose a more generous table. All this was possible due to a mystery property of the body to "supersaturate", one of the most mischievous terms to enter polite medical discussion since the four humours fell into disrepute. The discovery that most dives using compressed breathing gases were followed by bubbling discernible by Doppler has gradually led to an understanding that there is an element of Russian Roulette in whether one does, or does not, suffer clinical decompression sickness following a dive near to the profiles suggested in the tables. Classical scholars may choose to believe that Nemesis is back at work as a kind of Diving Supervisor. It is likely that the divers at Townsville who suffered Type II DCS feel that they did little different from those who suffered no ill effects. Comment must be reserved on this matter until more is known, however. Certainly only an ignorant, or foolhardy, person now fails to add into their dive table calculations a generous element of leeway in stating the depth and time of their dive, if only because the Authorities are becoming less amused at having to provide treatment and more open about the imperfect results of such therapy.

*It is obvious that the subject is much more complicated than was originally thought, so Brian Hills' review of Decompression Physiology is both timely and welcome. He deals with the question of site of the lesions which result in symptoms as well as the critical changes. At least we are still left with the bubbles as primary villains! Almost as an aside he directs our attention to the lucky chance that our lungs can survive the task of acting as bubble filters (usually), as otherwise we would be limited to the shallows. The corollary, that the presence of a patent foramen ovale (not too rare, apparently) or a circulation which by-passes the lungs (as in the foetus), enables bubbles to reach the brain, has considerable clinical significance. It is a readily acceptable **a**nd unforgiving consideration to be faced by the pregnant diver, in particular during the earliest weeks when the CNS, limbs, and other organs are being differentiated.*

The first of the papers given at the "PENGUIN" meeting to reach print is that given by John Pennefather. It is highly instructive to read of the events behind the smooth reports which are later published. There is a serious message within the humorous coating, that many "facts" taken on trust as basis for research may themselves be suspect and ill based. The unwritten assumptions included the belief that food intake would be the same however it was cooked and presented. The unintended(?) effect of introducing a new element, the observers, had at least a short term effect on the catering, and the intake of food by the participants. It also shows that with persistence and sufficient care satisfactory results can be obtained by motivated experimenters.

Overseas news includes an update on the training of disabled divers, while information from Norway reminds us of the several other routes to improve safety, in addition to strictly diving factors, which are being explored. The spin-offs from the North Sea Oil Industry's difficult environmental problems continues. Near at hand, in Fremantle, there has been a course to train Diver Medics. This, coupled with the intention to train divers to "North Sea Employability Standards" in Australia, will be a great advantage to those seeking to make a living (the word is meaningful) in the seas distant from Australia. The growing acceptance of the value of diver medics to provide immediate informed assistance to diving casualties has followed years of frustrating effort by the Commercial Diving Center which is now the College of Oceaneering, in the USA, and others. It is hoped to publish a report on the evolution of acceptance of the cash value of such training and the setting up of the National Association of Diver Medical Technicians at a later date.

SPUMS SCIENTIFIC MEETING

1983

Professor Brian Hills, the guest speaker for the 1983 Scientific meeting, has kindly provided this paper, the manuscript of a chapter in the now abandoned Volume II of his book "Decompression Sickness", which covers most of the topics he discussed in Fiji. "Decompression Sickness Volume I" was published by John Wiley and Sons in 1977 and is an excellent discussion of decompression sickness, the theories of its causation and its treatment. Professor Hills now works at the Department of Anesthesiology, The University of Texas Health Science Center at Houston.

Professor Hills also presented a paper on Surfactant, which is his current major interest, in Fiji. It is hoped to publish this in a future issue.

DECOMPRESSION PHYSIOLOGY

Brian Hills

Physiological changes during decompression can be divided into those associated with bubble formation and those directly attributable to the changes in alveolar partial pressures of the various gases which decompression must entail. Reversal of nitrogen narcosis for air diving or oxygen toxicity are described in standard texts of those diseases, while the manner in which these can influence the formulation of decompression is outlined in Decompression Sickness Volume I[1] (Chapter 8). This paper is directed towards the physical forms and locations for gas separated from solution by decompression and the physiological modes by which each form can then insult the body.

THE GENERAL ISSUES

Perhaps the most difficult task in pursuing the pathophysiology of decompression sickness in the literature is that of identifying the established facts and separating them from the numerous controversies and assumptions. Before entering into specific issues such as whether diffusion or blood perfusion limits the rate of uptake of inert gases, there are more general questions to be addressed. These include:

1. Is there just one mode of insult or many?
2. If more than one, do the mechanisms follow sequentially or proceed independently?
3. Are bubbles really the underlying cause or just a red herring?
4. What are the mechanisms for the various categories?
5. If current decompression tables and other means of preventing decompression sickness do not achieve their avowed intention of avoiding bubble formation, then what do they do? Do they achieve their goal, but only in the tissue(s) which can provoke symptoms?
6. What is really occurring at the tissue level during treatment?

One insult mode or many?

The wide diversity of symptoms resulting from inadequate decompression might be construed as indicating that there is a single mode of insult which occurs at such a basic level of physiological function that it can become manifest clinically in a most diverse manner. This is, perhaps, the sentiment underlying the statements often overheard, more often in Aviation Medicine, in that a subject starting with a limb "bend" can then "develop" into a neurologic case. It is indeed very common for neurologic symptoms to be preceded by limb pain but this does not necessarily mean that each reflects a different stage in the same underlying mechanism.

Considering the wide diversity of the list, the symptoms can be slotted particularly neatly into six categories consistent with a different physiological mediation of each insult (Fig. l). The best example is the Menière group of symptoms in Category IV whose occurrence as an end-organ injury immediately implies dysfunction of the vestibular apparatus. The classification adopted in Fig. 1 is an extension of the Medical Research Council (MRC) system of dividing cases into essentially local manifestations (Type I) and those with obvious neurologic involvement (Type II) in which, as Griffiths[2] aptly states, the subject really "feels and appears to be ill". With such relatively well defined categories, it would be short-sighted just to look for one mechanism for decompression sickness. Rather, there could be as many as six mechanisms or, at least, for there to be that many combinations of insults and target organs.

Sequential or simultaneous?

The concept that each mechanism can be triggered and proceed independently of the others is consistent with some ability to select the presenting symptom by changing conditions. Examples include:

1. A short deep "bounce" dive upon air is more likely to produce a CNS "hit" than a longer shallower dive for the same overall incidence of decompression sickness.
2. It is well known in commercial diving that a switch from helium to nitrogen as the inert gas breathed, such as often occurs in transferring a diver from a diving bell to a deck decompression chamber, can often precipitate vestibular (Category IV) symptoms (See Decompression Sickness Volume I)[1]
3. Spinal symptoms (III) occur more often than cerebral (II) in divers, about 3:1 as Hallenbeck, et al.[3] point out, and yet the reverse is true for aerial decompression sickness.
4. The percentage of spinal symptoms is much lower in heliox diving than air diving.[4]
5. In experimental animals undergoing the same decompression from the same exposure, a limb "bend" or neurologic "hit" can be selected as the presenting symptoms by the extent of the upward excursion interposed between the exposure and the same decompression,[5] the model adopted by Hallenbeck et al.[3] to ensure spinal injury in their dogs.

Many more examples can be cited of means by which the symptom category can be influenced by the conditions

CATEGORIES OF DECOMPRESSION SICKNESS

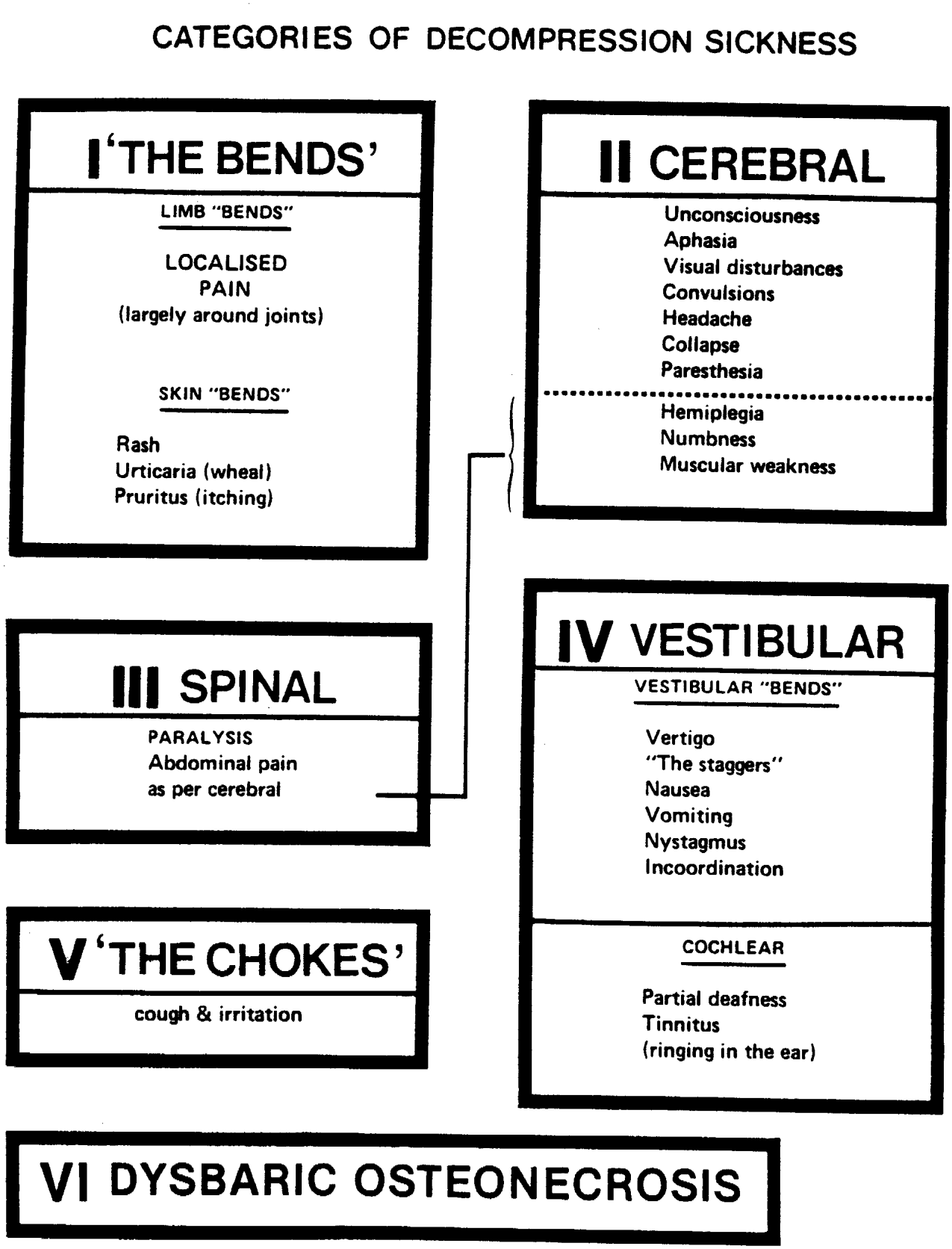

FIGURE 1

The numerous symptoms of decompression sickness arranged in categories of similar physiological mediation of the underlying insult and, hence, indicating as many mechanisms as categories.

prevailing during decompression. Thus it would appear that several insult processes are triggered by decompression and probably develop simultaneously and independently. The presenting category, if any, would then be determined by the relative kinetics of the various mechanisms and how prevailing conditions might tend to emphasize one over the others.

While emphasizing the point that different insult mechanisms *can* proceed simultaneously and independently, not all may do so. A particular example would occur where the production of large numbers of venous bubbles by decompression would produce the "chokes" when they were filtered by the lung. There is little doubt that these Category V symptoms are caused by massive pulmonary gas embolism.[6] In such quantity, the gas is more likely to overload the filtration capability of the lung[7,8] permitting arterial bubbles to embolise vital organs and produce the cerebral (Category II) symptoms or other neurologic forms of decompression sickness which commonly follow onset of "the chokes", a category which, understandably, seldom occurs alone.[9]

The above examples belabour the simple point that we should not be looking for just one mechanism by which to explain decompression sickness or to design measures for its prevention.

Are bubbles a red herring?

If there are different mechanisms for eliciting different symptoms, it could be further asked whether all of these need to be initiated by bubble formation, or separation of gas from solution in whatever other shape it may prefer to assume.

There has been the occasional implication that bubble formation may not be a necessary step in the aetiology of decompression sickness. One of the first was based upon the agglutination of red cells demonstrated in decompressed animals by End,[10] also noted by Wells et al.[11] who have emphasized increased blood viscosity as a possible source of tissue ischaemia leading to pain. However, Walder[12] makes the very pertinent point that blood "sludging" occurs in many other clinical situations without provoking bends-like symptoms.

Another no-bubble hypothesis was proposed for dysbaric osteonecrosis only[13] on the basis that bones are good osmometers for dissolved nitrogen; while the mineralisation process is particularly sensitive to the fluid shifts which might therefore be induced by gases. This approach had the primary advantage that it could explain the particularly long induction time of the first radiographic evidence of a bone lesion.[14] However, if gas-induced osmosis were the true mechanism, then one would expect aseptic osteonecrosis to be induced without decompression by the more potent osmotic gases such as nitrous oxide, but there is no record of any correlation between bone lesions and gaseous anaesthesia.

The primary event

The major evidence in favour of gas separation as the *primary event* in decompression sickness is that no other process has so far been conceived which could be so dependent upon the particular combination of two dominant features for its incidence and intensity, the syndrome being more likely to occur with

1. a greater decompression, and

2. a greater *inert* gas content of tissue prior to that decompression.

Other primary events could be conceived which are dependant upon one or other of the above factors, eg. gas-induced osmosis upon inert gas concentration, but none of these are so uniquely dependently upon the *combination* as the separation of the gas phase from solution, the vital first step to bubble formation.

In finding evidence to support the two principal features listed above, it is tempting to study very deep dives or the latest record for time or depth. From a decompression standpoint, however, this can be misleading since there are so many factors influencing the outcome of a long decompression that the basic trends can easily be obscured. For elucidating basic relationships, it is easier to consider a simple 'bounce" dive where the subject is returned directly to the surface, ie. with no-stop decompression as depicted in Fig. 2.

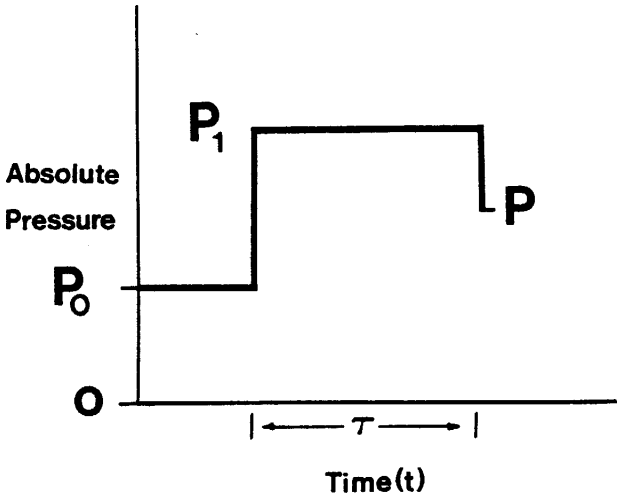

FIGURE 2

A simple exposure followed by no-stop decompression

Decompression per se

The evidence for decompression *per se*, ie. that greater decompression potentiates decompression sickness, is overwhelming with data from diving, tunnel work and aviation. This also applies to each category of decompression sickness with the possible exception of Category VI, dysbaric osteonecrosis. This possible exclusion is based upon the argument that for every decompression, there is also a compression and we should

therefore be sure to eliminate compression per se as an aetiological factor. This is easily done for categories I to V, since each can be induced at altitude, ie. after decompression only. However, bone lesions are not observed in aviators,[15] not even in those seriously incapacitated by other categories of decompression sickness. Thus the absence of Category VI from altitude DCS could be related to the fact that decompression precedes compression in this mode, but is more likely a consequence of the lower absolute pressures involved.

Inert gas content

Any factor which increases the *inert* gas content of the body prior to decompression increases the incidence of decompression sickness. Referring to the simple exposure depicted in Fig. 2, these include:

1. Deeper exposure (P_1 in Fig. 2).

2. Longer time at depth.

3. *Substitution* of a more soluble for a less soluble inert gas at a given depth, eg. He fi H_2.[16]

4. Substitution of inert gas for oxygen at a fixed depth.[17]

5. Increasing transport of inert gas to the tissue by selectively raising temperature,[18] selectively exercising that limb at pressure[19] and effecting other less obvious physiological changes designed to increase perfusion of the critical tissue by blood.

Recompression

Further to the above evidence, it can also be argued that gas separation must be the primary event in mechanisms where the phase gas also constitutes the critical insult, whether it needs to reach its critical state for clinical awareness by growing in volume, coalescing or undergoing any other transformation. Since the vast majority of symptoms are resolved or ameliorated with recompression, it is very difficult to argue that the insulting entity is not compressible and, hence, not in the gas phase. Thus gas separation is again overwhelmingly indicated as the primary event. Moreover this applies to all categories of symptoms since all are relieved by recompression, albeit with different success rates. The only possible exception is again dysbaric osteonecrosis in which there is no way of knowing whether recompression ever treated a potential bone lesion.

To summarise the above discussion, there is very little doubt that bubble formation is the primary event in all categories of decompression sickness with a remote possibility that dysbaric osteonecrosis may be an exception.

Primary event to critical insult

The reader might well ask why it is necessary to differentiate between the primary event as the initiating process and the mode of insult precipitating each category of symptom. In the past it has been argued by the mathematically inclined designers of diving tables that if you can avoid gas formation in the first place, ie. the primary event, then no harm can come to the diver anyway. However, the scientific evidence reviewed in Decompression Sickness, Volume I,[1] indicates that bubbles are formed when following many of the safest schedules and that what determines the occurrence of symptoms is not their presence but how far each mode of insult has progressed along its course towards its critical threshold for clinical awareness or injury. Since the extent of these progressions can be influenced by the overall decompression procedure, it is therefore most desirable to know the individual mechanism for each symptom category; while it is these progressions, rather than the primary event, which the clinician seeks to reverse by the treatment he prescribes.

