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EDITORIAL

It has always been the policy of this journal to print selected papers which have been presented at conferences and meetings dealing with diving medicine, a policy readers will find fully vindicated by the contents of this issue. Subjects range from the seemingly commonplace (though always very important) ones of decompression sickness, air embolism and near-drowning, to the "frontier" of diving where HPNS is a medical and commercial problem of real importance. In almost all types of diving situations there is also the increasingly appreciated potential problem of hypothermia. All these, and other matters, receive attention, clinical reports and advice on management being "salted" on occasion by fresh viewpoints on diving problems.

With so many authors and subjects it would be invidious to seem to select some for comment at the expense of others. However the paper by Dr Hong can be noted as illustrating one approach to research. He has observed events as an outsider, a non-interventionalist philosophy, noticing the effects of changes not initiated by himself. Other researchers take a more active approach, altering the subject's environment in a controlled manner. These varied approaches can be illustrated in the matter of hypothermia associated with immersion. Dr Hong's objective was to discover whether acclimatisation to cold exposure occurred and he used observation of a work situation which was in water, utilising an unplanned change (the introduction of the wearing of wet suits), which must initially have seemed to threaten his research, to advantage. He concluded that a limited and impermanent adaptation occurred. The experimental exposure of "volunteers" to severe cold stress is a quite different matter, raising ethical problems in these times when so much is known about these matters. The "pay off" must outweigh the discomfort and risk to the subjects. Some readers may have seen the TV film of the tests on the members of a recent Antarctic Expedition. They were placed, without any significant clothing, in a bath of icy water. As there was never any real intention to send them naked into the Antarctic wastes the alternative strategy of seeking to perfect protective wear would seem to be more logical. Mankind has extended over the world not so much by adapting its physiology as by adapting its environment. The Ama have it right!

In the context of diving these three research philosophies can be best illustrated in the matter of decompression sickness. There can be strict recording of dive profiles titrated against symptoms (dependent for validity on the informed self-interest of divers having a "Road to Damascus" effect on the truthfulness of their dive records); there can be chamber testing of new schedules on a few volunteers; and there can be efforts to accept the present ignorance of divers and produce tables safe for sport divers, although it is only an optimist who would expect divers to choose a "safe" table if a "more generous" one was in print. One problem is possibly the fact that until recently diving medicine has been too black-and-white, too absolute. In the real world minor, or even alarming, symptoms may spontaneously resolve. This leads the average diver (and doctor, undoubtedly) to believe that all is well. The clinical reports in this issue show how readily symptoms of air embolism into the CNS can be ignored if they are of short duration. This leads to a quite unjustified belief in the safety of certain diving actions, like very rapid ascents. Few realise that such emboli can dislodge later and result in secondary symptoms. Luckily, it would seem that many probable embolism producing events fail to register their presence clinically. But not every bubble lands in a "silent" area.

It is becoming steadily more certain that there remains great scope for improvement in our understanding of the rules nature has set for truly safe diving. It behoves us all to pay our dues for the privilege of being among the first to move freely in the marvellous world of the sea. Our dues are the recording and truthful reporting of anything and everything untoward which occurs in relation to diving and hyperbaric work. Our contributors are showing the way.

SPUMS-RAN MEETING AT HMAS PENGUIN
27.8.83

THE HIGH PRESSURE NERVOUS SYNDROME REVISITED

Ralph Brauer

As my contribution to this discussion, I would like to take you on a brief guided tour around central nervous system effects of pressures of the order of magnitude we encounter in deep diving.

To begin with, then, allow me to tell you that all vertebrates and, indeed, very many invertebrates that make their living at or near the surface of the ocean show evidence of profound changes in central nervous system functioning when transferred over a reasonable period of time to environments in which hydrostatic pressures are somewhere between 50-150 atmospheres absolute (ATA). Among vertebrates specifically, the two symptoms accompanying this change which are most readily observed are tremors and convulsions. Many years ago, we showed that if one assembles something like a zoological garden of representative vertebrates and one determines under given compression conditions the pressures at which each of these two symptoms develop, the series of data points that result when one plots the two types of pressures against each other for each species fall quite close to a single straight line. This very simple fact is of interest because, while we have seen tremors in man, we have not so far encountered anywhere in the world divers sustaining clear-cut high pressure convulsions. Knowing the tremor threshold pressure, however, and having at our disposal the linear relation between tremor and convulsion threshold pressure for many vertebrate species, we can infer with reasonable accuracy the point at which man (who after all is another vertebrate) might be expected to undergo high pressure convulsions under the same compression conditions.

The overall range of these phenomena is defined by the fact that the most sensitive animals show quite well defined high pressure tremors when exposed to about 20 ATA, while the most resistant ones seem to undergo convulsions when they are exposed to roughly 150 ATA. Multiply each of these values by ten and you have a range from 200-1500m over which symptoms of the high pressure neurologic syndrome can be observed, taking surface dwelling vertebrates as a group. Within that range, furthermore, since human tremors develop at pressures somewhere in the neighbourhood of 25-30 ATA, we can predict with some reasonable assurance that we might expect the mean threshold pressure for high pressure convulsions in man will be little higher than 70 ATA with an uncertainty of approximately ±10 atmospheres given, however, a compression rate which is substantially higher than those in current use. If we factor in the compression effect, the mean threshold is likely to fall around 100 ATA but given a standard deviation for such values of the order of ±10 ATA, this means that we must expect that about 5% of all people will have HPNS convulsions by about 75 or 80 ATA even at the slowest compression rates. You will recognize that regardless of how these figures are analysed they indicate that in the range of depths which are currently of interest for deep diving in support of such things as oil exploration, high pressure effects on the central nervous system can be expected to be of very serious significance.

I think it is fair to tell you that as of to date we really know terribly little about the biophysical mechanisms which underlie these phenomena. I think in a sense it is true for these phenomena, as it is for deep sea biology in general, that our problem is not so much a scarcity of possible effects, but quite on the contrary a plethora of such effects. Almost any process going on in living tissues and contributing to excitability is subject to some degree to modification by hydrostatic pressures of the order of magnitude that we are discussing here. Thus the real problem is to try to segregate out from among these the one, or the ones, that are of real importance. I think most physiologists who are in this business share the suspicion that a very major role in these events should be played by characteristics of the excitable cell membrane, and among these fluidity changes and phase changes of the lipoprotein membrane system are viewed with special suspicion.

Now, to give you only one example of the type of frustration from which we are suffering, let me tell you that there is quite good experimental evidence indicating that melting points of such membranes, which in turn presumably are closely linked to the ease of re-orienting the all important ion channels embedded in the membrane, are indeed affected by pressure: the magnitude of the shift to be expected is of the order of 4° for every 100 ATA. On the other hand, among many other data of this type, let me merely quote to you observations on very young mice which have not yet developed the machinery for actively defending their temperature in high pressure environments and whose deep body temperatures, therefore, can be safely assumed to be very close to those of their environments. In these animals, one can experimentally test the inference that change in hydrostatic pressure should elicit changes in the properties of those very membranes which in turn could be potentiated or reversed by manipulating the temperatures under which the compressions are carried out. The regrettable fact is that when the experiment is performed and one measures the mean pressures required to produce convulsions in these baby mice over a temperature range of approximately 8°C there is absolutely no change in convulsion threshold. Clearly, this observation will make it quite difficult to entertain the hypothesis that change in membrane fluidity can indeed constitute the principal point of attack giving rise to development of the high pressure syndrome. Indeed, despite 20 years of efforts in quite a number of excellent laboratories, we are still largely in the dark as to the real mechanisms brought into play to modify brain function in animals exposed to high pressure environments.

By and large, such neurophysiologic changes as are observed would lead us to predict that in high pressures animals ought to calm down and go to sleep, rather than to undergo exaggerated activity and show locomotor changes culminating in these vicious seizures.

In our ramble around HPNS, I would like to take you next briefly to another corner of the forest where interaction of

these phenomena with oxygen supply to the tissues is under review. This subject came up very early when some people, especially those who wished that the whole matter which seemed to limit our ability to penetrate to great depths would go away, suggested that what was being observed was merely a phenomenon secondary to the presumed respiratory difficulties that "must" attend breathing such dense atmospheres, with the actual seizures representing merely a special case of either hypoxic or hypercapnic seizures. That particular debate was probably resolved to a large extent 10-12 years ago when it became quite clear that sensitivity of animals to high pressure convulsions was quite independent of gas density, and quite unaffected by modification of oxygen content of those atmospheres over a range from roughly 0.2 to roughly 1.5 ATA. That conclusion was supported further by blood gas analyses which showed no changes, and by experiments with simulated high density atmospheres, the effects of which were clearly dominated by the narcotic effects of the heavier gases utilized rather than by impairment of respiratory exchange. Indeed, those experiments that involved manipulation of oxygen partial pressures in atmospheres which otherwise included only helium suggested that, in the high pressure environment increase of oxygen partial pressure beyond 1.5 ATA facilitated the onset of convulsions. More recently, we have come back to that particular line of inquiry and have found that if the relation between oxygen partial pressure and convulsion onset is investigated in greater detail two phenomena become evident. In the first place, we find that the convulsion stage of HPNS is not simple, but consists of at least two distinct events, one of which is potentiated by high pressure oxygen, while the other seems to be quite insensitive to it. These data are merely a special case of a more general observation that indeed the two stages of the HPNS convulsive phase which we call Type I and Type II seizures, behave as though they were two quite distinct entities, showing different genetics, different dependence upon compression rates, different response to drugs including narcotically effective gases, and different courses of development in very young animals.

All round, these particular phenomena are one of the examples where the study of underwater physiology has provided substantial and significant input to general medical physiology, in this case documenting for the first time the coexistence of two such quite distinct seizure events. Similar patterns had been suspected in other conditions including epilepsy, but it had never been possible to reproduce them at will and to discriminate them with the clarity with which this is possible in this situation.

Confining our attention now for the moment to the Type I seizure, the one that develops soonest, we found that if we modified oxygen partial pressures through a range from 0.2 to 10 ATA in the presence of various amounts of helium, three quite different patterns of interaction can be observed. There is a zone in which, as the total pressure is increased by helium additions to values up to about 20 ATA, the curves relating oxygen partial pressure to time of onset of convulsions retain essentially the same hyperbolic shade characteristic of uncomplicated hyperoxic convulsions, but displaced to shorter and shorter time intervals as the helium pressure is increased. We feel that this range is best described as the area in which oxygen convulsions are potentiated in the presence of high pressure helium. As the total pressure is further increased the character of this curve changes progressively, and develops a very dramatic plateau between helium pressures of 35 to 50 ATA at all oxygen partial pressure between 1.8 and 3 ATA. This behaviour strongly suggests that in this case we are observing high pressure convulsions potentiated by hyperoxia. Finally, if total pressures are increased further, time to convulsion onset decreases below those required for development of oxygen toxicity and it becomes apparent that we are now looking at high pressure convulsions which are virtually unaffected by oxygen partial pressure.

At the next point, I would like to briefly turn to a problem I touched upon briefly earlier, namely the effect of compression rate upon the onset of the severe stages of HPNS. Quite early in the game, we showed that high pressure convulsions develop at lower total pressures when the compression is rapid than when it is slow. More detailed analysis of this kind of experiment allows us to gain some insight into the scope of this phenomenon. In the case of mice, where we have the most complete data, convulsion threshold pressures at the very slowest compression rates that are practicable, requiring days to reach the threshold and corresponding to months long compressions for man, the maximum seizure threshold that can be attained is in the neighbourhood of 120 ATA. Indeed, there is little difference between compression rates of 1/5 of an atm/hr and of four or five atm/hr for this species. At the other extreme, at compression rates of 1000 atm/hr, picked originally because they bore some relation to the compression rates required in connection with some of the submarine free escape techniques, the same mice have convulsion threshold pressures in the neighbourhood of 75 ATA. The maximum scope of this phenomenon in the case of the mouse, therefore, is of the order of a 60-70% increase in convulsion threshold pressure. Here again, it is worth noting that the figures I have just given you apply to the Type I seizures.

Type II seizures are much less affected by compression rate, and it is possible that the limiting convulsion threshold pressure for very slow compression rates corresponds to the point of intersection between the compression rate dependence curve for Type I and Type II convulsion threshold pressures. Indeed, in the mouse at very slow compressions an increasing proportion of the animals show a seizure type that is intermediate between the clonic Type I and the hyperextension and rigidity of the tonic Type II seizure. Apart from its neurophysiological interest this observation serves to bring into focus a peculiarity of HPNS that seems to me not unimportant, namely, that as one takes measures to alleviate the severity of HPNS at a given depth and uses these measures to penetrate to ever greater depths, the clinical characteristics of the entity change, and more specifically they tend to change in a direction in which the presumed protective measure suppresses the warning symptoms, frequently without suppressing or postponing measurably the most alarming and potentially the most lethal components of this entity.

Here again, I am afraid that we do not have a really satisfactory analysis of the mechanisms which underlie the

compression rate effect. In the case of the mouse, I think one can make a reasonable case for the hypothesis that to an important extent this entity involves activation of central monoamine neurotransmitter pathways which are known to antagonize the development of convulsions in general. Suppression of the action of these neurotransmitters tends to abolish the compression rate effect in the mouse and in a number of other species. While we are far from understanding these events fully, I think one can make an excellent case for the hypothesis that we are looking at a complex event, the time dependence of which reflects its dual character. A largely time-independent effect of hydrostatic pressure on central neurones, is modulated by time-dependent events which appear to be triggered by the high pressure exposure and which tend to postpone or to lessen the severity of the locomotor effects consequent upon modification of central nervous system function by high pressure.

This brings me to the next brief stopover on our excursion, namely to what we are terming "acclimation phenomena". We came to these observations originally via the back door, through studies of interaction between high pressure effects and inert gases that I shall discuss briefly in the last section. Here, I will merely tell you that there is an antagonism between the effects of these two types of agents. When it was shown some years ago that mice can be acclimated to the anaesthetic effect of gases like nitrous oxide by prolonged exposure to sub-anaesthetic levels, we became interested in testing the possibility that such acclimation might entail a change in susceptibility to hydrostatic pressure. Experiments confirmed this suspicion dramatically. Animals that had been exposed for a week or so to half an atmosphere of nitrous oxide, and then transferred to a heliox environment underwent high pressure convulsions at pressures little more than half as high as those of control animals. That being the case, we became interested in the possibility that the reverse phenomenon might also be observable. We were able to show that this is indeed the case. Mice that have been exposed for four to five days to 80 ATA of heliox, when decompressed and recompressed to determine convulsion threshold pressures, undergo their convulsions at pressures which are 30-40 ATA higher than those of normal control animals under the same circumstances. This type of effect is dissipated relatively rapidly, and we wondered whether it might prove to be merely a special instance of activation of the monoamine type of protection we just discussed. However, the pattern of compression rate dependence of convulsion thresholds of such animals does not support this hypothesis. Pressure acclimated mice show exactly the same compression rate effects as non-acclimated ones. Thus, pressure acclimation must represent a more general type either of biochemical or electrophysiological change in the brain.

I might mention that this phenomenon, once again, is not merely a matter of concern to underwater physiologists, but has a rather broader interest. As I have told you, surface dwelling vertebrates in general, including fish, undergo high pressure convulsions at pressures which never exceed 150 ATA. Yet, as we all know, there are many fish that inhabit depths of the ocean corresponding to much greater depths. Working with abyssal fish from Lake Baikal in Siberia, we have been able to show that there is a rough relation between observed convulsion thresholds and capture depths which bespeaks either acclimation or adaptation to the high pressure environment. The fact that pressure tolerance of these abyssal fish does not decrease rapidly with exposure to one or a few atmospheres suggest that, in part at least, these fish must have invoked an adaptational mechanism different from the acclimation one that we have described so far. A theoretical basis for such adaptation exists. We have been able to show that in mice variations in pressure tolerance are to a very large extent under genetic control. Indeed the genetic mechanisms are much simpler and more direct than only a few years ago people would have thought possible for so-called quantitative genetic effects. Thus, it seems likely that the genetic machinery can exist also in fish upon which evolutionary mechanisms could act to increase the pressure tolerance so as to yield pressure tolerant deep sea species. As regards mechanisms that might account for this type of adaptation, the most appealing hypothesis is that these might involve changes in brain composition, and in particular perhaps of the ganglioside component which is so strongly concentrated in the synaptic areas of these tissues and which has been shown to be involved in low temperature acclimation of fishes.

I would like, finally, to turn briefly to the problem of interaction of high pressure effects with those of pharmacologically active inert gases. We have recently had the opportunity to undertake a fairly detailed review focussed on an attempt to separate the effects of pressure as such from the pharmacologic effects of helium and hydrogen in particular, and to a lesser extent of nitrogen and nitrous oxide, though these are much more potent narcotics and relatively much less affected by the effects of "pressure as such". That review showed that for the four effects for which sufficiently detailed data are available for such an analysis in excitable tissue or excitable tissue models there was a common pattern. In each case, hydrostatic pressure effects were antagonized to some degree by the so-called "inert gas", and in each case also the potency with which this effect was exerted was least for helium and increased in the order neon/hydrogen/nitrogen/nitrous oxide. At the same time, it became obvious that the balance of these effects was by no means uniform. Thus the "neutral" member of the series, ie. the gas in which a particular hydrostatic pressure effect associated with the total pressure of the gas was just counterbalanced by the pharmacologic, antagonistic, effect of that same gas, varied within the series almost all the way up and down from hydrogen or (rather a gas intermediate between neon and hydrogen) for anaesthesia, or reversal of anaesthesia, to a gas with properties intermediate between nitrogen and nitrous oxide for the phenomena of phase reversal in lipid bilayer models. These phenomena once again are of interest not only from the point of view of the biophysics of high pressure effects, but also from the point of view of diving physiology. They clearly establish the fact that the once hoped for "neutral gas mix" which would "abolish HPNS" simply does not exist, for the reasons that HPNS itself is a complex, not a simple, entity and that the different pressure effects which are responsible for this compound character show widely different sensitivities to the antagonistic effects exerted by the various inert gases.

That same inquiry turned up yet another previously unexpected phenomenon, namely the existence of numerous other effects of hydrostatic pressures which fall altogether outside the pattern of antagonism discussed so far. Here hydrostatic pressure effects either were not antagonized but even potentiated in the presence of pharmacologically active inert gas components. Here again the order of potentiation in producing effects often bore no recognizable relation to the order of potencies characteristic of the excitable tissue phenomena we have discussed so far. Phenomena in this category include such things as cell death, development of cell pathology, and changes in cell replication. I think it is probably not terribly surprising that a group of effects of this type should exist which may well reflect some of the many changes in cell functioning that can be brought about by high hydrostatic pressures without involving excitable cell membranes, or for that matter cell membranes at all as a primary target.

From a practical point of view, the existence of this group of pressure effects suggests the possibility that when we use addition of narcotically active gases to diving mixtures to minimize some of the manifestations of HPNS, in addition to the complexities of modification of the clinical picture by uneven action of these agents, we may be producing additional problems. By creating such conditions we may succeed in exposing our subjects to quite high hydrostatic pressures by suppressing the acute manifestations of HPNS while significant other pressure effects may be exerted upon our subjects which may not be relieved to the same extent, or which may even be exaggerated, by these modifications of our diving atmospheres. This, then, could confront us with the possibility of pressure-induced injury that might not become manifest until sometime after the dive is completed. Since we are dealing with human beings at risk, I think it is appropriate to recognize this possibility, and in future studies directed toward medical problems of extremely deep diving, we must include work designed to probe for, and if possible to dissipate, any such residual injury problems at the level of animal experiments rather than risking possibly painful surprises from the ultimate effects of what must be termed human experiments.

I hasten to add to this that at the present time such dangers constitute no more than a purely theoretical possibility. I would be inclined to question the validity of suggestions that some of the behaviour changes that have been reported in subjects undergoing very deep dives can be interpreted as valid evidence for residual changes of any kind resulting from such dives, although surely they do not allow us to discount that possibility. At the level of animal experiments, we have conducted experiments which involve repeated exposures of animals and which, in the obliging way nature has with such things, yielded equivocal data that would be equally compatible with an affirmative and a negative conclusion in this matter. We have currently underway what we hope will be more sensitive experiments utilizing behavioural criteria and quantifiable memory performance, but the results of those experiments will not be in for another year, and even then I would suspect that they can hardly furnish more than suggestive evidence tending to sway us one way or another.

I have tried to give you some feeling for the lines of investigation of high pressure effects on the central nervous system which have engaged our attention over the last several years. I hope I have conveyed to you three ideas.

The fact that these phenomena are real, and that coping with them is one of the prerequisites for further development of deep diving.

Some sense of the types of real and probable hazards these phenomena impose, and some concept of kinds of working hypotheses we currently entertain as to the biophysical mechanisms underlying them.

Finally, a sense of the fact that in addition to their immediate bearing on problems of deep diving these phenomena are characteristic of many problems in underwater physiology in that they bear upon and provide opportunities for studies of a wide range of problems in basic physiology and in particular in basic adaptive biology.

I have tried to give you some feeling for the lines of investigation of high pressure effects on the central nervous system which have engaged our attention over the last several years. I hope I have conveyed to you three ideas.

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COLD ADAPTATION IN HUMANS
A LESSON FROM KOREAN WOMEN DIVERS

Suk Ki Hong

I would like to express my thanks to the RAN for inviting me and giving me this opportunity to present some of the experimental results I have obtained over the past 20 years from studies on Korean women divers.

Professor Brauer has described very interesting effects of pressure which is an important environmental variable to diving individuals. There is another very important aspect of diving, body heat exchange in water. To dive, you have to go into water, stay there and pressurize.

I started my research on the physiology of Korean women divers exactly 24 years ago. At the beginning we were concerned with the respiratory physiology of breathhold diving, particularly the effects of pressure. However, it soon became apparent to us that the real problem with Korean women divers was not respiratory physiology but thermal physiology. This is because the water temperature is about 22°C in the middle of summer, which is relatively warm but still cool. In the middle of winter, the water temperature decreases to 10°C.

In that water temperature range these women are exposed to a severe cold stress. To overcome that they should dress

very heavily. However we observed that they wore just a thin cotton suit, which does not give much protection against cold water stress. Therefore we undertook an extensive series of studies. In fact, we spent the next 10 years documenting the quantitative pattern of heat exchange as well as the nature of cold adaptation.

To begin with we simply measured the rectal temperature during one work shift (45 min) in summer, when the water temperature is about 22°C. We found that the rectal temperature dropped slowly over a 45 minute period to about 35°C, at which time they quit diving because they could no longer tolerate the cold stress. At the same time, the skin temperature decreased to 27°C. Thus the mean body temperature decreased by about 5°C over the 45 minute period. In winter, when the water temperature is down to 10°C, the rectal temperature had dropped to about 35°C in about 30 minutes, at which time they again quit diving. In other words there is a critical rectal temperature (approximately 35°C) below which human beings cannot voluntarily tolerate the cold stress.

We then estimate the amount of body heat lost to the sea water during one shift. In summer, the cumulative heat production, determined from the increment in oxygen consumption, was about 94 Kcal, while the magnitude of heat lost from storage was about 293 Kcal. Thus, the total heat loss amounted to 387 Kcal in 45 minutes. Since they did about 3 shifts a day in summer about 1000 Kcal of body heat were lost daily. In Winter they lost about 575 Kcal in one shift, again with a daily heat loss of about 1000 Kcal.

The question arises whether, if they keep losing that much heat every day all year round, they adapt to cold? As of 1962 or so, there were debates among human physiologists whether human beings did indeed develop adaptations to cold. To be sure, adaptations to cold in other animals have been proven beyond any doubt, but many physiologists doubted that this was the case in human beings. We felt that we had a golden opportunity to study whether humans did indeed adapt to cold. So we started a series of studies to find out if there were any adaptations.

To make a long story short, I will briefly summarize the results of ten years of work. We found four major adaptive changes to cold in these women.

1. They had a reversible increase in basal metabolism during the cold season by about 3O%.

2. They had a greater body tissue insulation, by about 10%, compared to normal subjects.

3. The cold-induced constriction of the finger blood vessel during immersion of one hand in 6°C water was much greater in divers than in non-divers. Greater vasoconstriction means less blood flow so less heat loss. Blood flow was 30% lower in divers than in non-divers.