To return to specifics, we therefore need to address the question that, if gas phase formation is the *primary event*, what are the modes by which it can then insult the body?

MODES OF INSULT

Gas can separate from solution as bubbles in blood, as gas in extracellular sites of various tissues or in the natural potential cavities of the body such as the peritoneal cavity or the joint capsule.

Extravascular gas can form within cells, in the interstitium or within the lymphatic system; but it remains in essentially the site where it was formed. Intravascular bubbles, by comparison, have the capability of mobility and therefore have many opportunities for occluding vessels and producing either general hypoxia or local ischaemia, depending upon their location. Potential occlusion sites include the bifurcating vascular beds of the systemic arterial and pulmonary arterial systems. Venous occlusion has also been postulated.

There are three basic questions to apply to any site of separated gas in determining whether it could produce clinical symptoms of decompression sickness. These are:

1. Could the bubbles occlude a vessel to cause tissue ischaemia or oedema and do other diseases producing other emboli, or otherwise obstructing the same vessels, produce the same symptoms?

2. Is there any opportunity for degradation of fluids in contact with the gas and are the products, eg. aggregates or humoral factors, likely to persist after the bubble has dissolved?

3. Is the bubble in a location where it can press upon a nerve ending to provoke pain, upon an axon to disrupt impulse transmission or upon a vessel to occlude flow and, if so, does the compliance and overall morphology of the tissue prevent the gas from expanding or otherwise dissipating the local pressure which it might otherwise generate to induce these dysfunctions?

These questions can be applied to each of the bubble locations outlined earlier to produce a list of at least eight cases warranting closer scrutiny as possible mechanisms for the six categories of symptoms. There are:

1. Bubbles formed in the natural body cavities, eg. the joint capsule, causing pain.

2. The products of blood-bubble interaction occluding vessels or otherwise disrupting function.

3. Occlusion of lymph vessels.

4. A "venous" bubble occluding the pulmonary arterial system.

5. Occlusion of the venous system for a particular tissue.

6. A bubble in the arterial system directly occluding flow and so causing tissue ischaemia.

7. Bubbles pressing against a nerve ending to produce pain.

8. The same autochthonous bubble pressing against an axon to interfere with transmission.

9. An extravascular bubble pressing against a vessel to compromise either perfusion of the tissue or blood supply to a nerve, again compromising transmission.

Pathology

Before pursuing each of the above as a possible mechanism for each of the six categories of symptoms (Fig. 1), the next logical step might seem to be one of turning to the pathological evidence to try to reduce the number of likely combinations. Three of the most comprehensive pathological studies are those of Boycott et al.[20,21] Haymaker[22] and Rozsahegyi.[23] These and others are discussed in some detail in Decompression Sickness, Volume I[1] but, basically, pathological evidence from autopsy reports or studies of sacrificed animals can be found to support all nine of the above mechanisms and possibly more. The problem lies in knowing what is cause and what is effect. This is aptly described by one of the most eminent decompression pathologists[22] who wrote "from this vast mass of material, nothing really pertinent to establishing a model or mechanism can be extracted." This may be an understatement since, to take one example, the *absence* of bubbles in skeletal muscle, liver and heart[20] is good reason to look at other tissues for the source of the problem. However, it does mean that we must rely primarily upon physiological reasoning to try to reduce the fifty-four possible combinations of mechanism and symptom category. Let us therefore consider each of the mechanisms in the above list individually.

Gas in the body cavities

The natural body cavities are only so termed because it is easy to separate the surrounding tissues in forming them. Thus they do not resist the formation of this extravascular gas and are easily pushed aside without creating any significant pressure with which the enclosed gas can disrupt function. In fact, in a pneumothorax, the gas in the pleural cavity is at a negative pressure relative to atmospheric. The above argument also applies to the joint capsule where gas can easily escape from between the articular surfaces without deforming a nerve ending in the adjacent tissue as a rigid solid, such as sodium ureate crystals, can do in the case of gout. In fact, large volumes of gas can be injected into the synovial cavity without producing symptoms; while the same applies to gas formed by decompression and confirmed radiographically,[9] a condition more appropriately termed "aeroarthrosis".

Blood-bubble interaction

Potential infarcting agents which are incompressible and known to be associated with decompression include fat emboli[24] and microthrombi.[25] The whole subject of blood-bubble interaction became one of great interest a decade or so ago when some thought it held many of the answers to decompression problems. This may still be true for chronic cases but there is one inescapable fact which limits its relevance. This is the relief obtained by recompression which is effective in at least 99% of limb bends and 90% of neurologic cases. It is very difficult to envisage any mechanism whereby the application of hydrostatic pressure can restore blood flow to a vessel infarcted with an incompressible embolus.

When extensive coagulation or other degradation of blood is seen in pathological studies, eg. in vertebral venous lakes[3,22] it must be asked whether the observations are cause or effect.

Although the products of blood-bubble interaction may not be the primary agents in the aetiology of decompression sickness, they may have important secondary roles by way of the humoral factors released during those interactions. One example is the release of serotonin[26] which can sensitise nerve endings to other pain-provoking stimuli such as adjacent bubbles.

Although the products of blood-bubble interaction are likely to play no more than secondary roles in most categories of decompression sickness, the compressibility argument does not detract from their providing the primary mechanism in dysbaric osteonecrosis, as proposed by Jones et al.[27] This category (VI) is chronic only, since there is no way of telling whether recompression ever prevented a potential bone lesion.

Lymphatic bubbles

Bubbles have been found in the lymphatics of most organs in decompressed animals.[1,28,29] It is easy to envisage their occluding the lymph vessels to produce the oedema occasionally seen in decompression sickness and the "orange peel" appearance of the skin distal to the occlusion.

Venous bubbles

Venous bubbles are often produced in large number during decompression and usually remain asymptomatic. In decompressed animals they are seen mostly in the veins draining the fatty tissues in which nitrogen is six-fold more soluble than in water. The question concerning whether the bubbles form *de novo* within the vessels of those tissues or enter them pre-formed by rupture of the endothelial wall is an issue of largely academic interest, but the fact that, in either case, they are primarily derived from the large amount of nitrogen dissolving in adipose tissue must be borne in mind when interpreting the signals from precordial Doppler monitors.

In sacrificed animals it is tempting to speculate that the bubbles observed in the veins, or the products of blood-bubble degradation often seen adjacent to them, are responsible for any stasis observed. However, it must be asked whether these are effect rather than cause, since bubbles would remain in veins without flow whatever the cause of stasis, ie. they will reach systemic veins anyway but cannot be washed away without flow. Moreover, it is difficult to conceive bubbles occluding a flow system continuously converging into vessels of ever-increasing diameter. One exception to this confluence of blood flow is the vertebral venous lakes often implicated as the cause of spinal symptoms (Category III).[3] However it is difficult to envisage all of the many outlets to these lakes being occluded simultaneously, especially when they are not constricted by the valves common to veins elsewhere in the body.

Venous bubble detection in humans is particularly easy utilising ultrasonic probes which exploit the Doppler principle,[30,31] since it is non-invasive and the simple audio output can be interpreted with little training. When used in the precordial position, ie. looking down the pulmonary artery, there is the particular advantage that one is then scanning total venous return and, moreover, is doing so at the highest velocity of venous blood at which bubbles are more easily detected.[32] There have been many attempts to correlate Doppler signals with overt symptoms of decompression sickness with varying degrees of success. It is probably fair to summarise that data by concluding that the correlation is good for simple no-stop decompressions but very poor following a long, complex stage-decompression. This could reflect the fact that venous bubbles are derived largely from fatty depôts whereas an aqueous tissue is more likely to be responsible for the common forms of decompression sickness, ie. limb bends, with one becoming a poorer analogue for the other as the dive proceeds.

Pulmonary arterial bubbles

In tunnel workers, Nashimoto and Gotoh[33] have found a good correlation between "the chokes" and large numbers of venous bubbles as indicated by pre-cordial Doppler monitoring. This is consistent with the widely held view that these Category V symptoms are the result of extensive pulmonary air embolism.[6] This aetiology has been challenged by Ferris and Engel[34] on the basis that air accidentally introduced into the venous system does not elicit the same response, but such gas is more likely to resemble a bolus than the many microbubbles produced by decompression. It can then be argued that many smaller emboli could stimulate J-receptors in the respiratory exchange region of the lung more than a bolus by producing more local oedema or releasing a humoral factor as an intermediate step. Microbubbles can penetrate further into the pulmonary vascular bed by virtue of their size and, presumably, closer to the J-receptors. A good description of these receptors and the reflexes which can be invoked to elicit a laryngospasm are given by Paintal.[35]

Systemic arterial bubbles

There have been many pathological studies of arterial air embolism and a few in which the vessels have been observed following a bolus injection,[36] but the real question concerns bubbles of the sizes produced during decompression.

These have been measured in the venous blood of live decompressed dogs as ranging from 29-700 μm in diameter[37] with a median size in the region of 60 μm. When individual bubbles have been observed[38] in the middle cerebral artery of a guinea pig through a cranial window, they tend to reduce flow. They can be seen to proceed through bifurcations into arteries of smaller diameter until they reach those of comparable diameter. There is then a fairly sudden dilation of the arterial system distal to the bubble, diameters sometimes increasing two-fold. The bubble then proceeds until it is again of comparable diameter to the vessel when it now deforms and proceeds much more slowly. It may pass one more bifurcation, never splitting up, and then lodges at the next. The leading edge is rounded while the trailing edge is flatter and pulsatile. The bubble may remain for several minutes and then proceed to the next bifurcation where it will stop. When gas forms such a column following a bolus injection, it proceeds similarly, but gives the impression of not penetrating the vascular bed as deeply. This may be due to the absence of the trailing edge as a forward-propelling surface force. In other words, the trailing edge is now removed to such a large vessel ($r \neq\neq$) that the forward surface thrust (DP) as estimated by the Laplace equation ($DP = 2g/r$) is now very small. g is the surface tension.

When more microbubbles enter the cerebral arteries before the first has lodged, the first gives the appearance of slowing more than usual and letting the others catch up. When adjacent to each other, they then coalesce to form "slugs" of gas with a length about 1.5 times the diameter of the vessel which they slightly distend. These slugs then close upon each other until a thin liquid film separates them. If the bubbles are oxygen or the animal is ventilated upon oxygen during this phase of embolisation, the process can be reversed and fluid starts to enlarge the separating films as the slugs decrease in size until they finally move on. Otherwise, if left, the slugs will suddenly 'pop' together as though a shock wave had passed down the vessel. The time for this to occur is very variable but is of the order of 20 minutes from the initial slug formation. About this time, the venous blood can be seen to be noticeably more blue. Columns of gas do not reverse themselves and it requires a drastic procedure such as recompression to do so.

In terms of death or survival, the brain is slightly more tolerant to gas as microbubbles than as a bolus,[38] and can certainly tolerate more gas when administered at slower rates.[7] It still does not give us much of a handle on the awesome question of the rate at which the brain can disperse microbubbles asymptomatically or whether they have any chronic effects, ie. are there really "bubble heads" as some offshore communities somewhat callously refer to the divers.

The only hard figures readily available refer to bolus injection of gas into the arterial system of dogs. 0.5 ml/Kg of air injected into a dog's pulmonary veins cause death[39,40] or 0.25ml/Kg in the common carotid artery.[41] On the other

hand, 0.025 ml injected into the coronary circulation can cause myocardial ischaemia while 0.05 ml/Kg causes death.[42]

There is little doubt from autopsy findings on patients with air embolism or baboons with experimental air embolism[43,44,45] that the insult is ischaemic. Where there has been known arterial embolisation, eg. from pulmonary barotrauma following submarine escape,[46] the symptoms are the same as those listed for cerebral decompression sickness, see Fig. 1. Hence there is little doubt that arterial bubbles are responsible for Category II decompression sickness. Whether they are also responsible for other categories is quite another matter which is discussed later.

Role of the lung

The low tolerance of the body to arterial gas indicated by the above figures is in sharp contrast to the large tolerance of the venous system to air where dogs have survived after infusion of a litre.[47] This implies that the lung is a very efficient filter for the gas phase in all forms and direct experimental work upon dogs using both boluses and calibrated microbubbles has confirmed this.[8] If the lungs were not such a superb bubble trap, diving would probably be impossible.

This raises several unique situations, the first concerning anyone with a patent foramen ovals where accidental venous embolisation has been known to cause the patient to become comatose immediately. A somewhat similar situation would occur in the woman diving during pregnancy when the foetal brain would not be protected from venous bubbles in the manner afforded by the filtering capability of the lungs in adults.

For the purpose of designing preventive methods for decompression sickness, it therefore becomes very important to determine what factors are likely to compromise this superb capability of the lung to filter or otherwise trap bubbles in venous blood. The first reaction is to look for any agents which might cause vasodilation, remembering that many drugs have the opposite effect upon the pulmonary vasculature to that observed in peripheral tissues. Thus it is most interesting to find that, when administered in clinical doses, aminophylline allows gas to escape into the arterial system.[8] This implies a possible warning to the use of such drugs as a bronchodilator for a case of "chokes", known pulmonary gas embolism. The situation is complicated, however, by the observation that aminophylline does not compromise the filtering capability of the lung if administered post-embolisation, ie. it does not seem to release those bubbles already trapped but just those continuing to enter the pulmonary artery.

Another factor which allows gas to escape entrapment is overloading the lungs with air[7,8] when there is a delay of 10-30 minutes in the appearance of systemic arterial bubbles, a delay which does not seem dependent upon the size of the infused bubbles. In fact, contrary to expectations, size of the venous bubble does not seem to be the primary factor determining whether or not it will be trapped. From this observation, the time delay and the rough indication from Doppler pulse heights, there is the impression that the size of arterial bubbles escaping entrapment bear little relationship, if any, to the size of bubble entering the lung. It is almost as though gas coalesced in the pulmonary vasculature, as observed and described above for the cerebral circulation, and was then later re-injected into the arterial system if the insult to the lung were enough. The overload mechanism can be attributed to occlusion of a vessel depriving the wall of blood-borne nutrients or the ability to lose released humoral factors which would then cause vascular smooth muscle to relax, either directly or indirectly. Indeed air embolism is used in many animal preparations as a means of inducing permeability oedema of the lung. There could also be a sympathetic or, possibly, parasympathetic response to embolisation.

Another insult which can compromise the capability of the lung to filter bubbles is pulmonary oxygen toxicity.[48] The effect is quite variable and the critical insult has not been characterised in terms of a number of UPTDs[49] or a threshold value for the COTi.[50] However, it poses an aggravating complication to the treatment of decompression sickness in divers who have already received a large exposure to oxygen before symptoms appeared. It is no good to treat a limb bend with even more oxygen if any resulting toxicity is simply going to allow venous bubbles to reach the arterial system with the risk of Category II symptoms.

There may be many other factors which can effect pulmonary vascular tone and, hence, the capability of the lungs to trap bubbles, but this field of investigation is just starting to attract attention. There may also be factors tending to release bubbles already trapped and one is recompression.

Bubbles in peripheral arteries

In an earlier section we discussed how bubbles in blood flowing to the brain afforded a very good explanation for cerebral (Category II) symptoms. The next question is whether bubbles in the arteries leading to other organs could explain other categories, eg. limb "bends" (Category I) or whether such symptoms are central anyway.

To address the last question first, there is good reason to believe that limb bends are derived from an essentially *local* insult since:

1. The pain can be ameliorated by local anaesthetics, eg. novalgin.[51]

2. The application of local pressure can usually reverse a mild limb bend, eg. by applying a sphygmomanometer cuff to the site of pain[52,53] or immersing the joint in mercury.

These key points and others leave little doubt that Category 1 symptoms stem from an essentially *local* insult but this leads to the next question, whether the symptoms of limb bends could be caused by arterial bubbles. Since the era of Paul Bert,[55] it has been generally assumed that bubbles occlude arteries to cause ischaemic pain. This mechanism, however, has encountered increasing criticism for the following reasons:

1. The argument that if recompression therapy is to relieve limb bends, it must dislodge the occluding bubbles and probably flush them out into the venous system as

observed in other organs.[36,38] However, if the subject is returned to the symptom-provoking pressure within a few minutes, the "bends" return to exactly the same sites with virtually the same intensity.[56] It is far too much of a coincidence to suppose that another bubble, or set of them, would lodge in precisely the same site a second time. It is much better explained by extra-vascular gas which cannot move but only change volume in the same site.

2. The pain of ischaemia is unlike bends pain. Other diseases which produce arterial emboli do not produce bends-like pain.[12]

3. Using tail-biting in kangaroo rats as a model to simulate limb bends in men, hypoxia following decompression was found to protect against "bends"[57] rather than potentiate them, which would be expected if the offending tissue were already deficient in oxygen.

4. In goats exposed to a marginally safe partial pressure of nitrogen, an appreciable increase in the oxygen partial pressure of the exposure caused extensive symptoms upon decompression.[58] It is hard to explain how an increase in PO_2 could exacerbate the pain if oxygen deficiency were causing it. However, while there were some Category I symptoms, most were spinal (Category III).

5. The fact that venous bubbles are a much more common occurrence than arterial in all but exceptionally fast decompressions[1] and that such bubbles would normally be trapped by the lungs, as described above, before they could become arterial emboli.

Further evidence for the incompatibility of the clinical symptom with a mechanism based upon bubbles in peripheral arteries have been discussed by Ferris and Engel.[34]

Arterial bubbles in other organs

It is easy to conceive bubbles occluding any arterial system provided they can reach those arteries in the first place. The more likely systems would be those with an end-artery type of circulation as occurs in the inner ear and the eye. This would appear to offer a very convenient explanation for Category IV symptoms until one asks the now-familiar question of why such symptoms do not occur much more frequently in cases of known embolic disease such as subacute bacterial endocarditis.