4. The critical water temperature (the water temperature which a subject can tolerate for three hours without shivering) was also about 10% lower in divers, indicating the higher shivering threshold in divers.

There are a few other adaptive phenomena, but these major adaptive changes are very similar to those found in animals. In other words Homo Sap*iens is no differ*ent from other animals.

When we published these results another question was raised, “How do you know that these changes are due to cold?” I had no real answer to this question because I did not have the data to show that these changes can be reversed by eliminating cold water stress.

Fortunately, when I visited Korea in 1977, I found that the diving women who used to be very lightly dressed now all wear wet-suits. They told me that they wore them to keep warm. So, I felt that if I had a chance to get back to these women divers again and demonstrate the absence of cold water stress accompanied by disappearance of all the adaptive changes I had observed before, this would enable me to reinforce the earlier conclusions. So we went back to these women divers about four years ago and repeated everything we did in the olden days. I am greatly indebted to both Drs DW Rennie (State University of New York at Buffalo) and YS Park (Kosin Medical College in Korea) who collaborated with me in this study.

What are the effects of wearing wet-suits on body heat loss during diving work in the sea? In summer (water temperature 22.5°C), the heat loss in the 60’s was 380 Kcal in one shift of 45 minutes. Contemporary wet-suited divers do one shift lasting 2 hours, during which they lost about 300 Kcal. Similarly, in winter (10°C water) the heat loss in the olden days was 580 Kcal in one 30 minute shift. Contemporary divers can stay in the water for 2 hours during which they lost only about 400 Kcal. This means that indeed, by wearing wet-suits, they have eliminated a tremendous cold water stress to which they had been subjected daily prior to wearing wet-suits.

When we measured their basal metabolism in 1960 and 1961, we found that there was no difference between divers and non-divers in summer. However, in the middle of winter, the divers’ basal metabolism went up by 30% over the summer level in a reversible manner. When we repeated the same measurements in 1980 and 1981, there was no difference between divers and non-divers whatever the season. This means that a metabolic adaptation (ie. the reversible increase in basal metabolism) to cold no longer exists in divers using wet suits. If we had measured the basal metabolism in 1978 or 1979, we might have observed a partial disappearance of this type of adaptation. However, it took three years to write up the grant application and get the funds. By then the metabolic adaptation had disappeared completely.

Critical water temperature is a function of the skin fat thickness, which provides a barrier for heat loss. In 1961, we found that non-divers with fat thickness of 3-4 mm had a critical water temperature of around 32°C. However divers who had a similar skin fat thickness had a critical water temperature of only about 28°C. In other words, there was a marked difference in shivering threshold or critical water temperature in the olden days. When we went back to them in 1980, we found that they were all fatter. The skin fatness had increased in both divers and

non-divers. We compared the critical water temperature as a function of the skin fat thickness in these subjects. Overall there was an inverse relationship between the two variables as expected. However, we observed that the critical water temperature for a given subcutaneous fat thickness was lower in divers than in non-divers by about 2°C in 1980 and about 1°C in 1981, but the same in 1982. There had been a progressive, gradual loss of this adaptation over a period of three years.

The maximum tissue insulation (for a given subcutaneous fat thickness) in the olden days was consistently higher in divers than in non-divers. However, in 1980, we found that they were identical in both groups. In fact, in all three years we found no difference between the two groups, indicating that the insulative adaptation had already disappeared after wearing wet-suits for three years.

The finger blood flow response to immersion of one hand in 6°C water was then investigated. The finger blood flow before immersion was about 45 ml per minute per 100 ml of finger volume. When we immersed one hand in 6°C water, the flow dropped immediately and then increased (cold-induced vasodilation), followed by the so-called "hunting phenomenon". In the 1960's, we found that the women divers showed a greater vasoconstriction and a reduced hunting phenomenon than non-divers. When we repeated this test in 1980, we found that this peripheral vascular adaptation was already partially lost. By 1981, this adaptation had disappeared completely.

Our findings may be summarized as follows. Our early studies (conducted from 1960 through 1970) indicated at least four types of cold adaptation in women divers, a reversible increase (30%) in basal metabolism in winter, a 10% increase in tissue insulation, a 30% reduction in the finger blood flow response to immersion of one hand in 6°C water, and a 10% reduction in the critical water temperature. When we repeated the same studies in 1980 after the divers had been wearing wet-suits for three years, the basal metabolism and insulation returned to normal while the peripheral vascular response and the critical water temperature partially returned to the non-divers' level. In 1981, after wearing wet-suits for four years, we found that the finger blood flow response was back to normal, but the critical water temperature still remained lower for divers than for non-divers. It took five years to lose all the cold adaptation changes they had acquired when diving without wet-suits.

These studies have shown that it is quite certain that these women divers were indeed acclimatized to cold at one stage. In other words, our studies lend strong support to the notion that Homo sapiens can indeed develop adaptation to cold.

Question:
What about age? The divers would be getting older every year.

Dr SK Hong
I did not make it clear. We did not use the same subjects in every study. But our non-divers were all age matched to the divers and the results obtained from them were much the same in the old and new series.

Question:
The end point for the traditional diver was a rectal temperature of 35°C. The divers in the wet-suits have a longer diving time. Is their endpoint also the same as in cotton suited divers?

Dr SK Hong
This is a very important point. They stay in the water much longer, but their rectal temperature at the end of two hours in the water has hardly changed. There is a 0.2-0.3°C drop in the rectal temperature over a two hour period. The question then comes up, why do they not stay longer? They do not wear gloves and the hands get awfully cold. Also they get physically tired.

Question:
Do they wear hoods?

Dr SK Hong
Some people do, others do not. About half of them wear them.

Question:
Any difference between the ones that do and the ones that do not?

Dr SK Hong
We did not find any difference.

Question:
The original Ama divers, did they come from specific tribes or specific areas or families?

Dr SK Hong
No. Usually, they dive if their mothers do. When there are many women divers in a community, the young kids, at the age of about 11 or 12, just jump into the water and start practicing shallow dives. After 4 or 5 years of training they become professional divers. It really takes many years of training to become an independent diver.

Question:
It is almost similar to what John Pennefather mentioned earlier that it is natural selection.

Dr SK Hong
It is dependent on training and ability. There is nothing to indicate that there are other factors involved.

Question:
Are they still breathhold diving, or do they use compressed air?

Dr SK Hong
They are still breathhold divers. The union specifically prohibits scuba diving, because it would wipe out the resource.

Question:
Present day Ama are fatter than their mothers. This would increase their insulation.

Dr SK Hong
So we had to make comparisons for a given skin fat thickness. They were very skinny in 1960's. I think this reflected, to some extent, the poor economy.

Question:
The Ama divers are female. Is that because they adapt better, or because they are pushed into it?

Dr SK Hong
My theory is that they are there because they can tolerate cold water better than males. Because, if you look at history, both male and female divers were once involved but gradually male divers disappeared from the scene. However, in Japan, where they dive only during the warm season, you find both male and female divers. So there is nothing magic about the absence of male divers in Korea where they do dive all year round, they have to tolerate the cold stress and have to compete with women, and there is no way that they can win.

Question:
Why are present day Ama fatter?

Dr SK Hong
Nutrition accounts for the increase in the amount of body fat in contemporary divers. But remember, our data are based on a given skin fat thickness, so we have ruled out skin fat as a variable.

Question:
Did the effect of the wet-suit increase their breathhold capacity?

Dr SK Hong
We do not know. Breathhold time is longer in divers than in non-divers, but not much. Breathholding time is not an issue, because usually they do not breathhold for long. They hold their breath for a diving time of about 30 to 40 seconds, and we have reasons why they do that. To get the maximum bottom time and to increase the fraction of time they spend on the bottom, shorter and shallow dives are better.

Question:
When you say that breathholding is a matter of training, is it partly a personal thing, or is it endless practice that makes them able to breathhold just that bit longer? Do they go down on a shot line and get there quickly, or do they have to swim down?

Dr SK Hong
I think it does require skill. They have to learn to swim and to dive from shallow to deeper and deeper depths, and they have to learn all the tricks.

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IMAGINARY CONSULTATION

"MY STUPID EARS"

Noel Roydhouse

In a letter recently published in an Underwater Association newsletter a reader asked for their Medical Adviser's advice on solving his problem. It was a problem which troubles many divers, "Ears". I have set down the t*houghts* which would have occurred to me had he **told his tale** during a consultation concerning his problem to draw attention to the problem of misdirection of attention if the patient's diagnosis is uncritically accepted.

"I have a diving problem, my ears. I have a history of sinus problems." *Prior to 1920 this would have been "nasal catarrh" but owing to a world wide tendency for ENT Surgeons to operate on normal, non-infected sinuses in the 1920's, folk medicine changed this to "sinus trouble", as the patients underwent thousands of operations upon their normal sinuses. The reason they improved temporarily is well documented by the Immunologists but basically the shock of the operation gave three to six month cure, after which they relapsed and their catarrh was often worse. By the 1940's such operations were given up but folk medicine "sinus" problems have incorrectly and mistakenly persisted.* "At the age of thirteen I had my tonsils removed." *Probably unnecessarily so, as in my 34 years of taking out children's tonsils the age group of ten to fifteen years rarely need tonsillectomy.* "And ever since then I have been plagued by sinus." *Nasal catarrh really, and it is stuffiness or blockage of the nose with or without a mucous or thin discharge from the nose, sometimes with a "pressure" in the nose or under the eyes.*

"I noticed when snorkelling that I was unable to equalise pressure as readily as before the tonsillectomy." *No wonder. "Sinus" is often stress induced and the unnecessary operation of tonsillectomy would be enough to bring on nasal catarrh.* **"Over the years the problem has got steadily worse."** *I would guess four to five years - see later.* "Now I've only got my stupid ears." *He is getting emotionally involved with his problem and this adds to the stress and makes him worse again.* "Wet in the bath, and I've got earache." *I have published several papers on this[1,2] and he fits the typical case described. Appendix 1 is an abstract of Reference 2.*

"I have all but completed the divers' course necessary to get a certificate but was unable to pass the medical due to blocked Eustachian tubes." *Named after Bartolomea Eustachi, an Italian Anatomist (1520-1574) who first described this tube which is also known as the Internal Auditory tube.* "After a half hour dive to about 30 feet." *Diving to 30 feet meant that he had cleared his ears or he would have ruptured his ear drums. Diving, in itself, does not cause any permanent blockage of the Eustachian tube. If one does not clear one's ears on descent the water pressure compresses the inner soft tissue end of the Eustachian tube and at two metres unless equalisation has occurred the Eustachian tube is locked. This means that the chest muscles cannot produce enough pressure to open the Eustachian tubes as they cannot raise the pressure*

behind a blocked nose to this water pressure. "I had prolonged earache for about six weeks afterwards." *This is due to the Mandibular Dysfunction Syndrome.*[3]

"One of the two ENT Specialists I saw ..." *He obviously shopped around and was probably told by the first one that there was nothing wrong with his nose or sinuses. As he had already had his tonsils out I suspect he had been circumcised and had his appendix out and came from a surgically minded family*[4] *and was all set for another ENT operation. All surgeons are trained to operate!* "...performed a right anterior" *where else do you do it! He probably means intra-nasal.* "Antrostomy to dry and relieve the sinus." *This calls for prolonged comment. Apart from the intra-nasal antrostomy being a useless operation harking back to the witch doctoring of the 1920's, the diver produced no evidence that it was ever indicated. If purulent sinusitis, for which antrostomy is indicated, was present, and he was having trouble with both ears, one would assume that the operation would have been done on both sides if at all. Also, the vast majority of ENT specialists would have washed out his sinuses first to confirm that there was some infection present before embarking upon the antrostomy operation. The operation is not indicated to "dry" the sinuses but to provide immediate free drainage of pus from the antrum. He could have misinterpreted the reason or have been misguided. Anyway, he states that both sides of his nose are less blocked, further illustrating the "shock" or immunological effect of operating on normal sinuses.*

"While I admit that side is freer" *Does he mean more nasal discharge, a complication rather than a benefit.* "to the extent that my nose is seldom blocked, my ears always feel under pressure." *It is about time he had his impacted wisdom teeth removed, or he should give up grinding and clenching his teeth and give up biting his mouthpiece so hard, as the Mandibular Dysfunction Syndrome is about the only condition which can produce this pressure and the prolonged earache (six weeks) that he suffered previously.*

My advice to this diver would be to suggest cautery of his inferior turbinates, counselling on the techniques of clearing the ears, and to have his wisdom teeth removed or, if these were not troubling him, to do jaw muscle stretching exercises and give up worrying, or at least to give up grinding and clenching his teeth and biting his mouthpiece. Do not get me wrong but patients can strew the path with red herrings or even lobsters. To get at the bottom of a diver's problem can be difficult as he has not had a medical training which includes a new vocabulary of about 14,000 words that a medical student picks up in his first 6 years. The doctor has to act as his own interpreter.

REFERENCES

1. Roydhouse N. Earaches and the adolescent swimmers. In: Eriksson B and Fuberg B (eds). *Swimming Medicine IV*. Baltimore: University Park Press, 1978; 79-84.

2. Roydhouse N. Earache and adolescent swimmers. *NZ J Sports Med*. 1977; 5(2): 15-18.

3. Roydhouse N. The jaw and scuba diving. *J Otolaryn Soc Australia*. 1977; 4,2: 162-165.

4. Miller FJW, Court SDM, Walton WS, Knox EG. *Growing up in Newcastle Upon Tyne*. London, Nuffield Foundation: 1960; 21, 224.

APPENDIX I

Roydhouse N. Earache and adolescent swimmers. *NZ J Sports Med*. 1977; 5(2): 15-18.

"Swimmers, both surface and underwater, seem to suffer from earache more commonly than the average sports person. Because many doctors, including Ear, Nose and Throat Surgeons, regard water sports as the reason for the earache they tell their patients to give up swimming. Accordingly the case histories of twelve adolescents who suffered earache and were swimmers were examined. In all cases it was shown that the primary causative pathology was not in the ear and that immersion in the water was not the prime cause of the trouble. The prime cause was erupting or impacted molar teeth, teeth grinding and psychological upset, with the exposure to the cold water as the precipitating factor. Prevention is to put up with the problem until the molar teeth have erupted or to have the appropriate dental treatment. Reassurance after explanation with the cessation of grinding and clenching the teeth was often all that was needed."

COMMENTS FROM MELBOURNE
John Knight

Dr Roydhouse, in his entertaining and instructive Imaginary Consultation states that unless equalisation has occurred by the time the diver has descended to 2m the Eustachian tubes are blocked, or locked shut, by water pressure compressing the inner soft tissue end of the Eustachian tube, and that the chest muscles cannot generate sufficient pressure to overcome the external pressure. This would be true for a snorkeller, but is it true for a Scuba diver, whose chest and pharynx are at ambient pressure? In this case the pressure to be overcome is either negative, if the diver is descending feet first, or at most 0.5m if he is descending vertically head first, and these relativities will not change with depth.

Many scuba divers "clear" their ears to the extent that discomfort goes, but do they restore the normal middle ear volume? Judging from those I see they do not, as they have evidence of barotrauma. Oxygen is taken up from the middle ear by the tissues lining the middle ear, leading to a drop in middle ear pressure. Normal pressure is restored every time the Eustachian tube opens and air travels up it. The normal openers of the Eustachian tubes are movements of the pharyngeal muscles. Divers do not suck a dummy, which would move their pharyngeal muscles, they have their teeth biting into a regulator. They seldom talk, swallow or otherwise move their pharyngeal muscles. They usually have to make an effort to get air up their

Eustachian tubes. Many fail to maintain the middle ear volume normal and accept mild discomfort throughout their dive.

With a higher than normal PO2 in the middle ear and a normal tissue PO2, we would expect more rapid uptake of oxygen during diving, and unless the diver "clears" his ears at intervals an almost constant negative pressure in the middle ear. It has been shown that a negative pressure of 6.6 cm of sea water for 5 minutes in the middle ear of a guinea pig results in fluid exuding from the swollen middle ear lining.[1] A negative pressure of 13.2 cm of sea water results in a bloody fluid after 5 minutes. A diver who stays at 30 feet for 30 minutes without clearing his ears would, if human ears behave like guinea pig ears, be very likely to have fluid in his ears. That fluid may well have difficulty escaping down the Eustachian tube as the swelling of the middle ear lining will include the proximal part of the Eustachian tube, and this swelling, while developing in minutes, takes hours to disappear, which can explain uncomfortable ears after diving

I wholeheartedly agree with Dr Roydhouse that interpretation of the diver's story by discovering what he means by "sinusitis" and other misused words is essential for proper diagnosis and treatment of diver's disorders.

I would like to thank Dr Roydhouse for introducing me to the Mandibular Dysfunction Syndrome which now explains to me the symptomatology of a patient I saw last year.

REFERENCES

1. Hiraide F and Ericksson H. The effects of the vacuum on vascular permeability of the middle ear. *Acta Otolaryngol*. 1978; 85: 10-16.

THEY DIDN'T MEAN TO DO IT, BUT IT'S DEADLY ALL THE SAME

Twenty tonnes of Dinoseb, a deadly herbicide, were lost overboard from a freighter during a storm in the North Sea early in January, along with 80,000 bottles of whisky. The 80 drums will, hopefully, survive for a sufficient time to allow their localisation by a search ship, and their careful recovery. Should this operation fail there is danger that the leaking of their contents will kill all life on the Dogger Bank, a vital spawning ground for fish, and possibly be fatal to seamen if they come into any contact with it. It is lethal even through the skin. The crew of the freighter did not immediately notice the loss of the lethal cargo, and continued foul weather prevented an immediate attempt to recover the drums.

It is believed that the drums lie in 54 metres of water and special computers, television robots and sonar search gear is being employed.

It is a pity drums containing such deadly poisons are not more securely transported.

DIVING EMERGENCIES IN REMOTE SITUATIONS TWO CASE REPORTS

Based on reports made to the PROJECT STICKYBEAK Non-fatal Incidents file

Those who go diving in areas remote from easy access to centres equipped to provide definite care for decompression sickness (DCS) or cerebral air embolism (AE) must include a contingency plan for the management of such events. Serious incidents can arise even in groups of apparently experienced and trained divers and the quality of the immediate response will be critical to the ultimate outcome, as is demonstrated by these two cases.

Case 1

The victim had been diving on hookah at 60 ft for 50 minutes, working vigorously when the hose became kinked and he was forced to make an emergency ascent. He was able to exhale correctly during his ascent but was not able to make any decompression stops. About 90 minutes after surfacing he developed a discomfort in his chest, "a feeling of bubbles moving". A little later he had a similar feeling in his left elbow and became aware of paraesthesiae in both calves. His legs felt very heavy. He felt very weary and almost unable to walk after going up some stairs.

He was driven to the nearest hospital which was 4 hours away. There he reported paraesthesiae in both arms and the backs of both legs, also that his legs were very heavy, and that he felt unwell and very fatigued. The nearest diving medicine consultant was contacted by telephone and the hospital was advised that this was a case of DCS with spinal involvement. He was given 100% oxygen, aspirin and copious fluids while arrangements were made to fly him in a pressurised aircraft to the recompression facilities. Delays were experienced arranging this transport but the treatment given improved his condition somewhat. However there was some relapse during the flight.

Initial treatment was by Table 62 (RAN), which resolved the symptoms within half an hour. Following this he was given 100% oxgygen alternate hours overnight. There was total clinical recovery. However he was advised against further diving using compressed air because the response to the initial insult had been much greater than the gas burden of the dive profile would seem to predict, and because of the probability that, even after a clinical cure, residual cord damage remains.

Case 2

The victim, a healthy trained diver aged 35, was swimming at 20 ft through a reef channel to escape the worst of the rough water. His buddy was leading and only realised the victim's absence on arriving at their destination. He swam back and found the victim's weight belt. Other members of the party had observed the victim surface, without his mask, swim sluggishly for a few strokes, then roll onto his back and attempt to ditch his scuba set before becoming unconscious. He was rapidly pulled from the water and found to have had a cardiac arrest, with dilated pupils and white froth coming from his mouth.

Resuscitation was commenced by a medical member of the group and the pupils responded quickly. It took 25 minutes of resuscitation, including intravenous adrenalin, before independent heart and respiratory activity were regained.

He remained unconscious, though making spontaneous movements.. There appeared to be some inability to use one arm in involuntary movements for the first two hours, but this apparently recovered. The patient remained irritable and made restless movements but did not seem conscious of his surroundings.

He was taken by boat to harbour as attempts to air-lift him with a helicopter were thwarted by a combination of communication problems, extreme range and sea conditions. He was transferred to an RAN patrol boat at sea which brought him to shore more rapidly than would have been possible on the dive boat. He was still very irritable and totally unconscious when carried ashore, though moving and responding to stimuli. He was transported breathing 100% oxygen by Royal Flying Doctor Service pressurized aircraft to the nearest recompression chamber where he was recompressed, initially on Table 6, thereafter to 6A with maximum extensions.

Great difficulty was experienced in controlling the patient, who was totally irrational and uncoordinated. Little improvement occurred during this recompression treatment. At the end his behaviour was such that he could occasionally drink and obey simple commands, but he objected to being handled and was even biting the attendants. He had received some intravenous fluids and steriods but because of his violence it was impossible to maintain an IV.

He was maintained on 100% oxygen and transferred to the nearest major recompression facility by pressurised aircraft the next day. There he was assessed and received further therapeutic recompression, but without making significant improvement. Over the next few days he received repeated recompression to 9 metres, with adjunctive therapy. Improvement was slow and did not appear to be related to treatment.

The patient was finally transferred to a neurological recovery unit, and three months later is making an excellent recovery. It is expected that he will be able to resume his work, which requires the accurate use of his mind, six months from the episode.

The history suggests that this patient sustained an acute air embolism, probably as a result of uncontrolled ascent, following some sort of underwater crisis. Cardiac arrest resulted. Resuscitation was effectively carried out, but due to the extreme remoteness of the site there was an interval of 26 hours before the patient was first recompressed. Oxygen was only available for a portion of that time. The final picture was that of global brain ischaemia, probably due to hypoxia associated with cardiac arrest. He is making the expected slow progressive recovery from this major insult.

NEW ZEALAND DIVING-RELATED FATALITIES 1981-82

Douglas Walker

Four breath-hold (snorkel using) divers and eleven scuba divers have been identified as dying in New Zealand waters while diving during 1981-1982. Five had been diving alone and all the remainder were to some degree separated from their companion(s) during the critical time, though in case SC 6 this separation was minimal and non-contributory to the fatal outcome of the victim's "heart attack". One victim was an epileptic with a history of survival from previous in-water attacks. Three victims had a history of asthma, but in none was this of proven significance. Cold water, rough seas, aspiration of vomit and possible nitrogen narcosis, were other factors noted. The available records were, in general, unhelpful in establishing the training and experience levels of the victims and rarely noted the diving skills of the other divers present. Police reports were available concerning the two cases where no Inquest was thought to be necessary.

CASE NOTES

Case BH 1

The victim was diving for paua and kina from a beach, the sea being too rough for the intended boat dive. The victim and his companion were diving independently of each other while a third person remained ashore. He saw the victim surface and became alarmed when there was no plume of water to indicate the snorkel was being cleared. He immediately entered the water, but had to return to the beach to better identify the victim's position before he could find him and bring him back to the beach. Resuscitation efforts were ineffective. The victim was an epileptic on medication and with a history of previous in-water fits from which he had recovered without ill effects. He was wearing shorts, shirt, jersey, sandshoes, mask and snorkel.

EPILEPSY. ALONE. SANDSHOES. NO FINS.

Case BH 2

Three friends went diving, two deciding to spearfish about 20 ft away from where the victim was to dive for paua. They last saw him alive as he sat on a rock, apparently about to re-enter the water. About 45 minutes later one of the spearfishermen came across the victim floating at the surface, face down, minus mask and snorkel. The water was cold and the victim had worn a shorty wet-suit for protection. Attempts at resuscitation were unsuccessful. They had had "a hard night out" the previous evening but blood alcohol was absent on test. Similarly, the history that the victim previously suffered asthma attacks appears to lack significance. As his diving skills and the water depth are alike unknown the possibility of hyperventilation as a factor cannot be evaluated.

SEPARATION. COLD(?). POST LATE-NIGHT FATIGUE(?). FOUND FLOATING.