By far the most serious aspect of this question concerns the spinal cord, since Category III symptoms are currently the major cause of disablement in divers. Spinal cord decompression sickness, however, is restricted almost entirely to air diving, particularly in the range of 100-150 feet.[4] The popular explanation has been arterial bubbles as proposed for decompression sickness in general by Paul Bert[55] with specific reference to the spinal cord by Haldane and co-workers despite their clear demonstration of many extravascular bubbles in the white matter.[20]

Despite its continued popularity, arterial embolism would now seem an unlikely cause of spinal DCS for the following reasons:

1. Upon recompression most cases are relieved, indicating that any arterial bubble must have been dislodged but, upon return to the bends pressure, the symptoms return exactly as they were before. Thus the same argument invoked for limb bends can be used in that it would be a fantastic coincidence for another set of bubbles to lodge in precisely the same sites and cause the same symptoms with the same distribution of dysfunction as plotted on a neurologic atlas of the body. This is the same argument used earlier to discount other forms of embolism, eg. that of vertebral venous lakes.

2. It has been argued that the brain constitutes 98% of the spinal cord[59] and receives 78-85 times more blood flow than the spinal cord[60] and should therefore receive proportionately more arterial emboli. However the ratio is about 3:1 spinal:cerebral in divers, but not in aviators. Such reasoning has led Hallenbeck et al.[3] to discount arterial embolism, pointing out that, in other disorders producing systemic embolisation, the brain is the target organ with only 0.4% of cases involving the spinal cord.[61]

3. The almost total absence of spinal involvement in heliox diving[4] makes it very difficult to explain why systemic nitrogen bubbles would occlude the cord and yet helium bubbles would not do so for dives when the incidence of other forms of decompression sickness was comparable.

The above points would seem to make arterial embolisation just as unlikely as other embolic mechanisms for Category III symptoms. Care should also be exercised in reading standard neurological texts on embolic diseases not to invoke circular reasoning. The spinal cord is sometimes listed as a target organ for circulating arterial emboli but, often, this arises simply because the author has read a diving paper or two expressing the conventional (arterial) theory of spinal cord decompression sickness.

THE AUTOCHTHONOUS BUBBLE AND ENCASED GAS

The concept of the autochthonous bubble, forming de novo in the tissues, was probably first invoked by Haldane and co-workers to describe a bubble pressing onto a nerve or nerve ending, although the same authors still attributed decompression sickness to arterial bubbles. Theoretically, the extravascular gas bubble has the great advantage that it can explain the finding that symptoms, especially Categories I and III, can be relieved by recompression yet return in the same site upon return of the patient to the original symptom-provoking pressure. Thus it is most important to pursue all the ramifications of the remaining three possible insults, viz. those involving extravascular gas pressing upon a nerve ending, upon a nerve axon or upon a vessel.

Extravascular bubble pressing upon a nerve ending

The question of whether an extravascular bubble is going to elicit pain depends upon two factors: whether there are nerve endings which can give rise to "bends" pain and whether the gas in the bubble can generate the pressure needed to bend or otherwise distort that nerve ending to its pain threshold without being dissipated.

Most of these questions are answered by a very simple yet most fundamental experiment conducted by Inman and Saunders.[62] They inserted fine hypodermic needles into various tissues of Air Force cadets and found that, when they injected Ringers solution into many of the tight connective tissues, they could induce a pain virtually indistinguishable from "bends". This was particularly apparent for tendon. They found that the effect was reversible and that the pain threshold was determined by the pressure with which the Ringers solution was injected, the critical differential remaining the same for the same subject but varying between individuals within the range of 11-26 mmHg.

If a bubble can exert the same local pressure, then this offers a particularly attractive hypothesis for limb bends, since it can explain the titration of pain with decompression and its almost instantaneous reversibility with recompression. Many connective tissues are well innervated, particularly tendon in which other insults to the nerve endings produce a pain the description of which by Stilwell[63] virtually paraphrases the description of limb 'bends" given by diving physicians.

The next question concerns whether the gas separating from solution in a tendon would dissipate before it could reach a pressure of 11-26 mmHg in excess of tissue pressure. Obviously a bubble formed in a very compliant tissue would simply push the tissue aside rather than allow its formation to generate any excess pressure. It would therefore seem particularly meaningful that Inman and Saunders found their pain-pressure threshold in the "*tight"* connective tissues. Moreover, simple calculations based upon the compliance of these tissues and the volume of gas which could form in a diver with a minimum bends depth of 33 fsw on air provides quantitative confirmation of the 15 mmHg pain threshold.[64]

There are other factors in favour of extravascular bubbles in tendon as the cause of Category I symptoms, these including the effects of exercise and the location of bends pain as arising *around* the joints but not within the joint capsule itself. Regarding exercise, Behnke[19] quotes 25 minutes as the safe working time at 100 feet to be followed by no-stop decompression by comparison with 35 minutes for the same subject at rest. These values are much too close to reflect the 20 to 40-fold change in blood flow in muscle, but could well reflect the change in tendon. Thus an extravascular bubble pressing upon a nerve ending in a tight connective tissue offers a simple mechanism for Category I decompression sickness for which there would seem to be no adverse evidence. Bubbles are certainly seen in these tissues upon decompression, either directly in living animals[65] or as revealed by X-rays in humans.[1]

Extravascular bubbles pressing upon an axon

The spinal cord is another organ in which many extravascular bubbles can be seen following decompression from an air dive, particularly in the white matter and in those sections where the white/grey ratio is highest.[20] This probably reflects the much higher solubility of nitrogen in lipid, although there is some question whether the solubility in white matter is the same as in depôt fat. It can also explain why Category III symptoms are so much more common diving on air than on heliox and why neurological examinations tend to reveal a preponderance of lesions at T4 and L1. The preponderance of motor dysfunctions is also consistent with the greater myelination of nerves in the motor tract of the spinal columns, again reflecting the greater volume of gas separated from solution in those areas with a higher lipid content.

While all of these correlations may strongly implicate the numerous extravascular bubbles seen in the cord, and especially within myelin where Haymaker[22] remarks upon the propensity of bubbles as "fenestration", there is still the question of whether so much gas can actually press upon the axon with enough force to interfere with impulse transmission. This requires a close look at the complex anatomy of the spinal cord from which it can be seen that there are various mechanical barriers to gas expansion all acting in mutual support of each other, rather like an onion with many skins.

In order to determine whether transmission could be impaired by a bubble formed in the myelin adjacent to the axon or by gas formed outside the myelin sheath, we need to estimate the local distorting pressure and, hence, address the following questions:

1. Taking the outer shell first, we must ask by how much CSF pressure can rise during decompression.

2. Can extracellular gas dissipate and so reduce its local pressure by tracking along the cord between nerve fibres?

3. By how much can the pia and other membranes expand to accommodate the volume increase?

4. Can gas formed within the myelin sheath track along the axon to dissipate its local pressure and how compliant is the myelin sheath in resisting its expansion?

Mechanics of the spinal cord

Cerebro spinal fluid (CSF) pressure is normally about 11 mmHg[66] but can be raised by various physiological stimuli such as elevated PCO_2. In both men[67,68] and goats[6] with a lumbar spinal tap the volume of CSF was found to increase with decompression. If this fluid had not been allowed to expand then, presumably, it would have elevated the CSF pressure. Although lumbar puncture has produced remarkable relief from decompression sickness in some cases,[69] these were cerebral rather than spinal. Moreover the elevations measured in CSF pressure were inadequate to cut off blood flow to the cord. Thus elevation of CSF pressure can be regarded as a contribution to spinal cord pressures rather than a potential hazard in itself.

To take the second of the above questions, it was found that the ability of gas to track along the cord between nerve fibres was very variable but, on some occasions, back-pressures well in excess of 50 mmHg were found upon air injection into an open-ended dog cord.[70] When the cord was tied and fluid injected to eliminate capillarity effects from simple compliance of the adhering cord *in situ,* the back-pressure was related to injected volume as shown in Fig. 3. This is particularly interesting since the back-

pressure remains effectively zero until cord volume is increased about 11-19% and then rises very steeply, presumably when the convolutions of the pia are taken up and/or the non-compliant membranes of the arachnoid and dura also start to resist expansion. After an 11-19% cord expansion the "encased" gas now raises tissue pressure.

The gas *within* the myelin sheath has also been studied recently[70] by decompressing excised spinal cords and then raising and lowering its pressure when the gas itself must obey Boyle's Law. Any deviation offers a very simple means of estimating the pressure differential of the gas adjacent to the axon. Some of these autochthonous bubbles have estimated pressures up to 50 mmHg which would seem adequate to explain the occasional unilateral dysfunction of the cord after decompression. However, symptoms are usually bilateral and the pathology is more consistent with ischaemia and a vascular mechanism for Category III symptoms.

Encased gas closing a blood vessel

It is quite conceivable that a single bubble could press upon a blood vessel with sufficient force to close it, but only if the gas had a higher pressure than the perfusing blood *after* the bubble had indented the vessel. However it is difficult to envisage this occurring in most tissues since these are generally so compliant and, in any case, allow ample opportunity for the gas to expand in directions away from the vessel.

The most likely situation for a bubble to compress a blood vessel is where both the vessel and the bubble(s) are contained within a non-compliant structure. The bubble(s) would then not need to be adjacent to the vessel but their formation could act synergistically to cause a cumulative rise in local pressure which could be transmitted to the vessel wall by both the gas and the extravascular fluid acting as a hydraulic medium. This concept has been compared to a waterfall in explaining some aspects of pulmonary blood flow[71] where flow stops if the sill of the weir is raised above the upstream level just as perfusion ceases in the lung when alveolar pressure is raised above pulmonary arterial.

In bone the rigid walls provide the ideal non-compliant "casing" from which the many bubbles formed in the fatty marrow can raise intramedullary pressure and reduce blood flow proportionately. This has been confirmed experimentally.[72] Similar trends have been found upon decompression to simulated altitude.[73] Thus large volumes of extravascular nitrogen deposited in fatty marrow have been implicated as the cause of dysbaric osteonecrosis,[74] a concept compatible with the thin walls of bone blood vessels[75] and the remarkably symmetrical distribution of bone lesions.[23] However the occlusion should occur at the

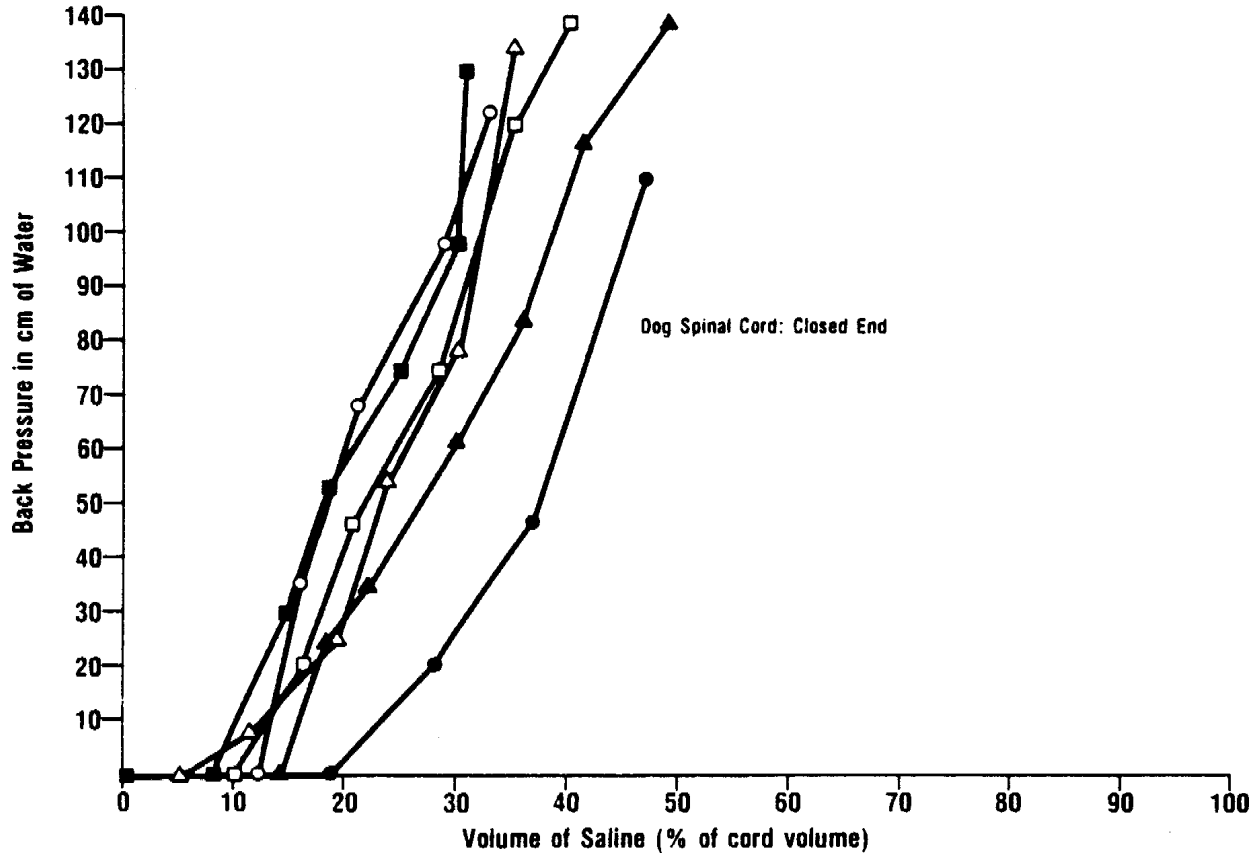

FIGURE 3

The relationship between the volume of ligated spinal cords of dogs and the internal pressure. Note the steep rise in pressure as soon as the convolutions are taken up and further volume increase requires deformation of the tough, non-compliant, encasing membranes such as the dura and arachnoid.

time of decompression and, along with embolic theories for Category VI symptoms, it is difficult to explain the long delay of several months[76] in the appearance of the lesion compared with the appearance of aseptic bone necrosis only weeks post-fracture.

Another situation where vessels could be compressed by gas encased by non-compliant mechanical structures is in the spinal cord. Upon decompression, the pressure within the various membranes, the dura, arachnoid and pia, could be raised by both the formation of extracellular bubbles and the distension of the myelin sheath by gas formed within the myelin. It can be seen from the mechanical studies illustrated in Fig. 3 that the net effect of the membranes is to allow distension of the cord to occur freely until the volume has increased by 7-19%, averaging 12%. After this, the pressure rises steeply and would reach the perfusion pressure of the cord, about 30 mmHg, for a volume increase of about 18%. For a fatty tissue reaching steady state before direct return to the surface, this volume of "encased" gas could be formed by an air dive to 100 feet. This is interesting, since it is just about the shallowest depth at which spinal "hits" start to become common in air diving.[4]

Thus the cumulative effect of extravascular gas in compromising blood flow could offer a simple mechanical explanation for the termination of motor function at a particular point in the cord. The system envisaged is thus a double waterfall where flow would stop when the extracellular fluid pressure exceeded capillary blood pressure. This is most likely to occur in the watershed zones and provide yet another reason for the preponderance of symptoms arising from T4 and L1.

It is impossible to assign a particular mechanism to each category of decompression sickness shown in Fig. 1. However, it is probably fair to say that although arterial bubbles are not the universal insult they were once thought to be, they are fairly certain to cause cerebral symptoms (Category II) while venous bubbles reaching the pulmonary arterial system are almost certain to produce "the chokes" (Category V). The autochthonous bubble would seem to offer the best correlation with the many features of limb "bends" (Category I) while many such bubbles, or "encased" gas, could be responsible for spinal decompression sickness (Category III), although more evidence is needed before arterial bubbles are definitely ruled out for the latter. Vestibular problems (Category IV) and dysbaric osteonecrosis (Category VI) remain open and several of the modes of insult could apply to each, remembering of course, that there could easily be more than one mechanism for each category.

PREVENTION

The prevention of decompression sickness is largely associated with the formulation of diving tables in which basic physiology is often obscured by mathematical complexity. Perhaps the most surprising fact is how little impact that basic research into the physics of bubble formation and the physiology of diving has had upon the decompression formats actually used in naval and commercial diving.

Conventional format

The vast majority of practical diving tables are based upon empirical calculation methods which are only loosely associated with the physiology of the body in so far as they are modifications of the original Haldane rationale.[20] Few of these calculated tables have not undergone further modification by pure trial and error. The Haldane rationale and the many calculation methods derived from it are described in detail in Decompression Sickness Volume I,[1] but it essentially consists of taking air as though it were one gas and then assuming that a tissue will take up that air exponentially. This means that the rate of uptake is proportional to the driving force (blood-tissue tension differential), ie. a linear relationship between the rate of "saturation" and the difference between the ambient air tension and the saturation value as represented by the depth of the dive. An exponential function is a particularly convenient one to adopt since it means, in effect, that this difference, ie. the deviation from "saturation", is continually halved in the same time interval. Thus it takes the same interval to proceed from 0% to 50% as from 50% to 75%, as from 75% to 87.5% "saturation" and so on. This interval for uptake is appropriately termed the "half-time" of the tissue and the same equation with the same half-time is used to calculate elimination of air from that tissue during decompression, such *linear* systems being particularly easy to program on computers.

Having calculated the tissue *(p)* at any instant, the next requirement for computing a diving table is to select some criterion by which to limit the decompression at that particular stage. In the original use of the Haldane method, *p* would be expressed as so many "footsworth of air". It was then argued that no air bubbles would form if the tissue was reduced to an *absolute* pressure (*P*) provided the decompression ratio *(p/P)* was less than 2. Many years later this critical value of the ratio was given the symbol 'M' with values other than 2 as it was redefined with *p* referring to inert gas tension only. Provided this 'M' value for the tissue was not violated, then it was assumed that the air remained in supersaturation solution. The same linear relationship was used to calculate the history of gas in that tissue during decompression as had been used to determine uptake.