Case BH 3

After a morning spent breath-hold diving the victim mentioned over his lunch that he felt seasick. Despite this

he insisted on resuming diving. He was later seen floating unconscious at the surface. He was brought ashore but failed to respond to resuscitative efforts. It was suggested as probable that he aspirated some vomit, then drowned. There was a history of mild asthma but nothing to suggest that it was in any way contributory to the tragedy.
SEASICKNESS. SEPARATION. ASPIRATION OF VOMIT.

Case BH 4
Diving in a 6 feet deep sea beach lagoon for kinoa, the two divers were unequally successful. The buddy handed his full bag to his companion, taking his in exchange. He told him to take it back to the shore while he obtained a few more kinoa. The water was calm but cold and the victim had only the protection of a wet-suit jacket. A short time later the buddy came across the other's snorkel floating on the surface, then noticed the victim floating face down at the surface about 20 feet away. He brought him ashore and attempted resuscitation, but without success.
SEPARATION. SURFACE. COLD. WET-SUIT JACKET ONLY. ALCOHOL. UNKNOWN EXPERIENCE.

Case SC 1
There was rough surf but the two men decided to kit up and try for some crayfish while their wives remained where they had just finished their picnic lunch. They swam out to just beyond the line of the breakers, where the leader made a short dive. He surfaced before the other made his descent and proposed that they abort the dive and return to the shore. They were swimming on the surface about 20 yards apart and finding it difficult to make progress in the rough surf when the buddy, by now in a mild panic state, noticed that the leader was seemingly resting with his compensator inflated and making no progress. He managed to reach him and found that he was lying on his back with his snorkel in his mouth but with its end underwater. He managed somehow to tow him to the beach where the two women helped him to get the victim ashore. Resuscitation attempts were commenced when they were beyond the surf, but were unsuccessful. The victim was an experienced diver but a tendency to get seasick in the water.
FAILURE TO RECOGNISE DANGEROUS SEA CONDITIONS. SEA SICKNESS. ASPIRATION OF VOMIT. SEPARATION. SURFACE. COMPENSATOR INFLATED. IMPERFECT REGULATOR PRODUCED WATER SPRAY ON INHALATION. VALIANT BUDDY ASSISTANCE.

Case SC 2
The victim and his friend attempted to hire two scuba sets to go spearfishing but the sports store manager would only sanction the hire of a single tank and regulator, for the use of the diver he knew to have been under instruction at one time. He gave strict orders that the scuba was not to be loaned to anyone else and made a point of stating that the contents gauge read high and needed to be discounted by 300 psi. He also briefly reviewed diving procedures. The two friends then went to the beach where the buddy scuba dived (alone!) and the other snorkelled. After a time they left the water and the buddy was persuaded to allow his friend to use the scuba in the still, clear waters of a nearby 15 foot deep rock pool. The victim wore a wet suit and all necessary equipment and seemed to be managing well so the buddy decided to collect his spear gun and place it for safety in his nearby car, lest it be stolen. While at his car he heard his friend cry out, so raced back to the pool. There he found the victim floating face up at the surface, weight belt off and compensator inflated. Resuscitation attempts were unsuccessful. Investigation showed that the tank was empty though the gauge showed remaining air, as predicted. It is not known whether the victim had used scuba previously, as he had implied, but he was certainly untrained. He had a history of asthma and the pathologist reported signs of asthma reaction in the lungs, but no evidence of pulmonary barotrauma.
POOL SITUATION. UNTRAINED. INEXPERIENCED. OUT OF AIR. BORROWED/HIRED EQUIPMENT, USING ANOTHER'S AUTHORITY. ASTHMA BRONCHIAL CHANGES. KNOWN INACCURATE GAUGE. ALONE. VEST INFLATED. WEIGHT BELT DROPPED. BUDDY IGNORED WARNINGS ABOUT LOANING.

Case SC 3
After their first dive the victim told his buddy that he felt tired, but after a 15-20 minute rest in the boat declared himself ready for another dive. About 10 minutes into this dive the buddy suddenly realised that he was alone among the kelp, so surfaced. He found that the people who had remained in the boat had seen the victim floating at the surface and retrieved him. The sea was cold and choppy and his tank was empty. Autopsy revealed the presence of severe coronary artery disease, though there is no record of any check of his previous health record. It is supposed that despite having a contents gauge he ran out of air and made an emergency ascent, the stress/panic causing a fatal heart failure. He was untrained and inexperienced, though possibly the owner of the scuba he used (the point was not checked).
UNTRAINED. INEXPERIENCED. SEPARATED. OUT OF AIR. IGNORED CONTENTS GAUGE READING. ARTERIOSCLEROTIC (L) CORONARY ARTERY. FOUND FLOATING. COLD, CHOPPY WATER.

Case SC 4
This diver was last seen just before he dived near a buoy, 50 yards from the beach, when he waved to a person ashore. He is said to have been trained and experienced and in good health. When he failed to surface after an hour a search was commenced. His body was found near where he was last seen, the weight belt undone but retained by the lower strap of his compensator, which was (incorrectly) over it. The compensator was partly inflated but lacked the buoyancy necessary to surface the weighted body. There was adequate remaining air in the tank so the reason for the tragedy is unknown.
ALONE. WEIGHT BELT INCORRECTLY PLACED AND TRAPPED BY BELT OF PARTLY INFLATED COMPENSATOR. EXCESS ALCOHOL.

Case SC 5
The victim was a fisherman, trained but very inexperienced, who was diving alone to repair the shackles of a mooring. He was using borrowed scuba, though had a set of his own, and left a person in his boat when he dived with strict instructions to summon help if he failed to surface after 20 minutes. It is not known whether or not he regarded this as

NEW ZEALAND DIVING FATALITIES 1981 and 1982

CASE	AGE	SKILL VICTIM	SKILL BUDDY	DIVE GROUP	DIVE BASE	WATER DEPTH DIVE	WATER DEPTH INCIDENT	WT ?	BELT WT	CONT. GAUGE	BUOY VEST	EQUIP TEST	REMAIN AIR	EQUIP. OWNER	WET SUIT	COMMENTS
BH 1	23	T N/S E some	N/S N/S	2 sepn	beach	N/S	surface	no	N/A	N/A	no	N/A	N/A	own	no	Epileptic. Diving for paua, kinoa. Found floating. Separation. No fins.
BH 2	22	T N/S E N/S	N/S Expd	3 sepn	beach	N/S	surface	no	N/A	N/A	no	N/A	N/A	own	short	Cold. Diving for paua. Separation. Found floating. Asthma history.
BH 3	65	T N/S E N/S	N/A	alone	beach	N/S	surface	no	N/A	N/A	no	N/A	N/A	own	yes	No Inquest. Alone. Seasick. Aspiration vomit. Mild asthmatic.
BH 4	37	T N/S E expd?	N/S	2 sepn	beach	6ft	surface	no	N/A	N/A	no	N/A	N/A	own	jacket	Cold water. Diving for kinoa. Alcohol. Separation. Surface with full bag. Found floating.
SC 1	39	T N/S E expd	N/S N/S	2 sepn	beach	N/S	surface	N/S	N/S	yes	infl.	yes	full	own	yes	Rough sea. Aborted dive. Found floating. Aspiration of vomit. Valiant buddy. Minor regulator defect. Seasick
SC 2	33	T N/S E N/S	part some	2 sepn	rocks	15ft	N/S	drop	N/S	yes	infl	yes	empty	Hired borrowed	yes	Alone. Rock pool. Found floating. Inaccurate gauge. Asthmatic. Called out. Inflated compensator.
SC 3	31	T no E inexp.	yes expd	2 sepn	boat	35ft	N/S	N/S	N/S	yes	no	yes	empty	??	yes	Underwater separation. Found floating. Out of Out of air. Coronary artery disease.
SC 4	31	T ?? E ??	N/A	alone	beach	3Oft	N/S	tangled	N/S	N/S	part infl	yes	1/2 full	own ?	N/S	Inadequate information
SC 5	31	T yes E inexp.	N/A	alone	boat	20ft	ascent	on 221b		yes	no	yes	1/2+ full	borrowed had own	yes	Borrowed tank, had own. Sudden surface without mask. Ditched tank, not weights. Inexperienced. Probable AE.
SC 6	49	T N/S E N/S	N/S N/S	2 sepn	boat	60ft surface		off	N/S	yes	part infl	no	low	own	yes	No inquest. Heart attack after surfaced.
SC 7	23	T N/S E Expd?	N/S N/S	4 sepn	beach	N/S	surface	off (buddy)	N/S	N/S	no	no	(full)	own	yes	Rough sea. Aborted dive. Group surface separation. Buddy valiant help. Loss consciousness.
SC 8	26	T N/S E N/S	N/A	alone	boat	N/S	N/S	lost	N/S	Equipment not found				own	yes	Inadequate information.
SC 9	34	T yes E inexp.	N/A	alone	shore	N/S	N/S	lost	N/S	yes	not lnfl.	yes	empty	own	yes	Strange delay starting search. Found floating. Rough sea No snorkel. Inexperienced
SC 10	34	T yes E expd.	N/S expd.	2 sepn	boat	180 ft	ascent 15Oft	off (buddy)	N/S	yes	not infl.	yes	3/4 full	own	Jacket	Unexplained ascent. Separation. Unconscious before surfaced.
SC 11	27	T N/S E N/S	N/S N/S	2 sepn	boat	N/S	surface	on	N/S	no	yes	yes	1/2 full	own	yes	Surfaced in the path of a speedboat. In boat channel. No flag. Vest defective. Continued dive after separation.

KEY T = successful competion scuba course. E = reported degree of experience. N/S = Not stated. N/A Not atpplicable

a safety measure. He was seen to surface momentarily 5 minutes after starting his dive, then not seen further. When the 20 minutes had expired the alarm was raised and a search organised. His body was found on the seabed, minus mask and scuba unit but still wearing the weight belt. The tank was still nearly full. As the body was brought to the surface some regurgitation of a milk-shake taken shortly before the dive occurred, but no evidence of aspiration of gastric contents was noted at the autopsy. The story is typical of a panic ascent (cause unknown) with cerebral air embolism and consequent drowning, though no pulmonary barotrauma was noted by the pathologist.
INEXPERIENCED. ALONE. DITCHED TANK. RETAINED WEIGHT BELT. ADEQUATE AIR.

Case SC 6
Careful diving practices were observed until the two divers surfaced and then decided to use up their remaining air beneath their boat, and the lapse, in that the buddy surfaced alone and got into the boat while his friend remained submerged, was probably not relevant to the outcome. The victim surfaced and handed his weight belt, mask and gloves into the boat, then suddenly clutched his chest and floated away. The buddy immediately jumped into the water and attempted to give EAR, but found it impossible and therefore (with great difficulty) got the victim into the boat. His resuscitation attempts were unsuccessful. Autopsy revealed evidence of previous myocardial damage and this death appears to have been due to a 'heart attack". His health history is not known.
HEART ATTACK. PREVIOUS HEART ATTACK. BUDDY VALIANT RESCUE ATTEMPT. DIFFICULTY GETTING VICTIM INTO BOAT.

Case SC 7
A group of four scuba divers swam out through the breakers using their snorkels in order to conserve their air. It is not known whether they had their air turned on. After swimming out about 100m the leader decided to abort the dive because of the rough sea conditions and they all started to swim back towards the beach, becoming scattered somewhat at this time. One of the party noticed the dive leader was floating at the surface about 40 feet distant, so swam to him. He then ditched the tank and weight belt of both himself and the victim in order to improve their buoyancy, for neither wore a compensator. In-water EAR was attempted before starting to tow the victim to the beach. Another of the group assisted with this towing. The victim was stated to be experienced but no facts are on record concerning training or experience of the divers involved.
SEVERE ADVERSE SEA CONDITIONS. GROUP SURFACE SWIM SEPARATION. NO COMPENSATOR. FAILURE TO USE SCUBA AIR. DROWNED. EXPERIENCE UNKNOWN.

Case SC 8
Very little is known about this incident (and background data will be welcome and useful) beyond the surprising facts that although his boat was discovered the day following his presumed date of death no alarm was raised about his absence until the fifth day. The body was washed up, minus all diving equipment, eight days after presumed death date. Nothing is recorded about his diving habits, experience, equipment or motivation.
ALONE. UNSTATED EXPERIENCE, TRAINING, DIVING MODE (SCUBA ASSUMED). UNEXPLAINED DELAY AT REPORTING MISSING. EQUIPMENT NOT RECOVERED.

Case SC 9
Two weeks after completing his basic scuba course, still very inexperienced, the victim made a solo dive to spearfish in rough seas. He left his snorkel in his car on the beach. About 4 1/2 hours later his body was found floating, his tank empty and compensator uninflated. Resuscitation was attempted, but unsuccessful. He had spent the previous evening at a late party but no alcohol was found in his blood. The weight belt had been dropped.
TRAINED. GROSS INEXPERIENCE. ROUGH SEA. ALONE. NO SNORKEL. OUT OF AIR. COMPENSATOR NOT INFLATED. DROPPED WEIGHT BELT.

Case SC 10
No explanation can be offered for the events of this incident, though nitrogen narcosis may have been the critical factor. There were about 14 divers on this boat dive trip and the buddy system of diving was being followed. He was trained and experienced, though it is not known whether he had deep-diving experience. Another buddy pair accompanied them to the pre-arranged depth of 180 feet, where the victim gave the signal for the ascent to commence. His buddy looked away for a moment to look at a rock formation, turning back to see the victim furiously finning upwards. He managed to catch up with him at about 130 feet, as he stopped his flippering and seemed to be resting there. Then he was seen to start to keel over backwards and to hold his arms out side ways from his body, the hands starting to shake. He fell back, landing on his back 20 feet lower, on a rock ledge. Initially his buddy thought the victim was fooling around but soon realised that he was in trouble. He ditched the victim's weight belt, held him upright, and part-inflated his own compensator to aid ascent. The demand valve remained in the victim's mouth and was seen to be bubbling air till about 10 feet from the surface. He was unconscious when surfaced. The dive boat was nearby and he was speedily taken on board. He did not respond to resuscitation attempts. No fault was found in the equipment and there was adequate remaining air. The first wild ascent, reason unknown, possibly resulted in an air embolism.
DEPTH. POSSIBLE NITROGEN NARCOSIS. UNEXPLAINED PANIC ASCENT. AIR EMBOLISM UNDERWATER (?). VALIANT BUDDY RESPONSE.

Case SC 11
Tragedy ended this outing by two couples. They took a boat and the two men scuba dived, then snorkelled, before they moved to anchor off a small island at the mouth of a bay, there to have lunch. Between them and the nearby rocky headland there was a marked boat channel. After their meal they decided to dive again, but became separated immediately they entered the water because of the poor visibility. About 10 minutes later one diver surfaced alongside the boat, and very shortly after this the other was seen to surface in the boat channel almost immediately in front of a boat towing a water skiier. There was inadequate time for evasive action by either party and the diver was hit by the hull and possibly the propeller of the boat, sinking immediately. The buddy and one of those in the speedboat made an immediate search but failed to locate the body, which was recovered by a formal search later. Death was due to drowning after being critically injured.

It was admitted that those in the speedboat were in no way at fault, because they had kept as far from the moored boat as the channel allowed. No "divers down" flag was being flown because it had been torn the previous day and

awaited repair. The victim's equipment was checked and the tank found to contain adequate air: there was no imperative need for an ascent while a speedboat was obviously nearby. It was noted that he was wearing a defective compensator, the oral inflation tube having been torn off sometime previously and placed in the vest's pocket and the CO2 cylinder was empty. Neither diver had followed accepted teaching of immediate surfacing when separated from their buddy.

SEPARATION. POOR VISIBILITY. SURFACED DISTANT FROM DIVE BOAT. NO FLAG. DEFECTIVE COMPENSATOR. EXPERIENCE NOT STATED. DIVED NEAR MARKED BOAT CHANNEL. TRAUMA FROM SPEEDBOAT.

DISCUSSION

The most immediately singular finding in this short series has been the fact that nine of the victims were found floating, dead, as the first indication that anything untoward had occurred. In a further three cases the divers were diving alone, the bodies being recovered too late for any chance of reviving them. Of the remainder, one suffered a fatal "heart attack", another surfaced in the path of a speedboat and the third seemingly suffered an underwater pulmonary barotrauma with air embolism. Such events are more readily avoided than successfully managed. As no evidence is in the records concerning the previous health of the "cardiac" divers it is not possible to comment on whether they should have been diving.

The breath-hold fatalities seem to be representative of deaths in other years associated with seeking paua, kinoa, and other shellfish. In many such cases it is uncertain whether the victim was using mask and snorkel or without such aids. That a known epileptic with a history of having "turns" while diving should be allowed to continue to dive alone demonstrates either fatalism or a surprising belief in providence. Cold water was a possible factor in two cases, while alcohol, inhalation of vomit and fatigue following a previous "late night" were additional adverse factors noted. The history of asthma in three cases is of doubtful significance but will undoubtedly be thought to be of importance by some. All victims were alone at the critical time, though this was minimal and non-contributory to the outcome in case SC6, and in SC10 the victim's action created the separation.

During the two years here considered there were eleven Scuba diving deaths in New Zealand, which contrasts adversely with the twelve which occurred throughout the entire Australian seaboard in the same period. Although the available information is far from complete it is probable that the single most critical factor was inexperience, which gives hope that the toll can be reduced through education of the diving population. Case SC2 is a terrible reminder of the ease and rapidity with which a seemingly safe situation can progress irreversibly to a fatal conclusion. A calm, relatively shallow pool would seem to be a totally safe diving situation, but in the absence of knowledge and experience it became a death trap, the sensation of restricted air supply not being recognised as an urgent warning before the out-of-air situation was complete. The dive shop manager had done everything in his power to avoid such an eventuality, leaving aside the question as to whether the hirer was sufficiently trained to be allowed to dive unsupervised (let alone alone). NEVER LOAN TO AN UNTRAINED PERSON!

Contents gauges were used on the equipment of seven of the eleven scuba divers, not used by one, and the situation is unknown in three cases. In three cases the user ran out of air, a known faulty reading gauge being a possible excuse in one, inexperience and lack of a snorkel in another.

The wearing of "compensators", type usually unstated, was noted in seven of the scuba deaths and none of the breath-hold ones. Full inflation of a compensator does not guarantee survival if there are sufficient adverse factors (cases SC1, 2, 11) but can be expected to favour survival. Diving with a non-functioning compensator has nothing to commend it.

It is noted that here, as in most previous such surveys, there are usually a number of adverse factors as well as the "trigger event". It is for this reason that diving instruction should always firmly inculcate into pupils the ongoing need to follow the guidelines for safe diving at all times. Do not pit yourself against the sea's power, dive with a buddy, surface with a useful reserve of air (ie. look at your gauge), have an efficient "compensator" (for choice inflated from a compressed air cylinder), and accept that fatigue, cold and narcosis are real dangers requiring respect. Naturally epilepsy, coronary artery disease (and asthma) should be absent factors. And fly the Diving Flag.

ACKNOWLEDGMENTS

This report could not have been prepared without the active and vital assistance of the Water Safety Council of New Zealand, whose recording of drownings was the source of identification of the cases. To Mr MJ Hetherington in particular thanks are due for his obtaining of copies of the Inquest documentation, and to the NZUA thanks for access to their information. The willing assistance of the New Zealand Department of Justice has also been invaluable. The comments are not to be taken as other than the author's own. Readers are free to come to their own conclusions on the evidence available.

DIVING SAFETY INVESTIGATION

Diving safety is improved by learning from the misadventures, both great and small, of divers. Much of value can be learnt from consideration of even seemingly minor problems experienced during dives. New Zealand readers are asked to make reports of such matters to the NZUA. Diving Incidents Scheme. Australian (and other) readers are asked to send reports to:

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AUSTRALIA

ANZICS

ROCKHAMPTON MEETING OCTOBER 1983

We are grateful to the Queensland Regional Committee of the Australian and New Zealand Intensive Care Society for permission to publish papers which were presented at their inaugural annual meeting held in Rockhampton. The Guest speakers were Dr Struan K Sutherland of the Commonwealth Serum Laboratories and Dr John Knight of SPUMS. Two sessions were devoted to envenomation and two to underwater medicine. Transcripts of most of the papers given during the first two sessions appear below.

MANAGEMENT OF SNAKE BITE IN AUSTRALIA

Struan K Sutherland

Introduction

The important thing about snake bite and indeed all our venomous creatures is that man being stung or bitten is an accident. By and large all the venoms and all the important animal toxins are to paralyse prey so that, for example, the snake does not have to fight with a marsupial rat. It can bite it and that creature will be paralysed and helpless within a minute, sometimes in seconds, and it can then be eaten. If man gets in the way it is an accident and the same toxins can have the same serious effect.

Fundamentals

Unfortunate things sometimes happen in managing a snake bite because people do not really consider the fundamentals. It is very unusual to have two identical cases because of the large number of variables. The first variable is the creature that bites. It may be a highly poisonous creature or it maybe one that only produces a small amount of weak venom. Secondly, the dose of venoms will vary enormously. The amount of venom that the snake or spider puts in may be zero or it may be a massive amount.

This brings me to a very important point. The presence of fang marks does not mean that a significant amount of venom has gone in. We must try and break the reflex action, "Look fang marks. We must give antivenom", because with a snake bite, generally speaking, only one in ten cases becomes seriously ill.

The site of the bite will determine to some degree the rate of absorption of the venom as will the health of the person, the blood supply and lymph drainage of the area. Then one has to take the size of the patient into consideration. Obviously a small child bitten by a large snake will have more venom per kilogram of body weight and will become more seriously ill than would a very healthy 20 stone man receiving the same amount of venom.

Another danger that sometimes occurs is that people think that when giving antivenom to a child, the child must need less antivenom than an adult. It is just the opposite. Because of the child's lower body weight, the child might need a lot of antivenom to neutralise the relatively larger dose of venom. Antivenom is the only specific antidote to any of our snake toxins. There are no pharmacological agents that will significantly reverse the effects of venom. So if there is antivenom available and the person is significantly poisoned, antivenom must be given.

How much antivenom should one give? One really cannot tell before treatment. If one does not give antivenom when it is indicated, the venom may win and kill the patient. The amount of antivenom has to be balanced. There must always be an excess of antivenom. Envenoming is a very dynamic business. A snake bite may produce a severe coagulation defect. If one takes a sample from the patient when he or she comes through the Emergency Department and the result of that investigation comes to the ward an hour or so later and the coagulation profile may be normal. That really does not mean anything because that was what was happening an hour ago in Casualty. In the meantime considerable venom has been absorbed and there may be a very severe coagulation defect. So the dynamic aspect of envenoming must be considered all the time. This is terribly important when a patient is transferred from one hospital to another or from the Emergency Department to the Intensive Care Unit. Unless someone stays with that patient all the time, the new doctors are quite often unaware of the subtle changes that have occurred.

Most venoms are specifically designed to paralyse and they do it in a phenomenally wide range of fashions. Snake venoms are a little bit different in the fact that, apart from having toxins that will cause paralysis, they have a lot of other active components as well. For example, Tiger snake venom and Taipan venom have at least 18 separate components. Some of these components cause severe clotting disturbances. This allows you to distinguish between snake bite and some other envenomations. A combination of paralysis and coagulopathy must be due to snake bite. It cannot be spider bite or any other bite.

If you want to take a deep breath, a nerve impulse starts from your brain, goes down the nerve and releases a transmitter substance at the end of the nerve and the muscle will then contract. That is putting it in its simplest fashion. Anything which can stop that message getting down and causing the muscle to contract will cause paralysis. There are many ways this is done. Snake venom does it at several sites. For example, both Tiger snake and Taipan venom have three separate neuro-toxins. Two hit post-synaptically acting on the acetylcholine receptors and work pretty quickly. The third is a much bigger, presynaptic, toxin which works very slowly. It gets into the membrane at the end of the nerve and causes steady changes. The dramatic new finding of recent years is the realisation that this big neurotoxin also attacks skeletal muscle. About seven or eight of our most important snakes all have this component which will cause rhabdomyolysis, so in delayed cases there is rhabdomyolysis leading to renal failure from blockage of renal tubules with myoglobin. On the other hand a certain number of our snakes do not have this. Brown snake and Death Adder do not cause rhabdomyolysis. The clinical differences between different types of snake bite are now being established. Hence the importance of venom detection kits.