This might seem a very simple means of calculating a decompression schedule but, unfortunately, no one equation has ever proved adequate for computing tables of widely differing bottom times. Hence Haldane invoked the concept of multiple tissues, in fact a spectrum of tissues from which he arbitrarily picked five with halftimes of 5, 10, 20, 40 and 75 minutes as representing almost equal geometric steps.

Upon diving deeper than 200 feet this calculation rationale was found inadequate and since then empiricism has run riot, with the numbers of hypothetical tissues reaching several hundred in some computer programmes, each tissue having an empirically determined 'M' value, an empirical half-time or even an 'M' value which is an empirical function of depth.[77]

In all of these approaches there is almost universal acceptance of the axiom that violation of the 'M' value can

cause gas to separate from solution. Conversely it represents a trigger point for bubble formation which, if not violated, implies that no gas has formed in that tissue. Thus most designers of diving tables take great care never to exceed any of their empirical 'M' values in the hope that they are not forming any gas phases and, therefore, they need not consider the mechanisms whereby the bubbles can provoke any of the insults discussed earlier. How nice it would be if decompression were that simple!

Fundamental assumptions

The development of decompression schedules has essentially followed a long series of modifications to the original "Haldane" calculation method necessitated by an unacceptable incidence of decompression sickness, usually found when venturing deeper or for longer than that rationale had previously been used to compute tables. With one notable exception,[78] the changes were not based upon physiological parameters. It is therefore interesting to consider that basic assumptions underly present commercial tables and why many of them seem to give an acceptable bends incidence.

Many questions arise but, on the whole, they can be reduced in number to include the following:

1. How is gas taken up by tissues?

2. If air cannot be regarded as one gas, how should allowance be made for the oxygen partial pressure?

3. Can tissue really retain any supersaturation and does the "trigger point" represent the primary event or the critical insult or what should replace it?

4. How is gas eliminated from tissue and is elimination really the mathematical reverse of uptake?

5. What modifications should be made to the tables to allow for the different categories of symptoms?

Gas uptake

Haldene's original adoption of the exponential function for describing gas transfer in a single tissue was based upon the realisation that this is the mathematical format followed if uptake is limited by blood perfusion. This means that the accumulation of gas in a tissue is limited entirely by the flow of blood to that tissue and not by its subsequent diffusion into extravascular tissue.

This assumption is generally accepted in physiology[79] and has only really been challenged in connection with the incidence of limb bends in divers where the no-stop limits for both air and heliox were found to follow a $\sqrt{t}$ relationship characteristic of diffusion limitation.[78,80] This has led to much argument in the literature, otherwise known as the perfusion-diffusion confusion described in detail in Decompression Sickness Volume I.[1] The controversy is somewhat academic but the conflicting evidence produced can be explained on the basis that blood perfusion is not a continuous process, especially in tendon[81] which has been implicated as the tissue responsible for limb bends and, hence, the tissue having the major influence upon decompression formulation. Thus, when a bundle of 20-140 capillaries in a tendon are closed with little collateral flow, gas transfer must be controlled by diffusion and this would apply particularly to dives of shorter duration and, hence, the $\sqrt{t}$ relationship for bounce dives.[78]

The observation that some tendon capillary bundles may close for long periods, as much as 2 hours or so,[81] queries the basic assumption in all calculations of diving tables that gas uptake and elimination are continuous processes even though the rates may vary depending upon the driving force. Thus one tissue zone may "saturate" in a series of curves with sharp breaks representing periods where the flow was diverted to other capillary bundles.

"Saturation"

Modifications of the Haldane approach have taken account of the fact that air cannot be regarded as one gas and that the inert gas and oxygen must be computed separately. The kinetics of inert gas uptake are such that the tissue nitrogen tension will eventually reach the alveolar nitrogen partial pressure if this is not changed. The question then arises as to what tension the metabolic gases will reach.

When microprobes are placed in tissues to try to measure $P0_2$, values can be obtained anywhere from zero to arterial values but most are at venous levels or below. The analysis of gas placed in the natural body cavities, eg. in the peritoneal cavity shows that it is saturated with water vapour at body temperature, but both oxygen and carbon dioxide soon attain venous values.[82] Let us consider the tissue in a diver who has been living in air at 100 feet for 24 hours. We find that the nitrogen tension has equilibrated with the alveolar N_2 partial pressure (2383 mmHg in this example) and the whole tissue was attained *steady-state*. If we now add up the total gas tensions in the tissue, the total is 2526 mmHg, which is 537 mmHg short of the total of alveolar partial pressures, see Table 1. By Dalton's law this must equal the absolute pressure since the gases are all in the gas phase in the alveolus. Hence, even after reaching *steady-state conditions*, there is a deficit of total gas tension in the tissue due to the metabolic assimilation of

TABLE 1

THE INHERENT UNSATURATION FOR STEADY STATE AT 100 fsw ON AIR

GAS	Alveolar partial pressures (in mmHg)	Tissue gas tensions (in mmHg)
N_2	2383	2383
O_2	593	50
CO_2	40	46
H_2O	47	47
	3063	2526
	↑	↑
	Absolute pressure	Total gas tensions

INHERENT UNSATURATION
= 3063-2526 = 537 mmHg

oxygen and the production of CO_2, a much more soluble gas. This difference or *inherent umsaturation* of tissue has been measured directly in tissue and found to increase with inspired oxygen partial pressure, whether this is effected by substituting oxygen for nitrogen at a fixed pressure or increasing pressure on a given breathing mix.[83]

This means that, whereas the inert gas may equilibrate, a tissue never comes to true saturation with the environment unless it is not metabolising and, therefore, is dead. Thus the diver with the typical gas tensions given in Table 1 who has reached steady-state could decompress by 537 mmHg, ie. from 100 to 77 feet before reaching saturation in the true physico-chemical sense of that word.

Phases of decompression

The next question to ask is by how much the decompression could overshoot the 77 foot mark (2526 mmHg in Table 1) before bubbles will actually form, ie. what is the degree of true supersaturation which the tissues can tolerate before bubbles form? Before addressing this question, however, we should be quite sure of what we mean by supersaturation and how this particular phase fits into the overall decompression since it has long been my contention that supersaturation has been unduly emphasized by popular calculation methods.[64]

Let us consider the diver at 100 feet on air who returns to the surface without stopping and then develops limb bends. His bends-provoking tissue must pass through the following three phases (Fig. 4):

I. Until he reaches 77 feet, this bends-provoking tissue must remain undersaturated by reason of the inherent unsaturation discussed above. If he had not attained steady state at 100 feet before starting his decompression, then he could carry on a bit further and ascend by the additional amount by which his tissue nitrogen had failed to reach the equilibrated value of 2383 mmHg given in Table 1. If the tissue were 80% equilibrated, this would amount to an additional 0.2 (2383-570) = 362 mmHg, or 16 feet closer to the surface.

II. After reaching 77-16 = 59 feet, any further decompression would then start to cause true supersaturation unless he were to stop and let the inert gas re-equilibrate with its new alveolar partial pressure. If the diver continues to ascend, then the degree of supersaturation will increase until, at some critical level, the solutions "breakdown" and a gas phase forms, ie. he has "triggered" the primary event. The growth centres, or nuclei, will form bubbles and, if left long enough, will take up all of the gas in supersaturated solution.

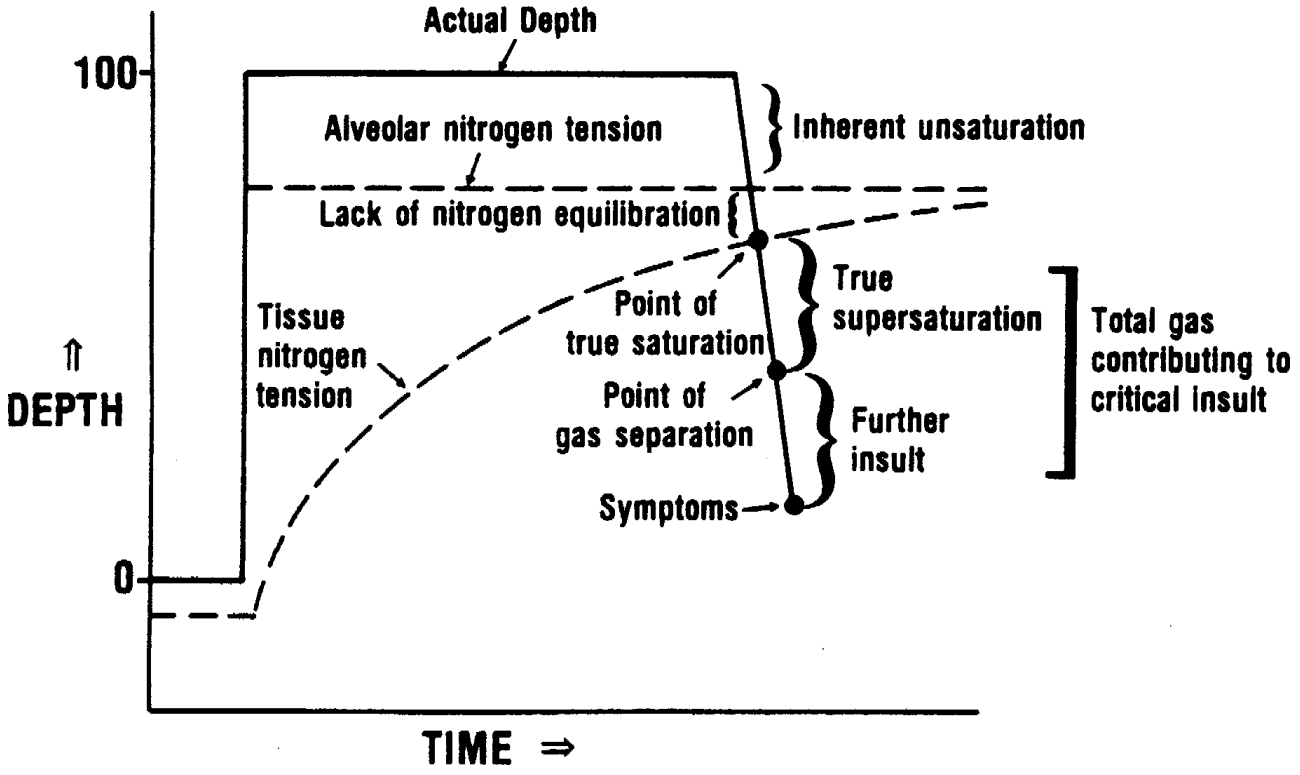

FIGURE 4

A no-stop decompression depicting the factors which can determine the degree of supersaturation and, when exceeded, how this and further decompression can contribute to the quantity of gas actually eliciting symptoms.

III. The diver would remain asymptomatic if he remained at the depth at which critical supersaturation broke down. By decompressing further, however, he now enlarges the gas phase which, in turn, increases the insult, by whichever mechanism applies, until it reaches its symptom-provoking threshold. It is during this phase of decompression that the mechanism becomes important and yet this is seldom considered in designing the table.

The above series of steps is depicted in Fig. 4.

True supersaturation

The physics of suppressed formation of the gas phase has been described in much detail in Decompression Sickness Volume I[1] but the discussion tends to become very academic as one pursues the questions of nucleation or what activates pre-formed nuclei into macro-nuclei which can then grow bubbles. Whatever the semantics involved, there does seem to be some degree of supersaturation needed before a fluid will start growing bubbles. The difficulty arises in trying to quantify this critical degree of supersaturation since the phenomenon seems fairly random and this tempts the investigator into using pure liquids to try to derive some underlying theme. Gelatin has been a popular model for many years[20,64,84] but this has no metabolism and does not reflect the many interfaces in tissue, especially a lipid-aqueous interface which is particularly conducive to bubble formation.[64]

The latter study has claimed that nucleation is very random and that, whereas about 70-80% of tissue can withstand substantial supersaturation,[57] some bubbles can form for negligible degrees of supersaturation, in fact much less than predicted by the Haldane ratio[20] or the fixed differential (*p-P*) first advocated by Hill.[85] The controversy essentially degenerates into a show of figures in which extremely high degrees of supersaturation can be shown in perfectly clean pre-pressurised pure liquids while some bubbles can be observed in animals following very small decompressions,[86,87] some barely exceeding the inherent unsaturation. Thus the controversy is transformed more into one of whether we should consider what the average tissue zone is doing or just the "worst possible case" of one bubble forming for a negligible level of true supersaturation.[64]

Alternating bubbles

The recent results on the tendon[81] would question the relevance of the above issue since gas will accumulate in non-perfused areas and soon exceed almost any published criterion for supersaturation. Thus bubbles should form whoever is correct in this academic argument. A bubble in living tendon, either intravascular or extravascular, can be seen to grow during decompression when the capillary bundle perfusing its site is closed and then shrink when that area is perfused. Thus one observes a whole range of bubble sizes as some shrink and others grow depending on the momentary distribution of blood flow. The important criterion then seems to be one of the sequence of perfusion, ie. vascular programming, rather than critical supersaturation. The same phenomenon can be seen on a much reduced time scale in the human hand[88] where areas do not change their boundaries but alternate in colour between pink and white. If one such area in a decompressed tendon misses its turn for perfusion, it can grow a bubble large enough to elicit pain. This assumes, of course, that the critical tissue for limb bends is tendon, but intermittent perfusion can offer a simple explanation why even the most conservative diving tables sometimes produce the odd limb 'bend". It would also provide a mechanism adding credence to the feelings of some designers of diving tables that the perfect bends-free table is either a myth or not cost effective. In fact it has been further suggested that it is the presence or otherwise of this phenomenon in a particular tissue type (eg. tendon, bone and skin) which determines whether that tissue is subject to insult and injury by decompression.

Practical diving tables

In the design of most diving tables, the inherent unsaturation and the growth of the insult are ignored. Ostensibly, the designer is avoiding the formation of the gas phase altogether by keeping on the safe side of his "trigger" points for the various hypothetical "tissues" he invokes. This is excellent providing the gas is remaining in true physical solution. In practice, however, it would appear that much of the gas is not remaining in solution but is forming bubbles and proceeding quite a way from the point of phase separation towards the critical insult for symptoms (Fig. 4). Thus even the much used tables of the US Navy would appear to be *treating* a gas phase rather than preventing it.[64] This may not be as serious a deficiency as it might sound since, according to my best estimates, it would take of the order of four times the total decompression time for the critical tissues to remain bubble-free. Thus, to be economically competitive, decompression schedules probably allow gas to separate from solution but prevent the insult from reaching the threshold for symptoms. This means, however, that the table was designed on the basis of preventing the gas phase from forming and yet, in practice, gas *did* separate from solution and, therefore, the table should have been formulated to minimise the insult. This is where it now becomes important to know the mechanism for provoking each category of symptom. Optimal conditions for preventing a gas phase which forms sooner than expected are unlikely to be the best for minimising development of the insult. This point is best illustrated by considering the effect of bubble formation upon the elimination of inert gas from a tissue.

Gas elimination

In gas uptake, there is no doubt that the driving force is the difference between alveolar partial pressure and tissue tension. Upon lowering the alveolar partial pressure by substituting another gas for the inert gas, the gradient can be reversed and so the exchange of gas will not only be reversed but will follow the same kinetics. If, on the other hand, lowering the alveolar partial pressure of the inert gas was effected not by substitution but by decompression and, moreover, by a decompression which caused gas to separate in the tissue, then everything is different. The tissue may contain the same total gas in all forms, but only the gas in true physical solution determines the tissue tension of that gas and hence the driving force for its elimination.

This very important point is demonstrated in Fig. 5 where dissolved gas simulated by the liquid in one tank is flowing into a lower tank representing the lung. If all gas remains in solution, the driving force is the head (H_1).

If gas is 'dumped' into the gas phase, however, this is equivalent to opening the valve when liquid will rapidly flow into a third tank representing separated gas until they are at the same head, ie. at the quasi-equilibrium where partial pressure in the bubble equals the tension of gas remaining in solution. However, the driving force for liquid flowing into the lower tank has now decreased from H_1 to H_2. So the driving force for eliminating inert gas from the tissue will decrease and allowance must be made for this in the formulation of a decompression table, yet none do, despite the many studies showing bubbles present even in the critical tissues for limb bends.

When the gas phase is present, then the driving force for inert gas elimination is simply the inherent unsaturation, which is much smaller than the hypothetical supersaturation used to compute standard tables, ie. we should be using H_2 instead of H_1 (Fig. 5) in our calculations.

Other symptoms

The above discussion applies primarily to limb bends (Category I) since these are by far the most common symptoms and, historically, have been taken as the ones to avoid when it was thought that other more serious symptoms were a further development of the same overall insult process. The question then arises as to what procedures should be taken to ensure that other categories are also avoided or that these are primarily avoided since, unlike limb bends, they have the capacity to cause permanent injury.

The two categories which can be made to precede limb bends by selecting the conditions are cerebral[89] and vestibular.[90] Taking Category II first since cerebral symptoms are fairly certain to be caused by arterial bubbles, it would seem most desirable to avoid any insult to the lungs which could cause them not to trap bubbles. This would suggest careful control of the oxygen prescribed during the decompression so as not to cause pulmonary oxygen toxicity.

The fact that Category IV symptoms can be provoked without decompression by inducing counter gradients of the 'heavier' inert gases indicates that it is also desirable to avoid a situation where one inert gas is adjacent to one body surface and another inert gas is in contact with another surface, unless favourably orientated. This creates the conditions for steady-state counterdiffusion[91] or exchange by counterperfusion,[92] or both, which might result in bubble formation in the vestibular apparatus.

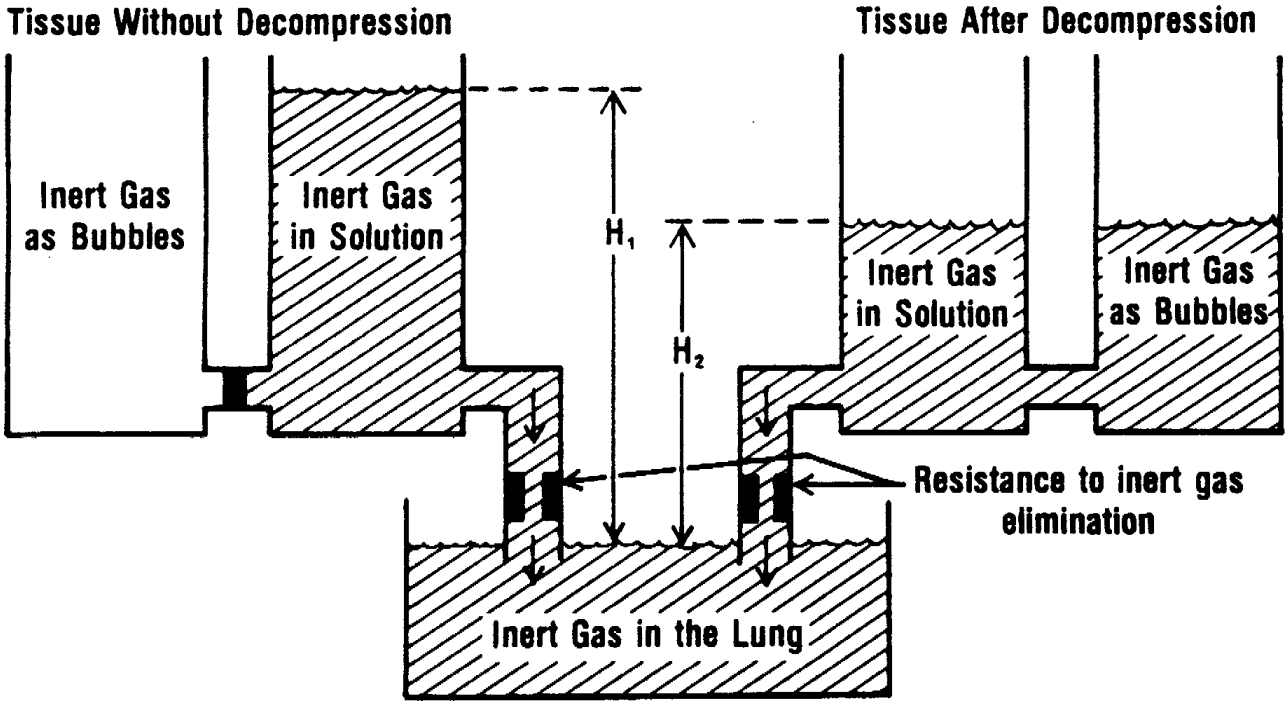

FIGURE 5

A hydraulic analogue for inert gas elimination from tissue, showing (on the left) how reduction of the alveolar inert gas partial pressure by substitution of another gas can produce a driving force for its elimination simulated by the head (H_1). On the other hand, reduction of the alveolar inert gas partial pressure to the same level by decompression *(on the right)* can also cause bubbles to separate from solution and so reduce the driving force for elimination, simulated by the head (H_2), even though the total gas in the tissue is the same as in the previous case depicted on the left.

Hence it is desirable not to switch from heliox to air rapidly as sometimes occurs in transferring divers from a bell ventilated with heliox to a deck decompression chamber pressurised with air.

These are some of the more obvious factors which can potentiate the more serious neurologic forms of decompression sickness which otherwise are produced by very few tables.

TREATMENT

Recompression

The resolution of a bubble depends upon its location. If it is extra-vascular, then it will be reduced in volume but remain in essentially the same site. Hence, if the diver is decompressed, the symptoms will return as before, as argued in favour of the extravascular mechanisms for Category I and Category III symptoms. Upon recompression, it is therefore necessary to hold the patient at pressure even though relief is complete in order to allow the much slower process of dissolving the gas to take effect.

The kinetics must be dependent upon any alternating patency of, say, a tendon for Category I or possibly the cord in Category III. It may be necessary to wait until the capillary bundle adjacent to the bubble is perfused before that bubble can be reduced in volume to any appreciable extent. The other factor influencing the kinetics is the driving force which is virtually the inherent unsaturation, as described in detail in Decompression Sickness, Volume I.[1] This inherent unsaturation can be greatly increased by breathing a high partial pressure of oxygen, in fact by roughly the elevation of the inspired PO_2. This offers a simple physical explanation for the efficacy of oxygen in resolving decompression sickness.

The other aspect of recompression is its effect upon intravascular bubbles, especially those occluding an artery from within. These require an appreciably larger volume change for complete dislodgment but are cleared most effectively by extensive recompression.[36,38] This may account for many unconfirmed reports of remarkable recoveries from neurologic symptoms upon a deep bounce with direct return to the surface. However, it is my experience with animals that, whereas most were cured, a few died. This could be attributed to the fact that recompression not only acts upon the occluded tissue but also upon the lung which is probably holding back many more trapped bubbles. If some of these were released, then the symptoms could be worse, depending upon where they happen to lodge.

Depth of recompression

According to the above argument, it takes a greater volume decrease to dislodge an intravascular bubble than it does to reduce the pressure with which a bubble is pressing upon a nerve ending to below the pain threshold. This is essentially reflected in the treatment tables where 165 feet (6 ATA) is the treatment depth for central nervous system involvement while 60 feet is recommended for limb bends only. In view of the possible effect of recompression releasing trapped lung bubbles,[93] lesser recompressions may be preferred for limb bends where there is complete relief shallower than 60 feet.

Gas for recompression

The last degree of freedom which the physician can prescribe in the treatment of decompression sickness is the gas mixture for recompression. For the reasons already discussed, the oxygen partial pressure should be high in order to increase the inherent unsaturation and, hence, the driving force for resolving the bubble. On the other hand, the overall exposure must not precipitate oxygen toxicity in any form. Naval treatment tables take account of the compromise necessary, but this is sometimes upset when the diver has already received an excessive oxygen exposure before the bend occurred.

The last question is which inert gas to use for diluting the desired oxygen to the point where adequate pressure can be applied to the bubble. This complex issue will not be discussed in this paper.

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SPUMS SCIENTIFIC MEETING 1983

The quality of the recordings of the question and answer sessions after Professor Hill's lectures left a lot to be desired. We were able, however, to salvage the following which covers three important points.

Dr Peter McCartney

What advice should I give to an abalone diver who asks about breathing oxygen after diving?

Dr Brian Hills

I think that what is going on between the primary event and the critical insult is that one triggers the gas phase almost instantaneously and the bubbles have to grow but they also have to coalesce. There was a classical study done by the US Air Force during World War II, in which they took continuous X-rays during decompression. When they went to a bends provoking altitude, say 25,0OO feet, a cloud came across the X-ray. Then when they exercised, one would see the cloud actually coalesce into bubbles. Those cadets did not complain of pain, until one could actually identify bubbles. So I would stick my neck out and say that the process which is probably rate determining is coalescence. Of course, exercise and the resulting movement of the facial planes, coalesces gas into a concentrated insult. Then the gas is in one place, and will press harder onto a nerve or a nerve ending or a blood vessel.

Now for the use of oxygen. I recommend to diving companies, to whom I am a consultant, that rather than use surface decompression tables, as one knows that if that diver remains on the surface and does not go back under pressure he is going to get the bends, give him a treatment table anyway. That has cleared up a lot of the problems which several of the companies have had in the Gulf of Mexico.

What is very controversial is what some companies are doing, changing to oxygen in the water. There have been some nasty accidents, because it is very, very unpleasant having what is virtually an epileptic fit on one of the diving stages. So one must make sure that the diver is well secured before giving him oxygen in the water. Many diving companies, for economic reasons, are letting their divers breathe oxygen at the 40 foot mark before bringing them up to the surface. But one must be careful. It is a controversial issue as to whether to do it or not, but it certainly cuts down the incidence of decompression sickness.

I would highly recommend that in place of the surface decompression table when the diver gets back to the surface one gives them a treatment, as though they have been bent. It is all treatment, anyway, as there is gas there the whole time and whether the diver has symptoms or not, one is really treating a gas phase.

ENCOUNTER WITH A BLUE-RINGED OCTOPUS A FAILURE OF MURPHY'S LAW

Based on a report to the PROJECT STICKYBEAK Non-fatal Incidents file.

Douglas Walker

To most people it is self evident that their lives are governed at all times by everything implied by saying "Murphy's Law", and to a casual observer this case fits the mould. That such is not the case is here related.

Joe, which naturally is not his real name, was on a day trip with his two young nieces to an island off the Queensland coast. They walked along the beach and the children saw some creatures in a pool. As they were too frightened of the unknown to pick one up, he did so. He seemingly had a vague feeling that the small octopus could be dangerous to children but felt safe in handling one himself. He threw away the first creature and picked up another, which he placed on the back of his left hand. It was after this one also had been discarded that he noticed a spot of blood where the animal had rested. There had been no sensation of a bite.

At this time he was near the launch which had brought them and told the skipper what had happened. This conversation was overheard by the pilot of a seaplane drawn up nearby in preparation for a flight. The pilot saw Joe collapse to the ground, which was due to his legs becoming weak, and remembered articles recently published in the local paper which gave warning that there were many blue ringed octopuses in the locality at that time. He had become interested and had read up the symptoms and correct management of victims of their bite, so immediately diagnosed this as such a case. With the help of two persons standing nearby he quickly assisted Joe into his plane and set off for the mainland where treatment would be available. During this short (7 minute) flight Joe twitched a few times and then became apparently pulseless and ceased to breath.

Joe's luck still held, for both the bystanders from the beach who had been co-opted to assist had accompanied him on the plane trip. They were not only aware of resuscitative measures but had recently attended CPR refresher courses. The crisis occurred about 7 minutes from the time of the bite. Because of their work Joe was alive on arrival at the airport, where an Emergency Ambulance awaited, summoned via the plane's radio. On arrival at the hospital an ECG confirmed asystole. Brain oxygenation was sufficient, despite the difficulty of resuscitative action in the confined space of a small plane, to prevent irreversible anoxic changes. The end result was complete recovery, though with some amnesia for the fine details of the incident and of the plane trip.

Remarkably few people are bitten by the blue ringed octopuses despite their large numbers. Which is extremely lucky, as the paralysing effect of their toxin cannot be reversed though it has a short period of action. Support for respiratory function is the total but essential management of anyone showing symptoms after contact with such an octopus.

SPUMS-RAN MEETING AT HMAS PENGUIN 27 August 1983

SOME RECENT RESEARCH AT THE ROYAL AUSTRALIAN NAVY SCHOOL OF UNDERWATER MEDICINE

John Pennefather

I will outline two studies undertaken at the School of Underwater Medicine. The first, a nutrition study, is reported to seek answers to the problems encountered. The second, a study on coelenterate stings, is mentioned again to correct some misapprehensions people got from the published paper.

NUTRITION OF DIVERS

A Clearance Diver in the RAN, undergoes a six month course. The first three weeks are spent learning to dive on air. A bit like a C Card with a lot of extra diving under supervision including night dives, and some PT. The next four weeks are spent learning to dive with oxygen sets. This can be very hard work involving up to 18 hours activity each day. It may include four one hour dives each involving an 1800 metre swim. This phase tends to act as a selection mechanism, people who are not strong or who lack a real drive tend to withdraw and change to another occupation.

The School of Underwater Medicine was asked a basic question about these men. Were they getting enough to eat?

Experiment 1

The first study of the problem was conducted by underwater medicine sailors who each observed one diver, noting his activity and food consumption. From this 14 men days of useable data were collected. This was processed by assigning an energy cost to each activity and an energy content to each part of the diet. The energy costs of each activity were based on published estimates.

The basic flaw was that the food intake was based on "standard" portions. It was not known if the helpings served were bigger or smaller. Also no allowance was made for uneaten food. The results obtained (Table 1) suggested that the food supply may have been inadequate.

Experiment 2

The study was repeated. With the instructor's agreement the students were given 30 minute meal breaks and at least six hours rest. This was because it could have been argued that the subjects in the first trial were too tired to eat or that they did not have time to eat their food. Subjects in the first experiment had as little as five hours sleep and meal breaks as short as ten minutes. In the second experiment the components of one meal selected as average size were weighed. Because of the lack of time and staff, waste was again ignored.

The result of the second study are shown in Table 1, Experiment 2. It will be noted that the intake and balance are closer but the balance is still negative. It will also be noted that although the time available for diving had been reduced, by the longer rest and meal breaks, there had been an increase in diving time. It was also noted that this group was quicker to perform their tasks. It is not possible to state if they were better supervised, or were a better quality group, or if the improved performance was a result of better feeding and more rest.

Experiment 3

The results of these two studies were mentioned in an internal navy report. The conclusion that the Royal Australian Navy might not have been giving its sailors enough food caused considerable concern. As a result the Army Food Science Establishment was directed to examine the problem. They expanded the study by following the weight and body fat content, estimated by skin fold thickness, through a course. They also reported the examination of the energy balance for 15 men days of oxygen diving training. This time food intake was measured as accurately as possible, each component was weighed, as was uneaten food. Because trained staff were available more detailed activity records were kept. This time the "average" diver had a positive energy balance, Table 1, Experiment 3. Sixty minute swimming is recorded with the diving time because one of the subjects developed ear trouble and could not dive. Rather than leave him out he swam the same distance on the surface when it would have been his dive. If both diving and swimming are counted the total time in the water was up again.

Most people were happy with the results. It had been shown that there was not a food problem so the administrators were happy. The instructors were happy because the students performed better.

TABLE 1

ENERGY BALANCE STUDIES

	EXPERIMENT 1	EXPERIMENT 2	EXPERIMENT 3
MAN DAYS	14	12	15
INTAKE	2796 KCAL	3892 KCAL	4533 KCAL
EXPENDITURE	3462 KCAL	4193 KCAL	4499 KCAL
BALANCE	-666 KCAL	-301 KCAL	134 KCAL
DIVING TIME	158 min	190 min	147 + 60 mins swimming

It is of concern that the experiment series may contain an unintentional bias. No warning was given of the first experiment, a few sailors appeared and made notes. The second time it was obvious that something was going on. Food was weighed and I was taking notes of activity and food consumption. By the time the third experiment started the catering staff knew that the quality and quantity of food was being studied. At each stage the food quality and quantity went up. For example in the first experiment food was often left on the plate. During the third experiment the divers often went back for second helpings.

It may be significant that the two divers with the most negative energy balance in the third experiment subsequently gave up diving. These divers also showed the greatest weight and body fat loss. They had no major reason for giving up so it is possible that energy deficiency caused them to lose interest.

Another problem is the energy cost of rewarming a diver. A 70 kg diver having four dives in cold water loses heat. If the heat lost is replaced by extra metabolism he uses extra energy. If the body heat content is slowly made up by conserving heat, by vasoconstriction, little extra energy is needed. Conservation was assumed in our experiments and this could result in an underestimate of energy expenditure of 600-800 Kcal. An answer to this question, or a technique for solving the problem that caused no interference to the subject, would be of great interest.

COELENTERATE STINGS

The second study was of the treatment of Portuguese man-of-war or blue bottle, stings. There are two species of coelenterates that cause significant problems in Australian waters. The box jellyfish, Chironex fleckeri, in the north has caused over 50 deaths. The Portuguese man-of-war, or blue bottle, a physalia, often causes the closure of Sydney beaches.

Until the late 1970's methylated spirits was the most common treatment, applied liberally to the sting area. Then a group in Townsville reported that the application of methylated spirits caused box Jellyfish nematocysts to discharge. They reported that vinegar and other dilute acetic acid mixtures inhibited discharge. Their interest in the subject may have been caused by a case they reported of a woman who got no relief from methylated spirit but got drunk on the fumes.

We decided to see if vinegar was of use in treating blue bottle stings. It was compared with methylated spirit, "Stingose", which is a commercial anti-sting preparation containing aluminium sulphate, and sea water.

Twenty "volunteers" were stung with bits of tentacle on four sites on the inner side of the fore-arms. After two minutes one site was treated with each remedy.

All the tricks required by the statisticians were followed. The subjects and observers did not know which remedy was used on each site, treatments were changed from site to site so subjects could not compare results, etc. The subjects were asked to assess pain just after treatment, and 5 and 10 minutes later. Skin reaction was assessed by an observer 5 and 10 minutes after treatment.

The results obtained are summarised in Table 2. The Unsure line indicates that the subject or observer could not decide which area had the greatest or least response.

The first and most obvious conclusion was that methylated spirits was worse than no treatment. The second was that vinegar and "Stingose" worked some of the time with vinegar marginally better. As neither gave a good response the conclusion was that local anaesthetic was needed for severe stings, which had previously been suggested by Carl Edmonds in Dangerous Marine Animals.

The response to these mild conclusions suggests that they were published on a no-news day. TV, radio and newspapers suggested we had found a wonder drug. The results justify no such claim. Methylated spirit would appear to cause more pain than salt water so one would have been better staying in the surf (provided one was not stung again). "Stingose" and vinegar have some use but do not work all the time.

Our results are of interest to two groups of people. For researchers it is an interesting area where a better answer is needed. For divers and medicos who practise near the beach, the message is try vinegar but have your local anaesthetic handy.

TABLE 2

BLUEBOTTLE TENTACLE STINGS
ASSESSMENT OF PAIN AND SKIN REACTION

TREATMENT	MOST PAIN	MOST RELIEF	MOST SKIN REACTION	LEAST SKIN REACTION
Vinegar	7	25	1	17
Stingose	4	19	6	10
Methylated Spirits	27	1	17	2
Salt water	19	9	12	4
Unsure	1	6	4	7

Question.

How did you calculate the energy expended in the first study?

John Pennefather.

There are books of tables of energy intakes and expenditure. For most activities we used these tables. For the oxygen diving time we could calculate the energy used as we know how much oxygen the set receives each minute and the divers swim along using just that amount of oxygen.

Question.

So really it was not very accurate?

John Pennefather.

No, but that is the way every nutritionist does it. They use the tables and make no allowances for changes in temperature, which surprised me. I have had very little nutrition training, and I always believed the figures.

Question.

Did you follow the body weight of the divers? That is the best indicator of their imbalance, providing they have adequate food.

John Pennefather.