Snakes

We can be very proud of our snakes in Australia. We have the most impressive collection in the world. As one goes north the variety, size, and shall we say deadliness of our snakes steadily increases. Tasmania has got a pretty nasty Tiger snake which is fairly sluggish but that State needs only one antivenom. In Victoria there are also Brown snakes, Copperhead snakes and the Red-Bellied Black snake. We require two antivenoms in Victoria. Once across the border, there are broadly five distinct and extremely dangerous species of snakes all requiring their own individual antivenom. By the time one gets to Queensland there is quite a collection of very, very special snakes, including the three most dangerous snakes in the world.

Snake Identification

Only one in ten cases of snake bite is the snake correctly identified. Sometimes it is mistakenly identified even by the so-called experts. The patient may then be given the wrong antivenom and may die. The problem of identification of snakes has been solved to some degree by venom detection kits. In reality many people do not see the snake that bit them or they are bitten at night or they were walking through grass and they just see the tail disappearing. They may be children who cannot identify the snake. Some of our common snakes are quite confusing in their appearances. There is not much point knowing the difference between the Brown snake and a Tiger snake when the difference can only be determined by looking at the anal scales. If you have just been bitten by a snake, the last thing you are going to do is get on the ground and examine its fundamental orifice. The Tiger snake is the commonest cause of serious snake bite because it is found around the more heavily inhabited areas of Victoria and New South Wales. The standard Mark I banded Tiger snakes are reasonably easy to identify. However these snakes are often unbanded. If one saw such a snake and did not know snakes one might call it a Brown snake, and that really would not help the patient.

The common Brown snake is the second most important cause of snake bite. Its venom is the second most potent of all the snakes in the world. When they are young, they often have stripes for the first eighteen months. So when a child says it was bitten by a snake with stripes the doctor might dismiss Brown snake envenomation.

The Red-Bellied Black snake is the least dangerous of these snakes. Generally speaking, one holds back antivenom if someone is bitten by a Red-Bellied Black snake.

The Copperhead is the only venomous Australian snake found above the snow line.

The Death Adder which has a highly neurotoxic venom, is a bit different to our other snakes. Most snakes will get out of the way if they are awake and sense people approaching. Death Adders do not, they stay perfectly still.

Our heaviest snake is the King Brown which causes occasional deaths amongst aborigines.

The Taipan is our longest and most venomous snake. Very few survived an effective bite by a Taipan before antivenom was available, at least 8 out of 10 died.

The Small-Scales or Fierce snake disappeared as far as man was concerned for 100 years. They were known last century and then it was not until 1976 that live specimens were found in Western Queensland. When Jeanette Covacevich sent us the venom from this snake, it killed mice no matter what dilution we employed. It took six weeks or so before we could determine an exact lethal dose 50% (LD50) of this venom. It is far more lethal than anything described in the literature.

Venom Detection

Venom detection kits are held in most hospitals now. The Flying Doctor can actually do this assay on a plane. It is about the only laboratory test that I think doctors can do without prior training. Doctors are pretty rotten technicians. Venom detection is very important because if one knows what venom is present at the site, then the correct antivenom can be given to the patient when he gets ill. If you do not know, in most parts of Australia, the first dose you have to give is polyvalent antivenom. It is a big volume, 50 ml of antivenom versus 4 ml say for Brown snake antivenom. It is very expensive. Antivenoms are free to the patient so that taxpayers are paying $300 or so per ampoule versus much less for the other antivenoms. Serum sickness is much more likely with delayed sickness a ten per cent possibility with polyvalent antivenom. So the less antivenom one gives the better.

The venom detection kit is very simple with six capillary tubes all joined together on a syringe. They are coated inside with a very special antivenom. It is 1 molecule thick and covalently bound to the glass. A swab from the bite site, or a little bit of clothing, are the ideal samples because of the high concentration of venom. Blood or urine can also be used. The samples are drawn through the capillary tubing, and left 10 minutes. If there is a particular venom then it will stick to its antibody or antivenom on the inside of the tube. If there is venom present in the sample there will be a colour change.

Venom Lethality

The maximum output when the Small-Scaled snake is milked is able to kill a quarter of a million mice. That is really quite phenomenal. The Taipan kills 150,000 mice. The average output from a Taipan is 120 mg. Now 120 mg of Taipan venom will kill 12,000 guinea pigs. Try imagining 12,000 dead guinea pigs. If it bites a rat, the Taipan does not hang on to it, it knows the rat is only going to go a few metres and it just cruises off. We have gone from about 15 deaths from snake bite a year down to about one every two years. The most tragic recent death was a small boy mauled by a Taipan. He got multiple bites around his buttocks and groin. I think he was probably dead within 10 minutes. He would not have had very much hope with a massive envenomating in an area not accessible to first aid.

The Indian Cobra is about tenth on the list. It is the first overseas snake that gets into the list. It kills about 10,000 people a year. The most dangerous American snake is

capable of killing 3000 mice. The potential killing power of Australian snakes leads the world.

First Aid

Until a few years ago we could not research first aid properly because we could not follow venom in the serum of patients or experimental animals. Then radioimmunoassay was developed. For the first time ever in patients, alive or dead, my colleagues and I could measure all the types of snake venom and also the neurotoxins. This meant that coroner's cases could be cleared up and also one could study first aid. When we put venom in little monkeys that were fully conscious and could move we could reproduce the syndromes seen in children two years old or so. We could see how the venom peaked and when paralysis started. Over a two hour period venom peaks in the plasma and after two hours it is getting into the nerve tissue and causing paralysis. We could give them antivenom and they would recover. This allowed us to put venom in monkeys and try out all sorts of first aid.

For a number of reasons arterial tourniquets are not a good thing for snake bite, or other envenomings. They are hard to put on and very painful. Most people cannot stand an arterial tourniquet for more than about five minutes unless they are under an anaesthetic.

An arterial tourniquet stops venom movement but when it is taken off after 30 minutes the venom surges away. It was an accidental finding that if you put firm pressure over the bitten area, and kept the limb still then the venom stayed within the tissues.

When venom is injected it has no blood supply. It is an avascular blob of liquid. If we can empty the lymphatics and capillaries around it, it will stay there. The limb must be kept still to prevent the venom moving.

This brings me to a very important point on first aid and that is when to take the first aid measures off. Take them off when the situation is under control, with antivenom available, the drugs that are going to have to be used and resuscitation equipment all ready and tested. Ideally one gets a venom detection kit, cuts away over the bitten area, takes a swab and puts the first aid measures back on until the correct antivenom is available. What should not be done is to leave them wrapped up like mummies for eight hours in the Intensive Care Unit while waiting for signs of envenomation to appear because the venom is just going to sit in that limb until unwrapped. The neurotoxins are not destroyed, they persist in the body.

It is a very unscientific method of first aid because everyone will put the pressure on at a different pressure. If there is no crepe bandage available pantyhose seems to be quite ideal. One does not have to bandage the whole of the limb. We like to start below and go upwards because that empties the vessels but only a little of the venom is pushed centrally. If the patient says the bandage is painful, the bandage is too tight. If you are going to get bitten by a snake it is best to get bitten on the hand because you can then walk back to civilization.

Signs and Symptoms

If someone has received a significant amount of venom and no first aid has been given they will be getting sick within about 5-10 minutes from the time of being bitten. Very rarely does one get a child with multiple bites. We had one in Melbourne a couple of weeks ago, with bites from a Tiger snake all over his calf. He was critically ill.

The time immediately after the bite is often the time of panic. We are trying to take panic out of snake bite. From one to three hours is when most snake bites are treated, and cured with antivenom. Some of the best cases are treated in a small country hospital by a country GP who treats three or four cases every summer. They are not written up because they are treated promptly and there is nothing special about them. We only hear of them on the antivenom reports.

After three hours an untreated case can be in real trouble with paralysis, coagulation problem and myoglobinuria. Over the past 100 years there are reports of patients who were not paralysed, and passed very dark urine or highly coloured urine and then went into renal failure. Everyone thought that was haemoglobinuria. But we now know that it was myoglobinuria.

Giving Antivenom

If one gives pre-medication before an anaesthetic to make the course smoother, why not give it before an antivenom? Antivenoms are improving all the time but a proportion of the population do have reactions to antivenoms. It has been established that with good pre-medication and only giving antivenom when it is indicated the reaction rate is acceptably low. If a person has no history of allergy a little non-sedative parenteral anti-histamine, not a big dose of Phenergan if you can avoid it as they might go to sleep from it and a touch of adrenaline. That is what we recommend for a person with a healthy cardio-vascular system. Steroids should also be given if the patient has a history of allergy.

Allergy is a strange thing. Often it is like what happens with bee stings, a person might have a severe anaphylactic reaction in childhood or several years ago but the allergy seems to have disappeared. We have a few snake handlers who have had severe reactions in the past, who when they are given pre-medication take the anti-venom without any reaction. Others will have had delayed serum sickness twice before and are given antivenom the third time and do not develop delayed serum sickness. It is all a bit of a mystery how allergy waxes and wanes. On the other hand snake handlers often become highly allergic to the venom. So when bitten by a small Tiger snake, say one foot long, and the person will have an anaphylactic reaction and die. That has explained a number of snake bite deaths.

Points to remember

Pressure bandage first aid immobilises venom. When the bandage is removed venom (if present) is absorbed. Only those patients who develop symptoms need antivenom. How much antivenom to give depends on the clinical response

CLINICAL SEQUELAE OF SNAKE BITE

A McKillop.

If Dr Sutherland has trouble being original I do not know what chance I have got, talking about snake bite. It is a bit like neurosurgical anaesthesia, speaking fourth after three other experts, a lot of repetition, probably about 5% panic and no idea what the end result is going to be.

It is my brief to describe the clinical conditions which should be anticipated when patients have been envenomated by Australian land snakes. Much of it has been said. The protocol for managing land snake envenomation developed by Dr Sutherland and his colleagues is well documented. No doubt due to the availability of information, expertise and anti-venom in Australia, it is possible to gain the impression in clinical practice that it is a very uncommon event for our patients to develop serious complications. It is these uncommon events that I shall be describing today.

Snake bitten people still die in Australia. Why is that? The short answer is that the major lesions in envenomation are neurological, haemostatic and cytoycotoxic and they can rapidly lead to either serious illness or death. Usually, appropriate first-aid, if applied, will delay systemic envenomation and its consequences, until medical expertise is available to manage them. Rarely, patients suffer intravenous envenomation or massive envenomation following multiple bites and even rapidly applied first-aid may not prevent systemic envenomation. Anaphylactic reactions may claim other lives. Perhaps most of these deaths are inevitable. The mistake of assuming that a snake is harmless has recently in Queensland caused a death because of belated presentation for medical care. The situation more tragic than all of these is that of an ill patient reaching medical care with no history of envenomation and the diagnosis not being made because it is not considered.

Major sequelae are rare if appropriate first-aid has been applied and appropriate antivenom given. If the incorrect anti-venom is given based on correct identification of a snake, or if larger than standard volumes of anti-venoms are not administered when indicated, or if there is a significant delay before administration of antivenom, or if antivenom is not given at all when indicated, then the instance of major sequelae is greatly increased.

To return to the uncommon yet, particularly to those working in intensive therapy, the ever present possibility of failing to diagnose envenomation in ill patients giving no history of snake bite. The bite episode may not have been noted, the lesion may be invisible or atypical or, particularly in children, be unreported. A 7 year old child was bitten three times on the foot by a Taipan. The child presented 36 hours after envenomation to the practitioner, not knowing he had been bitten, with a history of not being able to swallow his Vita-Brits. At this stage the presenting symptom was difficulty in breathing. It was decided that perhaps the child could have epiglottitis, so the child was taken to theatre and intubated for epiglottitis and the epiglottis was normal. A further search was made and the bites were found. These situations can occur.