Over six months quite often these people will lose three or four kilos, but in some cases it is an obvious loss of fat. The ones that I am worried about are the thin ones. I said two had dropped out, they were fairly scrawny people at the beginning, they became more scrawny, and they eventually gave up, without giving any reasons, they just said "It is not for us." I think they are the people that we might be losing. What we could do next is to follow skin fold thickness and bodyweight.

TESTING SURVIVAL SUITS *ARE THEY GOOD ENOUGH?*

Arvid Påsche & Susan Gordon

In 1982 the Norwegian Underwater Technology Center evaluated the thermal insulating properties of a number of survival suits, such as are routinely worn by all personnel during helicopter transport offshore. The purpose of the suits is to keep their wearers afloat, dry and reasonably warm in the event of a crash-landing in the sea. Hypothermia is recognised as the major danger for survivors of helicopter crashes.

The purpose of the study, which was to be as realistic as possible, was to highlight the practical aspects of using various makes of suit, and provide a clearer picture of each suit's advantages and drawbacks. The study was funded by Statoil, and was fully described in NUTEC Report 43-82.

There were three parts to the testing routine:

1. Evacuation: evaluation of suits during evacuation of a sinking helicopter; determination of water leakage in the suit during evacuation.
2. Buoyancy: determination of total buoyancy for a person dressed in a survival suit.
3. Thermal insulation: evaluation of the insulative properties of suits after evacuation.

Part 1, Evacuation, was carried out at the Offshore Survival Centre in Aberdeen, where a "helicopter underwater escape trainer" enables personnel to be trained in evacuation procedures. These tests showed that the chances of water entering a suit during evacuation is fairly high, even if the suit is zipped up and the hood is worn. Between nine and ten kilos of water can enter a suit which is not fully zipped up.

The buoyancy tests were performed in the pool at NUTEC, using a specially weighted chair equipped with a safety belt to simulate a helicopter seat. The chair could be turned so that the subject would find himself sitting in a head-down with closed (A) or open (C) zippers, or head-up (B) position underwater (for results, see Table I). Some of the suits had buoyancies as high as 40 or 50 kp. It is unlikely that many people would have the strength and training necessary to force themselves down and out of an upturned helicopter if they were carrying so much buoyancy.

TABLE I

Suit		Buoyancy A	B	C
HELLY HANSEN E353	Mean:	41.7	19.38	14.35
AQUA SUIT	Mean:	26.15	15.33	13.42
MUSK OX	Mean:	10.67	8.3	13.42
NORD 15	Mean:	39.53	18.7	12.23
MULTIFAB	Mean:	16.8	12.10	7.8
IMPERIAL "H"	Mean:	20.5	12.2	10.45
PIONER/LIUKKO "COMBIE"		46.5	27.4	17.5
PIONER/LIUKKO "PILOT"		31.4	27.4	10.0

The thermal tests were also carried out in NUTEC's indoor pool, which was filled with seawater at a temperature of 9-11°C. With air temperatures as high as 15°C and no windchill factor, the test did not attempt to reproduce "worst case" conditions. All subjects wore a cotton jacket and trousers under the suits, and in some cases (see Table II), woollen underwear as well.

Both wet and dry tests were run. In the wet tests, subjects let 12 - 13 kg cold seawater into the suit by immersing with a partially open zipper, much as might happen in a real-life emergency. Then they lay quietly in the water for a maximum of two hours. Tests were terminated early if a subject's rectal temperature dropped to 35.5°C or if he felt too cold and uncomfortable to continue. Rectal temperatures were used to calculate average cooling rates, which are presented in Table II.

TABLE II

Average rectal cooling rates ($°C\ hr^{-1}$)
(mean values)

Suit	Dry	Wet
AQUA SUIT	0.65	0.70
HELLY HANSEN E353	0.45	1.68
MUSK OX	0.88	2.51
MUSK OX with woollen underwear	0.78	1.33
NORD 15	0.65	1.85
IMPERIAL "H"	0.47	0.49
MULTIFAB	0.48	1.66
MULTIFAB with woollen underwear	-	1.56
PIONER/LIUKKO "PILOT" with woollen underwear	0.73	0.96
PIONER/LIUKKO "COMBIE"	0.41	1.01

In addition to the test series, observations were also made on certain aspects of the suits' performance.

A wet Nord 15 suit, for example, was found to have a buoyancy of only 3.6-3.7 kp, or 60% of the buoyancy required for approval by the Norwegian Maritime Directorate. This is not enough to keep a person afloat.

Wet Helly Hansen E353 and wet Nord suits acquired significant alterations in stability. The subject in these suits had a tendency to tip forward and end up with his face down in the water.

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ESCAPE FROM DITCHED HELICOPTERS

SHOULD WE HAVE A SIMULATOR IN NORWAY?

Odd Pedersen
Norwegian Underwater Technology Centre

In 1982 some 600,000 persons were transported by helicopter between Aberdeen and offshore installations. By 1980 total helicopter passenger trips from Norwegian airports were about 540,000. In 1982, Helikopter Service A/S alone carried some 9,000 persons per month between Bergen Airport (Flesland) and the installations, plus another 2,000 between platforms, and this company expects its traffic to double from this figure before the end of 1984. By the end of the 1980s, we can expect half a million passenger trips per year from Flesland. These figures were obtained in the course of an NTNF-financed pilot project undertaken by the Norwegian Underwater Technology Centre (NUTEG) to investigate the possibility of reducing fatal accidents through training courses in helicopter evacuation.

ACCIDENTS WILL HAPPEN

Even with the high maintenance standards and strict procedures of helicopter companies operating in Norway, such large figures suggest that accidents will occur from time to time. Statistics are difficult to come by, since so much of the world's helicopter traffic is military, but a few figures obtained from diverse sources suggest the extent of the problem.

In the USA, in 1978, there were 16 civil helicopter crashes per 100,000 flying hours. US non-war-zone military helicopter accidents occurred at a rate of 6.5 per 100,000 flying hours in the period 1968-1978.

Between 1968 and 1978, there was one civil helicopter accident per 100,000 hours flown by British-registered aircraft. The British authorities recorded three accidents to foreign-registered machines in the same period.

Norwegian military authorities point to 17 helicopters written off (ie. non-repairable) between 1953 and 1982. The total accident rate has been 32.8 per 100,000 flying hours.

There have been 32 crashes of Norwegian-registered civil helicopters between 1972 and 1981, plus a number of controlled emergency landings.

Even though many offshore workers are unhappy about flying to work by helicopter, it is unlikely that other methods of transport will be used to any great extent in the foreseeable future. The overwhelming proportion of helicopter trips end of course in a safe landing on a helideck, but a tiny number will have one of the following outcomes:

a controlled landing on the sea, with the machine floating upright, or

a controlled landing, but the machine capsizes because of its high centre of gravity and sea motion, or

a crash landing in the sea.

TRAINING COURSES CAN SAVE LIVES

Both the USA and the UK have set up training programmes in evacuating crashed helicopters, and the Norwegian military authorities send helicopter crews to the British training centre near Aberdeen. Helicopter evacuation courses have dramatically reduced the death rate among helicopter crews in both the UK and the USA, where it dropped from 34% among untrained to less than 9% among trained personnel (234 accidents involving 1093 persons). In Norway, however, the Church and Education Ministry has refused to invest in an evacuation simulator and training pool for seamen and offshore personnel who regularly fly in helicopters either as crew or as passengers.

The numbers who might benefit are difficult to estimate accurately, but the various maritime training centres in Bergen currently turn out some 600 students per year, and this figure will rise by 400 when the new school at Sund near Bergen opens in a few years. It is expected that the offshore companies now being established in and around Bergen will have about 1,000 employees whose work will take them offshore while there will be a similar number of contractor's personnel based in Bergen. These figures do not take into account personnel operating out of other offshore centres along the west coast of the country. After generous allowances have been made for overlap between the groups, a reasonable figure for potential students for a helicopter evacuation course would be of the order of 1,600 per year. To this figure should be added a certain number of students from the Norwegian Navy and from Helikopter Service A/S, both of which have expressed interest in training their personnel in Norway rather than in the UK as at present.

The conclusion of the NUTEC study was that with the increased level of activity we can expect in the years to come, a training course in helicopter evacuation would be highly desirable. With its existing facilities and range of interests, NUTEC could be an appropriate institution to run training courses in helicopter evacuation. An annual programme of such courses could have synergetic effects and help to flatten out peaks and troughs of activity at NUTEC caused by large-scale short-term projects. New facilities might include the helicopter simulator itself and possibly a new outdoor pool with a crane to lower and raise the simulator. The training staff would include at least three trained divers plus ancillary personnel. NUTEC will make a final proposal about a course in September of this year.

HELICOPTER EVACUATION
A POSSIBLE COURSE OUTLINE

A course should consist of both theoretical and practical elements. A preliminary lecture would describe modern passenger helicopters and introduce the students to such physiological concepts as hypothermia. Survival suits and their use could also be described, and relevant test results presented (see for example NURN Vol. 4, No. 1 1983, article by Påsche and Gordon, reprinted in this issue of the SPUMS Journal).

Following the theoretical introduction, a class could be divided into small groups, each of which would be given practical (ie. wet) instruction, during which the simulator would be:

a. sunk slowly into the pool, in an upright position.

b. dropped from 2m over the surface, in an upright position.

c. sunk slowly into the pool, turning round as it goes down.

d. dropped from 2m over the surface, "capsizing" as it sinks.

Such a course would require about five hours and involve several personnel. Capital equipment depreciation, plus likely running costs, maintenance, survival suits and salaries suggest a cost per student of between 500 and 600 Norwegian Kroner. In addition to reducing yet further the number of helicopter fatalities suffered over the Norwegian continental shelf, introduction of such a course would also help to lower the psychological stress of regular helicopter flying for offshore personnel.

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DEMONSTRATION DIVE TO 350 MSW

Stein Tønjum

Norwegian Underwater Technology Center

In the Statpipe project, a combined oil/gas pipeline will link Statfjord with Kårstø in Rogaland county, crossing the Norwegian Trench at a maximum depth of 297m on the way. Although the pipelaying contractors Seaway Diving (Stolt-Nielsen) plan to carry out most of their work using diverless systems, they want to be sure that currently available diving techniques (and divers) are up to the job if they need to be called in. This is the background for the 350 msw chamber dive which the Norwegian Underwater Technology Center (NUTEC) has successfully completed for Seaway Diving.

Although NUTEC has already made chamber dives up to 500 msw, the current limit for operational North Sea diving is still around 170 msw. The Seaway dive was carried out to bridge the still wide gap between experimental and operational dives to greater depths.

The six Seaway divers selected for the demonstration dive were given four weeks of intensive training and medical-physiological examinations at NUTEC. On January 31st they entered NUTEC's chamber complex and reached 350 msw on heliox in 24 hours. The new compression profile which was tested in this dive gave rise to only minor symptoms of High Pressure Nervous Syndrome (HPNS) and the divers were all able to carry out their scheduled tasks. These consisted for the most part of cold-water simulations of the sort of medium to heavy work they may have to do on Statpipe. For this purpose, a specially constructed test-rig was placed in the wet chamber.

The divers worked in two eight-hour shifts from 0800 until midnight with at least one diver in each team working in the water for three to four hours per shift. This schedule corresponds to the rules laid down by the Norwegian Labour Directorate for shallow North Sea dives, and provided experience which will help determine whether the same schedules can be used for greater depths.

The divers and their equipment were also closely monitored during the whole dive. The divers underwent neuropsychological tests, checks of thermal balance in water, blood and food balance studies, bacteriological survey, measurements of muscle strength, lung function

tests, and circulation studies which were combined with detection of gas bubbles during decompression.

After five days at 350 msw the divers started decompression and reached surface eleven days later. More than 40 people were directly involved in the operation, which lasted for a total of 18 days.

The comprehensive post-dive medical examination was identical to the pre-dive routine and will be repeated yet again in a year, as part of the process of identifying possible long term physiological effects of deep diving.

The successful dive was thus an important step towards clearing the way for deeper operational diving. NUTEC is now in the process of analysing the mountain of data collected by the various projects.

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DECOMPRESSION AND CIRCULATING BUBBLES

Alf O Brubakk

For many years, ultrasound has been used to study the gas bubbles which can develop after a decompression procedure. These gas bubbles can be present either in the tissue or the blood stream. Gas is a very effective reflector for ultrasound, and hence the reflected intensities from the gas bubbles will be high.

Circulating bubbles can be studied by using ultrasound and the doppler principle. This principle, well known in everyday life, states that the reflected sound from a moving object will have a frequency shift proportional to the velocity of that object. Ultrasound reflected from the blood cells will thus describe the velocity of these cells. Heart valves and vessel walls will also reflect ultrasound, but the velocity of these structures are much lower than that of blood. Circulating bubbles will be very strong reflectors and can thus clearly be heard above the signals from the blood.

In most studies till now, continuous ultrasound has been used. In this system a continuous beam of ultrasound is transmitted towards the body and the frequency shifts of all reflectors in the beam are recorded. Ultrasound can also be used in the pulsed mode. Here ultrasound is transmitted as pulses and at a certain time after the transmission of the pulse, the reflected signal is received. As the velocity in the body is known, this will mean that velocities are recorded in a rather well defined sample volume. The advantage of this is that small segments of vessel can be studied and that the signal-to-noise ratio will increase.

Although studies of bubbles in the circulation have been performed for many years, there is still no good theory to describe how these bubbles are generated, how they are transported and how they behave. In the same dive, two divers can have vastly different amounts of circulating bubbles, and there is no clear-cut relationship between bubbles and symptoms of decompression sickness. We have therefore decided to work on methods to improve the detection and sizing of bubbles in order to clear some of these points. The work has considerable theoretical interest, but is also of practical value, as it could allow us to develop better decompression schedules.

During the two Deep-Ex dives, bubbles were recorded in the vascular systems of the divers participating. A system was developed that permits recording of ultrasonic doppler data inside the chamber. The divers were trained to record from several arteries and veins, in the extremities, inside the heart and to the head.

During the 300m Deep-Ex 1 dive, the divers performed several ascending excursions going from 300m to 250m. During these excursions doppler data were recorded. We found a considerable number of bubbles particularly in the veins coming from the legs. In the arms considerably fewer bubbles were found. Of particular interest was the observation that some of the divers had bubbles in the carotid artery. These bubbles in an artery leading to the brain were particularly disturbing, but none of the divers experienced any clinical symptoms.

During the 500m dive, bubbles were also recorded. We performed one excursion from 500 to 445m. During this excursion, one of the two divers had a large number of circulating bubbles in the veins and also some arterial bubbles. During decompression from 400 to 300m at 3m an hour, considerable numbers of bubbles were found at 325m, also with bubbles in the carotid artery. During decompression to the surface very few bubbles were found.

As we were interested in developing a method for counting the number of bubbles as well as for trying to estimate their size, a collaboration with the Department of Engineering Cybernetics, The Norwegian Institute of Technology, was established.

They have for many years worked on the development of doppler ultrasonic equipment. Hans Torp at that institute is working on a doctoral thesis on the reflection of ultrasound from gas in bubbled blood. He has developed a method for estimating bubble size and number, based on the reflected intensities from the bubbles and the use of several ultrasonic frequencies. This method is implemented on a microprocessor, which enables us to get an instantaneous plot of signal intensities, which has a relationship to distribution of bubble size. This system was first tested during the 50Om dive and continuous testing is going on during shallow dives. The preliminary results are encouraging.

In order to test the method it is necessary to know the actual size of the bubbles studied. This will be done in collaboration with the Institute for Surgical Research, University of Bergen, where we will use a microscopic technique to visualize the bubbles at the same time as we record the signal with the doppler system. This work will be started in autumn 1982.

The aim of this project is two-fold. One is to get more information about the basic behaviour of bubbles and the laws governing their development and transport. The second is to develop practical methods to monitor divers during actual decompression procedures. This is of particular importance for deep divers, as it seems that arterial bubbles can be generated during accepted procedures in deep dives. These gas bubbles will have high internal pressure and hence will stay for some time in the circulation and it is conceivable that repeated dives using these procedures can lead to damage.

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DIVERS DO IT DEEP

Nine skilled and experienced professional divers recently completed a course of training to qualify them as Diver-Medics at Fremantle Hospital.

It is believed to be the first such course to be held in the southern hemisphere and probably the only such course in the South East Asian Pacific region.

Students came from Singapore, from Bass Strait and from Western Australia to train in the techniques needed to examine and care for divers who become ill or injured during their work.

Many divers working in the oil fields have to live under pressure for many days at a time. They cannot be brought out to normal pressure outside quickly without killing them, so if they become ill or injured they must be dealt with under pressure.

The students learned techniques of giving injections, putting up intravenous drips, and other skills so that they can act as a doctor's eyes, ears and hands within the decompression chamber. A doctor may be many hundreds of miles away and only able to communicate by radio in the first instance.

Students were able to view at first hand many of the techniques that they learned by observing in the Operating Theatres and Emergency Department at Fremantle Hospital.

OFFSHORE DIVING
NORTH SEA PROGRESS REPORT 1984

Commander SA Warner
Chief Inspector of Diving
Department of Energy UK

This is the tenth successive year that I have had the honour of being invited to read a paper at your symposium. This year, with the permission of your Secretary, I have invited Per Rosengren of the Norwegian Petroleum Department to share this slot with me. In the North Sea we work very closely together and in particular, I know he would wish to talk to you about the tragic accident, in which five divers lost their lives. He will describe how it happened and all the lessons that one has to learn from such a tragedy. *(It is hoped to publish this paper in a later issue).*

Legislation

UK diving legislation is now well established and understood almost throughout the world, but, like any legislation in a rapidly changing arena one has to periodically review the situation and amend where necessary. In the UK we are currently studying the guidance notes which support the legislation with the intention of updating those notes. We believe that the actual law with minimum amendment is at present adequate. However, it is our intention to introduce minor amendments to legislation which will require every diver and supervisor to hold a first aid qualification varying in levels of knowledge with his particular diving standard. We will also require a three year refresher for the renewal of the appropriate first aid certificate. In actual practice, all the UK approved diving training schools already teach to the proposed standard.

In addition, we have looked at the legal requirements for a diving supervisor. It is our intention to remove the present requirement for him to have experience either as a diver or as a supervisor of the diving technique to be used in the operation for which he is appointed.

The original requirement has become outdated because of the increased complexity of operations (especially bell and saturation operations) where specialised supervisors are required and also because, of the inhibiting effect, such requirements have in relation to the introduction of new technology. In the future the duty to ensure that the supervisor is competent will rest more firmly with the diving contractor. This will also mean that the various technicians in the control team may achieve supervisor status.

Training

In the UK and Europe we are still putting considerable emphasis on training. The pattern of diving is changing with the increased need for inspection, repair and maintenance of structures which means, more and more diving within the air range. The introduction of dynamically positioned vessels also emphasises the need for new techniques and with the changing pattern it is necessary to review the training standards to ensure that they are keeping up with modern trends. This is being done in consultation with Norway and the rest of Europe in parallel with the updating of the guidance notes.

Dynamically Positioned Diving Support Vessels

When dynamically positioned (DP) support vessels were first introduced very little thought went into the design of the air diving stations in these vessels. The main emphasis was placed on deep diving bell operations. However, with the increase in air diving requirements they are being used more and more for such operations revealing many short-

comings for such work. Good communications between the dive station and bridge, which is so important, have sometimes been lacking. The appreciation of the dangers of divers or their umbilicals being caught in propellers or thrusters has sometimes been ignored and the fact that DP vessels go "walkabout" on occasions, forgotten. In the UK we insist upon the length of diver umbilicals/life lines being restricted to a length which absolutely prevents diver or umbilical coming into contact with any propeller or thruster. DP capability for diving support vessels is attractive and they have helped to advance diving techniques but they are not the panacea of all diving ills and they do have limitations especially when operating in the close vicinity of a platform. These limitations must be recognised and catered for otherwise unacceptable hazards could be introduced into the diving programmes.

Accidents and Incidents

1983 showed a total of 40 incidents in the UK Sector, one of which, was fatal. This fatality happened when the diver was surveying a pipeline, unattended but using a surface marker float, in 54 feet of water using SCUBA with a free mouthpiece. The consequential post mortem showed that death was due to drowning.

TABLE 1

1983 DIVING INCIDENTS IN THE UK SECTOR OF THE NORTH SEA

(Rearranged and expanded from original table)

40 INCIDENTS		ONE FATAL
Type of incident	Number	% of total
Human error	13*	33%
Decompression Sickness		
Type 1	10	24%
Type 2	8	18%
Equipment failure	8	18%
Lack of training	2	5%
Lightning strike	1	2%

* *This figure is calculated from 33% of 40 incidents. The other numbers are taken from the text. The total number of incidents becomes 42. So presumably two were due to human error and another cause.*

One worrying trend in 1983 was the number of fires in air atmosphere welding habitats. Although none of the incidents caused actual harm to any diver they were all potentially dangerous. Fire, once again, provided reason for concern when a chamber complex under air pressure test at a pressure equivalent to 600 feet went on fire. Fortunately, nobody was hurt. However, it does bring home the fire risk in enhanced oxygen atmospheres.

There were ten recorded cases of Type 1 decompression sickness and eight of Type 2. This seemed a very strange ratio which we are investigating. All cases occurred in air diving with a vast majority in the 100 to 200 feet range.

Human error comes top of the list as always but there appears to be a recognised reduction in this factor.

Lack of training, I am pleased to say, was only considered to be a cause or contributory cause in two incidents where equipment failure was a factor in eight incidents.

The computer came up with one incident under the heading of "Act of God" which generated from a "lightening strike".

Research Projects

Considerable effort and money is still being invested into research projects effecting diving safety. It is hoped that we will be able to issue guidance on the physiological design parameters and test procedures for underwater breathing apparatus during 1984. However, it is not an easy task and was described at one stage as "a contest of few informed participants surrounded by many ill-informed spectators".

Other projects covering diver heating, atmosphere control, communications, acceptable acoustic levels etc., are progressing.

One medical research project is looking into possible chromosomal damage in divers and the first report on the study will be published this year. Once again, this is an extremely complex project but, in the UK we believe that it is our duty to look at any and every aspect of diving that may have so far been undiscovered or have long term effects on the diver.

Before handing you over to Per Rosengren I would like to leave you with three thoughts for the offshore industry for the next year:-

1. Never dive deeper than the depth of your onsite chamber.
2. Treat all oxygen/helium bends with an oxygen/helium schedule.
3. Treat all air bends regardless of apparently trivial symptoms as Type 2.

SO SAY SOME OF US

"2i Protection of Species"

Practice of deliberate free ascents in underwater competitions is not warranted except with certain safety precautions which must include a functional recompression chamber available directly at the site along with medical personnel experienced in diving medicine. Although it may be possible to provide such precautions for an Australian Championships, they would not be generally available for pre-competition practice. Consequently deliberate free ascents should not be included in Australian Underwater Federation Competition in the foreseeable future."

Extract from By-Laws Rules and Regulations of the Australian Underwater Federation, 1984.

UPDATE ON DISABLED SCUBA DIVERS

VANCOUVER, BRITISH COLUMBIA, CANADA

Margaret Campbell

It was 7.30 am, October 2, 1983. As I drove down to the dock, the brilliant red-orange sun was just rising. It promised a beautiful autumn day. The drive site was to be Croker Island, approximately two hours northeast of Vancouver, up the scenic Indian Arm. Individuals could be seen piling Scuba gear onto the Sun Seeker, a Scuba charter boat. This wasn't an average Scuba trip but the first outing of the newly formed disabled Scuba club. Five of the 12 divers were disabled - 2 paraplegics (Peter and Ken), a transverse myelitic (Jeff), 1 poliomyelitic (Lenny) and 1 legally blind (Lisa). Two of the divers, Lisa and Jeff, an Olympic disabled swimming champion, were completing their course with this last checkout dive. Two cameramen (both divers) from the Canadian Broadcasting Corporation (CBC) were also on board to film the dive for a future newscast.

Interest in Scuba diving by the disabled community has been growing ever since the original 5 divers completed their PADI course in May 1982. (Letter to the Editor, SPUMS J, January-March 1983). Up to now there have been 3 classes, the first consisting entirely of disabled students and the other classes combining disabled and able-bodied students. One of the able-bodied students scheduled to complete the boat dive, unfortunately, had an accident two days before with her bike and had broken her elbow. She was on board, however, and provided one handed assistance.

The weather was warm and sunny for most of the day, which aided the film crew but quickly overheated the suited-up divers. Becoming a "film star" isn't all glamour by any stretch of the imagination! We were put in shifts so that each buddy pair or team of three could be filmed for different purposes - donning gear, entries, descents, underwater and exits. Another lady and I were teamed with Ken (incomplete T12) who hadn't been in the water since he was certified (May 1982). Ken was very eager to return to the weightless condition. What with the excitement of 'getting in' and the novelty of being filmed, the first mistake was putting the wetsuit 'john' on backwards. That was okay because the camera wasn't ready.

Ken was using rental gear which didn't fit very well as his lower extremities have considerable muscle wasting whereas the upper extremities have hypertrophied. This necessitated more assistance with suiting up than would normally be required. We adjusted the weight belt so that the buckle was on the side, as he needed the bulk of the weight in the front of his body. In the excitement, Ken had left his fins in his van so we had to borrow from Peter, who wasn't to be filmed until later. Due to the strength of Ken's upper body, he required minimal assistance to perform a back roll entry. Once into the water, we had to wait for the film crew to suit up and film the descent from above and below water. Ken required a moderate steering assist to snorkel to the bow. He had a difficult time clearing his ears on the descent down the anchor chain. This involved approximately 10 minutes but we did clear and descended to the reef 45 feet below. The visibility was 20-25 feet and the reef looked very inviting. Unfortunately, at this time, Ken's weight belt came unbuckled and with him hanging onto the ledge, his two buddies finally managed to buckle him up. With time getting short, we cruised briefly over the reef, seeing white plumose anemones, chiltons, convict fish, sea cucumbers, small shrimp, lingcod and rockfish. We ascended the anchor line without incident but once to the surface, the weight belt slipped down his legs and dropped to the bottom. We manoeuvred Ken, who by this time was quite tired, back to the stern.

The film crew meanwhile had gone on to film Peter (incomplete T5) and Lenny (polio) who have been diving regularly since they were certified. Normally, we would not have two disabled buddies pair up and it definitely is not recommended procedure, but there were 7 other divers in the immediate area at the time. Lenny, with one atrophied leg, uses only one fin. Peter's biggest problem is clearing his ears while swimming with his hands. Boat dives with descents down the anchor line ease this problem considerably. Lisa, with her buddy replacing her guide dog, was managing very well.

After lunch, the film crew decided to film Jeff (transverse myelitis) underwater. Jeff uses adapted hand flippers, rectangles of firm plastic held on with rubber tubing. The remaining divers were free to do a pleasure dive. The reef was beautiful and the autumn threat of red jelly fish never materialized to spoil our pleasure. Once the last divers were aboard, the Sun Seeker weighed anchor and a tired group headed back to Vancouver. During our return we discussed and brain stormed on various possible adaptations, such as safer weight belt buckles or catches, and ankle weights to maintain a disabled diver in a more vertical position for descent. We all agreed that a lot of work is needed to increase the safety for the disabled diver, that being a buddy for a disabled diver is a great responsibility and that the able-bodied buddy needs to be even more aware of the potential problems. The best source of ideas is the disabled diver, who has had to learn to cope on land.

As with most film productions, problems arise and ours was no exception. The underwater footage of Peter and Lenny didn't develop properly and we are now awaiting a free weekend when the two of them are not off wheelchair marathoning, playing competition tennis or basketball, so that the extra footage can be completed. Once that is done, we should be on national television!

BACK TO THE DRAWING BOARD!

Operators of an off-shore platform on the North-West Shelf have laid great emphasis on safety, frequently casting a dummy into the seas to monitor crew reaction. Recently this life-like dummy was dropped into the water for another exercise, only to be rapidly devoured by a passing shark.

The company is re-examining procedures.

Reprinted from The Australian, 27 October 1983.

DECOMPRESSION SICKNESS IN THE GREATER TOWNSVILLE AREA

Report of a public meeting, held in Townsville on 25/11/83, on the subject of Decompression sickness occurring in divers in the greater Townsville Area.

Fifty-one persons attended, comprising a cross-section of the Townsville and district professional and sport diving community.

ANALYSIS OF 15 CASES OF DECOMPRESSION SICKNESS TREATED AT THE AUSTRALIAN INSTITUTE OF MARINE SCIENCE RECOMPRESSION CHAMBER BETWEEN 1977 AND 1983

Dr John Williamson

Numbers

Males	12
Females	3
TOTAL	15
Sports divers	14

Type of Bends

Spinal	10
Cerebral	4
Joint muscle	4
Skin	1

Experience of Divers

Hours	
Less than 100 hours	14
Range: over 1000 to less than 3 hours	
Certification	
"C" card	11
Higher	3
Student	1

Season of Year

Summer	7
Winter	8

Dive Trip Duration

1 day	3
2 days (weekend)	10
3 day	2

Dive Tables Used

Repetitive (US Navy) type	12
Unknown	2
Not classified	1

Table Compliance

Initially "inside" tables	12
Initially over tables	3
After correction for "fudge factors"	
"Inside" tables	9
Over tables	6

Depths

Deepest depth in dive profile	
average	78 feet
deepest	110 feet
Average of all 79 dives	57 feet

Bottom Times

Longest bottom times in dive profile	
average	43 minutes
longest	70 minutes
Average of all 79 dives	34 minutes

Rates of Ascent

As fast as possible	2 dives
Unknown	77 dives

Time of Appearance of Symptoms

During diving	3
First 6 hours after the last dive	8
7-24 hours after the last dive	4

Time from the onset of symptoms to seeking medical advice

Less than 6 hours	3
7-12 hours	1
13-24 hours	4
Over 24 hours	7

Factors involved which may predispose to bends

* Bad dive profile (ie. shallow then deep)	11
* Exertion during dives (retrieve anchor-3)	10
* Bounce dives	6
* Impaired health (hangover-3)	5
* Anxiety	4
* Unfit, long lay off	4
* Previous spinal injury (previous bend-2)	3
* Age over 40 years	2
* Cold during dive	2

SUMMARY OF THE DISCUSSION

Chairman: Dr Vic Callanan

Depth Gauges

It is obvious that the accuracy of gauges varies considerably with even better brands (ie. expensive) varying after use. Opinion was that only the more reliable models should be used and that they should be checked for accuracy at regular intervals.

Maximum depth indicators (MDI) are only as accurate as the gauge and may show creep of the MDI and increased needle friction. However the overall accuracy is better than that of memory, when allowance is made for human error. New technology is improving in this field.

Essential Gear

Some divers on trips are not fully equipped and divemasters arrange loan equipment where possible; but preventing a diver from diving without some item of gear is difficult. Fortunately skippers back up the divemasters.

Dive Tables

Some authorities now claim the US Navy Tables and other modifications of these repetitive dive schedules are unsafe especially at shallower depths.

Too many divers do not realise that Tables refer to the DEEPEST depth on a dive, not the average depth.

Some "C" card holders cannot use dive tables correctly.

Many divers and dive schools are now recommending and using extra safety factors, eg.

i. add 10 feet and 10 minutes to actual dives when calculating repetitive dives.

ii. decompression stops as a routine for all dives.

iii. slower ascent rates - eg. down to 25 feet per minute.

It was emphasised that divers must understand that there are no dive tables in existence which guarantee 100% freedom from risk of decompression sickness.

Dive Profiles

Many dive profiles do not follow the principle of deepest dives first and shallow dives later. It was pointed out that this is not always easy and that winds and currents may make the bottom depth under the boat greater than expected. However the principle of avoiding deep dives at the end of a trip was emphasised as being important.

Supervision of Dives

Some persons argued for:

i. divemaster logging times in and out and checking maximum depth on MDI, rather than divers doing same.

ii. fresh and fit diver and gear available at all times to free anchor, or to perform any other short notice, in-water tasks.

iii. divemasters to be more aware of the experience and capabilities of divers on a trip.

Others were of the opinion that it is the responsibility of the individual diver to log his dives. The legal position of divemasters, dive schools and skippers who undertake tuition and supervision was questioned, but no discussion ensued.

Seriousness of Decompression Sickness

The fact that bends is not just pain in the joints which can be successfully treated, was emphasised. As in most other series, spinal bends were the most common in Townsville. Once symptoms occur widespread damage may be present in several organs. Serious permanent disabilities may result. One such case is present in the Townsville series

Education

It was obviously the feeling of the meeting that better education of divers was the most effective way of reducing the incidence of decompression sickness. Points which need emphasising were suggested:

i. the seriousness of the disease

ii. the recognition of signs and symptoms

iii. how easy it is to get bends
"There is no such thing as a safe dive".

iv. correct use and understanding of dive tables.

Education was felt to be particularly important in the initial training of divers, but re-education of divers at intervals throughout their diving careers was also stressed.

Recertification

At present none of the diving schools require re-examination or recertification of holders of basic qualifications. Many agreed that a "C card" should be valid for a finite time and that re-certification should be necessary for the card to remain valid.

Update courses are available.

CONCLUSIONS

a. Trainee divers must be made more aware of the risks and seriousness of decompression sickness.

b. Divers should be educated to realise that no set of dive tables is 100% safe.

c. No depth gauge is 100% reliable, and regular calibration (every 6 months at least) is strongly advised.

d. The depth of a dive is the deepest point of the dive, no matter how brief the time spent at that depth.

e. Rates of ascent should be kept as slow as possible - never faster than 60ft/minute, 45ft/minute is better.

f. Decompression stops at 10ft(3m) should be routine.

g. Divers who cheat on tables, depths and/or bottom times are only cheating themselves and their buddies!

CLEANING UP THE SHARK PERIL

Dr Samual Gruber, of the University of Miami in Florida, has been reported as describing a new shark repellent. Tests on many products had been made and sodium docecyl sulphate (SDS), used in shampoos as a detergent, performed better than many better known natural compounds. It was better than the US Navy's "Shark Chaser" repellent, too.

SDS was discovered during tests using paradaxin, the toxin found in the Red Sea flatfish, the Moses Sole. The chemical similarity of this natural chemical to commercial detergents was noticed and fifteen such detergents were tested, the lemon shark being the test species. SDS and another detergent were found to be both consistently effective, and cheap.

So even if the repellent doesn't work you can die satisfied that you haven't wasted much money.

Reprinted from The Australian, 30 August 1983.

PUBLICATIONS OF INTEREST

THE NATURE AND TREATMENT OF HYPOTHERMIA

Robert S Pozos and Lorentz E Wittmers Jr, editors

The publisher's leaflet is reproduced below.

Hypothermia, or abnormally low body temperature, occurs both in accidental situations (exposure to cold) and in relation to such medical disorders as cardiac disease, diabetes, coma, insulin shock, drunkenness, and certain conditions in the very young and the very old. The hypothermic individual is unable to regulate body temperature, which can drop in accidental hypothermia to as low as 27 degrees Centigrade (80 degrees Fahrenheit). Medical risk is increased by heat loss, and if the victim already suffers from disease, from a blood circulation impairment - often true in the elderly - or from a physical impairment like drunkenness, the vulnerability to cold injury increases.

The 18 papers in this book, prepared for a Continuing Medical Education conference at the University of Minnesota-Duluth Medical School, provide state-of-the-art information on the diagnosis, physiology, and treatment of hypothermia. Since severe hypothermia is a major medical emergency and the victim is often found far from shelter, this book devotes primary attention to emergency and in-hospital procedures for re-warming the body, starting an arrested heart, dealing with shock, and treating cold damage to the body. Yet its comprehensive approach - its emphasis upon physiology as well as treatment - will make it a useful book for research scientists and students of medicine and human physiology as well as for physicians.

The contributors to this volume are clinical and research scientists from Canada, Great Britain, and the United States. Editors Robert Pozos and Lorentz E Wittmers Jr, are associate professors in the department of physiology, School of Medicine, University of Minnesota-Duluth.

Contents

University of Minnesota Continuing Medical Education, Volume 2. ISBN 0-8166-1154-8. xxiii + 277 pages; 53 line illustrations, 4 photographs, 1983.

Available from:
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* These publications are included in the cost of membership.

Write to:

Underwater Medical Society,
9650 Rockville Pike,
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USA

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The Physiology and Medicine of Diving Edited by PB Bennett & DH Elliott	$56.00
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The above prices do NOT include packing and postage. Further details from:

Best Publishing Company,
PO Box 1978,
San Pedro,
California, 90732.
USA

NO COMMENTS, PLEASE!

In a recent newspaper article on The Mid Life Crisis the journalist quoted a Sydney doctor specialising in sexual therapy (Dr David White) thus: "I think it's a media-induced thing over the last 10 years caused by too much leisure, too little education and the unreal expectations that have been raised by the women's movement. I'm 42. I find most men of my age seem to be falling to pieces. They are taking up physical things like scuba diving, duck farming, jogging and becoming part of what I call the walnut-sandwich-and-bar-bells set. If at 22 you've had sex, drugs and rock and roll, what's left for you at 40?"

No survey of SPUMS membership is contemplated.

BOOK REVIEW

(Reprinted from Undersea Biomedical Research)

THE PHYSIOLOGY AND MEDICINE OF DIVING

Edited by PB Bennett and DH Elliott,
Best Publishing Co.,
PO Box 1978, San Pedro, CA 90732.

Price: US$56.00 plus $3.00 postage and handling.
Australian Recommended Price $105.00
(3 to 4 weeks delay in delivery).

This new volume of what are essentially reviews of broad areas of medicine and physiology in diving and compressed air work deserves more than ever to be called a classic. It does more than bring the information contained in it up-to-date, which is the general function of a new edition. In this volume the information on practical diving has been increased and integrated more fully with background and theoretical information.

The chapter on oxygen toxicity has been completely revised from beginning to end and provides much more information on this long studied but still very much unknown subject. A new chapter in this volume considers the problem of isobaric gas exchange and supersaturation by counter diffusion, a problem relatively unknown when this book was last published but one which has aroused a great deal of interest. This edition also handles the conflicting and controversial area of decompression theory in a much better fashion. The revised chapters on recompression therapy and the management of diving accidents provide the practical guidance often missing in diving texts and provide valuable information to those who are not closely familiar with techniques or new advances in handling these problems.

This book is essential for the personal library of anyone with more than a passing interest in diving and deserves to be a standard reference in all medical libraries. It will also be of great value to engineers or other persons closely involved in diving equipment design and diving operations.

James Vorosmarti.

The Reviewer: Captai*n James Vorosmarti MD,* is the Assistant for Medical Research, Office of the Undersecretary of Defense, The Pentagon, Washington, DC.

UNDERWATER MEDICINE COURSE

The Institute of Environmental and Offshore Medicine, of the University of Aberdeen, will be running an Advanced Course in Diving Medicine, entitled "MEDICAL EMERGENCIES IN DIVING" in June 1984.

The course starts on 24 June 1984 and lasts for two weeks. A preliminary 5 day course of guided study and tutorials is available for those who have not attended an introductory course from June 18th to 22nd.

The course director is Dr TG Shields, other members of the teaching staff are Prof DH Elliott, Dr PB James and Dr NKI McIvor and guest speakers.

The fee for the main course is £1,000. The fee for the preliminary week of guided study is £250.

Enquiries should be directed to:

The Department of Underwater Medicine,
Institute of Environmental and Offshore Medicine,
University of Aberdeen,
Foresterhill,
Aberdeen AB9 2ZD.
Scotland.

HYPERBARIC MEDICINE CONFERENCES

NINTH ANNUAL CONFERENCE ON THE CLINICAL APPLICATION OF HYPERBARIC OXYGEN

11-15 JUNE 1984

Acapulco Princess Hotel, Acapulco, Mexico.

This five day conference will provide an open forum for discussion of controversial topics in the field of hyperbaric oxygen therapy through plenary talks, debates, original communications and presentation of case reports.

CONTROVERSIES TO BE DISCUSSED AND DEBATED:

- Who needs an HBO chamber
- The monoplace vs the multiplace chamber
- What should the specialist know about HBO?
- Using HBO for approved and experimental conditions.
- Who should operate the hyperbaric chamber.

OBJECTIVES

- To determine which providers of health care need hyperbaric chambers
- To know the advantages and disadvantages of the multiplace and the monoplace hyperbaric chamber.
- To learn how HBO can be an adjunct to medical and surgical care. To recognize the currently accepted uses for HBO.
- To appreciate what conditions are being treated with HBO on an experimental basis and the reasoning for such.
- To understand the rationale in determining who should operate hyperbaric chambers.
- To apply research findings from the original communications to clinical applications.
- To summarize the recommendations from the debates on controversial issues.

Programme Director: Dr George Hart.

For further details contact:
Assistant Director,
Center for Health Education,
Memorial Medical Center,
2801 Atlantic Avenue (PO Box 1428),
Long Beach, California. 90801-1428.
USA

Telephone: (213) 595-3613.

VIII INTERNATIONAL CONGRESS ON HYPERBARIC MEDICINE - 20-22 AUGUST 1984

Hyatt Regency Hotel, Long Beach, California, USA.

The conference will include original papers, exhibits, poster presentations and plenary sessions.

For further information contact:

The Secretariat,
VIII International Congress on
Hyperbaric Medicine, c/- Baromedical Department,
PO Box 1428,
Long Beach, California, 90801-1428, USA.

Telephone: (213) 595-3613.

DIVING SAFETY MEMORANDA

Department of Energy,
Petroleum Engineering Division
Thames House South,
Millbank, London, SWIP 4QJ.

DIVING SAFETY MEMORANDUM NO 4/1983 CODE OF PRACTICE ON THE SAFE USE OF ELECTRICITY UNDERWATER

11 April, 1983

The safety philosophy of the UK Diving Inspectorate is that in areas of doubt, at some time somebody has to take action in order to make progress, hence the Diving Safety Memoranda 18/1982 which drew attention to a Code on the safe use of electricity underwater.

As a result of comments received, it is clear that the actual status of the document is causing some confusion. I would like to make it clear that it is not at present designed for use in a mandatory or contractual sense. The document does contain the best information which was available to the authors at the time of publication, but it is appreciated that in some cases this was limited - hence reference to use of equipment and techniques which will be useful, but which are not currently available.

The document should be regarded as a draft for discussion and development and any comments on its scope and content would be welcomed.

It is hoped that a more authoritative version will be published in a year or so's time. In the meanwhile, all concerned with the use of electricity underwater (contractors, clients, operators, manufacturers, suppliers and others) should use the best and most suitable equipment and techniques available, consistent with safe practice, whilst at the same time taking into account all reasonably available technical advice.

Non compliance with the code in any particular respect in favour of some other reasonable practice would not be regarded as a breach by employers of their responsibilities under relevant legislation.

DIVING SAFETY MEMORANDUM NO 5/1983 TESTING OF GAUGES

6 May 1983

The Diving Operations at Work Regulations 1981 Regulation 13(1) states "The plant and equipment specified in Regulation 12(1)(2) and (5) shall not be used in any diving operation unless (c) there is in force a certificate issued under para (2) by a competent person that it has been tested and that it may be safely used". Para (2) (a)(v) states that "The period during which it can be safely used shall not exceed six months".

This regulation implies that all pressure and depth gauges fitted in the diving system must be tested every six months by a competent person.

Diving companies have complied with the above regulations by removing the gauges and sending them ashore for full calibration. This routine is expensive and requires companies to hold large stocks of gauges. Gauges can suffer damage in transit. It is suggested that the requirement can be met by testing the gauges on site against a master gauge. The master gauge should be recalibrated at intervals not exceeding six months.

The competent person may be a technician who has had training in gauge testing.

DIVING SAFETY MEMORANDUM NO 6/1983 SURFACE ORIENTED DIVING FROM DYNAMICALLY POSITIONED VESSELS

6 May 1983

The level of diving accidents and incidents in diving operations from dynamically positioned vessels is giving cause for concern and, in particular, the potential hazard arising from divers or their umbilicals becoming fouled in propellers or thrusters.

Diving Safety Memorandum No 21 of 1982 drew attention to these potential dangers. However, some companies, supervisors and divers do not appear to be following the advice provided.

Regulation 5(2)(a) of the Diving Operations at Work Regulations 1981 requires that every diving contractor, so far as is reasonably practical, ensures that each diving operation is carried out from a suitable and safe place.

Care must be taken in the selection of vessels and the personnel used for these operations to ensure that they have the necessary capabilities and are operated in a manner which meets the statutory requirements.

Proper procedures must be written into the diving rules emphasising the importance of close and efficient supervision, good diver attendance and full communication coverage at all times.

A vessel operating on DP may not always provide the necessary suitable and safe place for diving operations and other means must be considered.

If a safe diving position cannot be maintained, then diving should not take place.

The following list, which is not definitive, provides points for consideration when planning surface orientated diving from a vessel operating in the DP mode.

1. The need for good supervision.
2. The need for good diver tending.
3. The provision of efficient bridge/diving control communications.
4. The provision of efficient diving communications.
5. The use of an open bell or conventional bell.
6. The position of the diver close to the task.
7. The type of diving (ie. splash zone area, shallow, deep, etc).
8. The choice of diver umbilical (buoyant or heavy).
9. The use of cranes.
10. The use of anchors.
11. The employment of lines to the structure.
12. The possibility of demobilising the azimuth thruster, tunnel thruster or propellers in the vicinity of the diving operation.
13. The need for emergency and abort procedures.

DIVING SAFETY MEMORANDUM NO 7/1983
OIL FIRED HEATERS

8 July 1983

A recent investigation into a fire onboard a diving support vessel has shown the need for care when siting oil fired heaters and during operation. The attention of all diving contractors is drawn to the following recommendations:

1. Careful consideration is necessary when selecting a location for the installation of the heating unit and fire fighting equipment should be an integral part of that system.

2. A fireproof bulkhead should be provided between the header tank and the burner.

3. The supply of the header tank should be fitted with a valve which shuts automatically when the tank is full (Failsafe).

4. In addition to the supply, an overflow line from the header tank to a safe dump area should be fitted. The overflow pipe should be of a larger diameter than the supply.

5. Each heater unit should be standing in a suitable "spill-catch tray" and a drain provided from the tray to a safe dump area.

6. Preference should be given to hard plumbing the supply from the header tank to the burner and the same for the return pipe to the header tank.

7. A set of operation procedures, including the procedure for topping up of header tanks is to be secured at each unit. Ideally, the procedures should be under the headings of "Do's and Don'ts".

DIVING SAFETY MEMORANDUM NO 8/1983
LIFTING EQUIPMENT TESTING AND MAINTENANCE

24 August, 1983

Investigations into recent diving incidents involving diving winches has shown:-

(a) The testing of winch primary and secondary motors are sometimes carried out independently. This is not regarded as being satisfactory. The routine testing of such lifting equipment should be carried out independently and also during the transfer between the primary and secondary motors with the systems under test load. Particular attention should be given to the suitability of the brake and clutch mechanism during such changeover operations.

(b) The primary cause of a recent incident offshore was the fracture of a coupling shaft between a motor and a hydraulic pump. The initiation site of the fatigue crack was the root of the shaft keyway. Oxidisation at the face of the crack indicated that it had been in existence and growing for some time. Diving companies and equipment hirers should ensure that maintenance and inspection procedures are carried out in such a way as to minimise the likelihood of similar failures occurring in the field.

DIVING SAFETY MEMORANDUM NO 9/1983
DIVING ON PIPELINES UNDER TEST OR MAINTENANCE

24 August 1983

Diving in the vicinity of pipelines under test could be hazardous.

Divers should not approach within 100 metres of a pipeline which is under pneumatic test.

During hydrostatic testing divers may work in the vicinity of the pipeline but should not carry out any operations on it.

The Department of Energy, Submarines Pipelines' Guidance Notes, recommend when a pipeline is known or suspected of being defective, divers should not approach within 100 metres unless the pressure has first been reduced to 80% of the highest pressure to which the pipeline has been subjected since the defect was first discovered or suspected.

Additional care should be taken when the pressure in a pipeline is reduced to less than the pressure of the water surrounding the pipeline, as differential pressure is a hazard to divers.

When a pipeline is under test, the diving supervisor must discuss any operations with the owner of the pipeline or his representative before divers are permitted to work on or near the pipeline.

DIVING SAFETY MEMORANDUM NO 10/1983
DIVERS' AIR GAS SUPPLY SYSTEMS

21 October, 1983.

Diving Safety Memorandum No 14/1976 called attention to potential design faults regarding the gas supply to divers and the consequences of a primary failure.

It appeared that some principles behind these recommendations are misunderstood or misinterpreted and therefore to clarify the situation the following recommendations should be followed:-

Surface Orientated Diving

1. The air supply system to a diver should be designed in such a way that in the event of the diver's umbilical being cut or severed it should not deprive any other diver or standby diver of their air supply. It should be noted that it is impractical for the affected air supply to be isolated by manually shutting a valve.

Bell Diving

2. The gas supply system in a diving bell should be designed in such a way that in the case of the loss of the main surface to bell umbilical pressure, the emergency bell onboard gas is brought on line to the diver or divers either manually or automatically with a safeguard against exhausting back into the main umbilical.

3. The gas supply system to the bell standby diver should give him the option of using either unlimited surface or the independent limited onboard gas supply.

Divers Bail Out

4. Breathing gas supply to divers' masks must be designed in such a way that in the event of failure of the diver's umbilical supply, the gas from the reserve or bailout cylinder does not exhaust into the sea.

5. When designing new diving bells or modifying existing bells to accommodate two divers plus the standby diver consideration should be given to the provision of independent gas supply to each diver.

DIVING SAFETY MEMORANDUM NO 11/1983
DIVERS' MEDICAL AND COMPETENCY CERTIFICATES

14 November 1983

Diving companies should not accept photostat copies of divers' certificates of competency or *photostat* copies of log book entries regarding medical fitness.

DIVING SAFETY MEMORANDUM NO 2/1984

23 January 1984

The following Safety Notice has been issued by the Norwegian Petroleum Directorate and is supported by the UK PED Diving Inspectorate.

"In connection with diving accidents on the Norwegian Continental Shelf, the Norwegian Petroleum Directorate will give the following recommendations:

1. Clamping mechanisms which is necessary for the chamber complex, evacuation device and bell to remain under pressure, shall be equipped with an inter-locking mechanism which makes it impossible to open when the opening may cause an unwanted drop in pressure. We will emphasize that it shall be impossible to open the mating clamp between the bell and the chamber while the tunnel is under pressure.

2. The person who operates the clamping mechanism shall be able to ensure for himself that opening will not cause an unwanted drop in pressure.

3. The doors between the different compartments in the chamber complex shall be kept closed whenever possible.

4. All stations which are necessary for a safe diving operation shall be equipped with a communications system which enables the person on the station to understand clearly orders given to him. It shall be possible to contact the diving supervisor from all these stations.

5. The diving operation shall be carried out in accordance with laid down procedures. During operations of special importance to the divers' safety, checklists shall be used."

COMMANDER SA WARNER
Chief Inspector of Diving

COURSES IN MARINE BIOLOGY

Sea Studies Services conducts marine biology courses at various levels. The A level course assumes no previous knowledge; B, C, and D courses build on the foundations laid during the A course. The courses are run by Reg Lipson, who is a Senior Lecturer at the Hawthorn Institute of Education. He is a FAUI instructor and an examiner for the NQS Underwater Naturalist award. Those who attend the Oceans meetings in Melbourne know him as the MC of those very successful meetings.

Not all these courses are conducted in Victoria. In May 1984 there will be an overseas Marine Natural History Excursion to the Isle of Pines, New Caledonia.

Other courses are:

Course SSS/D/'84/1
THE MOLLUSCA - A DIVER'S LOOK
D Level Course Fee $95
Hawthorn Institute of Education,
9 am - 5 pm June 2 and 3.

Course SSS/D/'84/2
OUR FINNY FRIENDS - A Diver's Look at Fish.
D LEVEL. Course Fee $85
Hawthorn Institute of Education,
9 am - 5 pm September 22 plus boat diving excursion.

Course SSS/A/'84/3
Marine Biology - A FIRST COURSE*
A Level. Course Fee $105
Hawthorn Institute of Education,
7 - 9.30 pm. November 7, 14, 21, 28, December 5 and 12.
Excursion will be conducted within the above period.

For further details contact Reg or Kay Lipson.

Sea Studies Services,
70 Railway Parade South,
CHADSTONE VIC 3148.

Telephone: (03) 277-0773

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

SPUMS NOTICES

OBJECTS OF THE SOCIETY

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine.

To provide information on underwater and hyperbaric medicine.

To publish a journal.

To convene members of the Society annually at a scientific conference.

MEMBERSHIP OF SPUMS

Membership is open to medical practitioners and those engaged in research in underwater medicine and related subjects. Associate membership is open to all those, who are not medical practitioners, who are interested in the aims of the society.

At present the subscription for Full Members is $A20.00 and for Associate Members is $A15.00.

Membership entitles attendance at the Annual Scientific Conferences and receipt of the Journal.

Anyone interested in joining SPUMS should write to:

Dr Chris Acott,
Secretary of SPUMS,
Rockhampton Base Hospital,
Rockhampton QLD 4700

NOTES TO CORRESPONDENTS AND AUTHORS

Please type all correspondence, in double spacing and only on one side of the paper, and be certain to give your name and address even though they may not be for publication.

Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide these in a presentation suitable for photoreduction direct. Books, journals, notices for symposia etc., will be given consideration for notice in this journal.

REPRINTING OF ARTICLES

Permission to reprint articles from this journal will be granted on application to the Editor in the case of original contributions. Papers that are here reprinted from another (stated) source require direct application to the original publisher, this being the condition of publication in the SPUMS Journal.