How may these worrisome patients present? The triad of headache, nausea, and vomiting are common early symptoms of envenomated patients. The diagnosis of envenomation, probably should always be excluded in areas where envenomation may have occurred in such presentations. The recent onset of cranial nerve palsy particularly ptosis, diplopia and dysarthria, limb weakness or ataxia should direct attention to a possible toxic origin. Drowsiness or coma, usually secondary to respiratory insufficiency, may be misinterpreted if envenomation is not included in the differential diagnosis. Spontaneous haemorrhage from acute coagulopathy is a rare yet potential presenting symptom. Bruising and swelling of the bitten area may, and has, mimicked traumatic, inflammatory or infective lesions. A band-aid placed over the bite area on the foot, could easily lead a Casualty RMO astray in managing a patient to consider his illness due to a septic arthritis. Many conditions have, in the literature, been noted as being incorrectly diagnosed in patients suffering from snake bites. Venom detection kits should be part of our diagnostic work-up in such conditions if any possibility of envenomation exists.

What then should we anticipate in those patients who have a history of envenomation or potential envenomation? I will exclude anaphylactic and anaphylactoid reactions which have been mentioned by Dr Airey. The major systemic dysfunctions which our patients may develop, are neurological, haemostatic and renal. The physician attending the patient must remember these when faced with a case of multiple poisoning. Three or more toxic reactions or changes may occur simultaneously or consecutively. The resultant syndromes are ventilatory failure, coagulopathy and acute renal failure. Each may be fatal in its own right, or may set the scene for sepsis, circulatory failure or complications of therapy. Although venom of various species may show a predilection towards a particular lesion, in practice, any envenomated, or possibly envenomated, patient must be handled with multi-system vigilance.

In looking in more detail at these pathological processes, I would like to make some comments on the neurological lesion. Accepting, as an anaesthetist, that diaphragmatic and intercostal paralysis is a worrisome clinical event, following envenomation you must remember that the progressive neuro-muscular blocking actions of the venom can precipitate severe pulmonary complications much earlier. The respiratory morbidity will be exacerbated by failure to appreciate the contribution of upper airway obstruction, aspiration of oropharyngeal secretions and gastric contents to the clinical events of atelectasis and pulmonary soiling. The cranial and peripheral neurological impairment may be of insidious or dramatic onset and vary from transient weakness to complete and persistent paralysis. Careful and repeated observations of neurological and respiratory status, should continue until resolution of neurological symptomatology has occurred. It is a common clinical observation that once significant neurological impairment exists, the response to anti-venom may be quite unimpressive. The management of patients with progressive respiratory impairment is familiar to us all. The lateral position, frequent oropharyngeal suction,

supplementary oxygenation and cessation of oral intake are appropriate initial responses. Correct, adequate and early anti-venom administration may minimize further impairment but progression may occur despite such therapy. Early tracheal intubation and skilled ventilatory support will minimize respiratory morbidity and may be needed for several days, even a week or more, until ventilatory adequacy and pharyngeal reflexes have returned. Adequate nutrition should be maintained and narrow-bore enteral feeding is usually tolerated by these patients.

The haemostatic deficit is initiated by prothrombin conversion and subsequent fibrogen consumption and depletion. The coagulopathy can be life-threatening. Even in the absence of significant neuro-toxicity it is of particular importance in Tiger, Taipan, Black and Brown snake envenomation. Clinically the disorder may produce oozing from the wound site, venipuncture bleeding, epistaxis, gastro-intestinal, uterine or even intra-cranial haemorrhage. Although death directly attributable to haemorrhage is uncommon, the additional physiological stress placed upon renal and pulmonary function may be a serious consequence. A coagulation profile is of great value and should be performed early and repeated regularly, if it is abnormal or if clinical signs of envenomation persist. The expected abnormalities are prolongation of the whole blood clotting time, prolongation of the prothrombin time and thrombin time, decreased fibrogin, decrease in factor V and VIII and an elevated level of fibrin degeneration products. Although Australian snake venoms may act on platelets in vitro the effect in vivo is variable. Thrombocytopenia accompanying the above abnormalities suggests disseminated intravascular coagulopathy (DIC) which can occur in severe envenomation.

The correct initial response to severe abnormalities is to give further anti-venom which most often produces a clinical response within 12 hours. If despite this therapy the profile remains abnormal and bleeding is worrisome, component therapy may be necessary. There is a real danger of exacerbation of fibrinogen consumption if adequate anti-venom has not been given before fibrogen administration. Secondary fibrinolysis activated by intravascular coagulation frequently accompanies systemic envenomation. It is usually not severe enough of itself to produce serious haemorrhage. It can be readily corrected by the use of Epsilon Aminocaproic Acid (EACA) but in this may have no effect on the clinical state of the patient or on the parameters of defibrination. Animal studies strongly suggest that EACA may increase mortality by inducing widespread intravascular clotting.

Snake venom may produce haemolysis which may be detected by blood film examination, bilirubin elevation, the presence of free haemoglobin, and haemoglobinuria. Microangiopathic haemolytic anaemia has also been described following envenomation in Australia. This is thought to be due to red blood cell injury by fibrin deposition in the micro-circulation.

The cytotoxic nature of venom components is most obvious in its destruction of muscle cells. The patient may complain of muscular pain, muscular tenderness and weakness. Although little is described in clinical literature myocardial involvement is probably concurrent and may contribute to the tachydysrhythmias often seen in snake envenomation. Elevation of all CPK isoenzymes has been shown. This suggests myocardial injury in these patients. The urine may be dark from myoglobin but this should be distinguished from haemoglobinuria by laboratory assay. The most serious consequence of cell destruction is myoglobinaemia and its contribution to the deterioration of renal function which may occur following envenomation. Shock, DIC, haemolysis and the defibrination syndrome have also been incriminated in the appearance of acute renal failure. There maybe a direct nephrotoxic action of a venom component. Renal function, particularly when myolysis is detected, must be monitored carefully. The renal complications may be minimized by correcting dehydration and avoiding hypotension. Therapy includes intravenous hydration, colloid, and vasopressor therapy to maintain renal perfusion and perhaps the use of diuretics to maintain a good urine output. Dialysis may be needed despite this therapy. Acute tubular necrosis is the pathological lesion most often found in envenomation associated with acute renal failure.

Fortunately with effective first-aid, skilled anti-venom and supportive therapy these sequelae of snake bite are relatively uncommon. When they do occur their seriousness may be minimized by anticipation, early recognition and appropriate therapy.

We have been unable to obtain copies of two of the papers presented during this session. These were "Taipan snake bite: a case presentation" by Dr John Orton and "Anaphylaxis complicating snake bite and its treatment" by Dr Ian Airey.

PANEL DISCUSSION

The panel consisted of Drs S Sutherland, J Orton, I Airey and A McKillop.

Question
Can Dr Sutherland explain why Australian snakes are so venomous?

Dr S Sutherland
That is a question that is often raised. There is really no firm answer. The fangs on our snakes are quite small compared with overseas snakes. It seems that instead of developing big jaws and teeth and weak toxins Australian snakes have gone in for powerful toxins and weak teeth.

Question
Although the bandaging technique is of undoubted benefit and represents a huge advance in the management of snake bite, does Dr Sutherland think it is going to increase the incidence of local tissue damage at the bite site. Secondly, whether this form of treatment would be suitable for envenomation, or snake bite, where neurotoxicity is not a major problem?

Dr S Sutherland
By and large the Australian snake venoms do not cause tissue damage such as is caused by Rattlesnake venom, which is full of enzymes to digest the tissues. One worry is what is going to happen when venom is trapped in the tissues for a long time. Will there be necrosis? In some cases, particularly when Tiger snake venom has been kept trapped in the proximity of a blood vessel for five or six hours, an area of necrosis does develop. It may be only 1 cm by 1 cm or the area looks as though it may lose its viability. When that has happened it has only been around the puncture site. If it is a finger the damage only appears on one side of the finger, where the venom is, and does not appear on the other side. If an arterial tourniquet has been applied in that situation there is. quite symmetrical damage around the finger.

Why does this happen? The venom causes intravascular coagulation in all the little vessels in that area. One little boy had first aid on and he had quite a wide area of marked cyanosis when the crepe bandage was taken off. Next day it was all back to normal, with just little fang marks visible. There is a small risk from leaving venom trapped for a long time in the tissues, there may be tissue damage. It does not appear to be serious. It is certainly better than the patient being dead. It varies from one venom to another. For example a bite by a King Brown snake is perhaps the most painful of the snake bites, and it is more likely to cause tissue damage than any other of the snakes. It also puts the biggest volume in so one would expect tissue damage.

People who die from Rattlesnake bite generally die from shock because the whole limb blows up with extravasation of fluid. The central effects are pretty harmless. We did work with Rattlesnake venom and found that the first aid measure kept the venom static. There was some necrosis but it is better to get necrosis at the site of injection than of the whole limb. The most important thing was that with crepe bandages there was no oedema of the limb. Take off the crepe bandage and one could watch the oedema appear. We cannot tell the Americans or Indians what to do. However, we have published material on American venoms in America. There is quite a following in the States for our first aid treatment. It is certainly better than the old ones of using Bowie knives or shooting the finger off.

Question

What is the incidence of false negatives with the venom detection kit?

Dr S Sutherland
You get a false negative when there is no venom or too little in the sample. Even if the test is negative we treat the patient. By and large every time that someone has been sick and the fang marks have been swabbed the venom has been found.

Question

Have you any comment on the attempts by the persons who have been envenomated to bring the reptile with them. In my opinion it is a particularly stupid and dangerous thing to do. It might be then difficult to catch the animal and the staff in hospital are placed in danger.

A voice from the audience
I had an interesting experience in Darwin. A snake was sold to three chaps in the bar at a hotel. It only cost them $1.00. They let it bite them many times. Then we had three patients presenting at once at the hospital. One with gastrointestinal haemorrhage, in the next cubicle was a chap with a fractured jaw, who had collapsed and had broken it on the way to the hospital, and the third chap (this is true, because it was recorded in the papers), went to the labour ward to visit his wife and child taking the snake with him. He collapsed on the ground, lost the snake and the whole Obstetric Department had to be evacuated. The snake, a Western Brown snake, was found two weeks later under the Occupational Therapy Department.

Another voice from the audience
A patient presented to the PA Casualty one day and said he had been bitten by a snake. They asked what sort of snake was it? He replied “I don’t know”. They said, “Did you catch it?” He said, “Yes,” and put his hand into his pocket and pulled out the head of the snake. They asked, “How did you get the head?” He said, “I bit it off.” The casualty staff enquired why. His answer was “The snake bit me so I decided to bite it!”

The old chap had performed the old fashioned trick of sucking the bite. Can you tell me anything about the oral absorption of toxin?

Dr S Sutherland
With an Australian snake bite you would not be able to suck anything out. It is a deep injection and the injection site closes up when you suck. But you can eat snake venom quite happily because it is destroyed by the pepsin in your stomach. There is no problem there.

Question

How quickly do people recover from coagulation disorders?

Dr S Sutherland
The young seem to recover their fibrinogen level very quickly after antivenom, sometimes in a couple of hours. Older people sometimes seem to take much, much longer. There is some aspect of liver function here. With the older person who has got a severe coagulation defect, it is a good idea to correct this with fresh frozen plasma. We have had one death after a Brown snake envenomation. He had a cerebral haemorrhage, although he was not hypertensive at the time. In Hobart a little while back a man had a massive gastro-intestinal haemorrhage after a Tiger snake bite. These two events make me feel that it is not a bad idea, in the older person, to correct the defect.

Question

How did you track how well you have reversed the circulating venom? If the patient still had circulating venom you have not given enough antivenom. If you then give him fresh frozen plasma, you could, theoretically, be making things worse.

Dr S Sutherland
We always say give adequate antivenom before you correct blood coagulopathy. It is a good point. I think you have

just got to make sure that all your clinical investigations indicate that every other aspect is under control including the neurological side and that the only thing you are treating is the coagulation problem. Never give fresh whole blood to snake bite victims, that has happened a few times without antivenom having been given. The patient has died with gangrenous arms and legs. By and large, properly treated snake bite has a fairly smooth course. The correction of a coagulation defect is usually achieved with more antivenom. The trend now is to give more antivenom than is really necessary.

I would like to see antivenoms improved further in Australia. I saw a little boy two weeks ago who had to have a lot of antivenom. I was distressed to watch the delayed serum sickness he developed. A lot of antivenom that goes in, perhaps 95% of it, has no specific activity. So there is still a lot to be done.

Unknown speaker
In relation to the coagulopathy, I have not had a lot of clinical experience with this, but in the couple of patients that I have dealt with I have found that if you give the antivenom and then try to correct the coagulopathy by factor replacement, that can actually be a guide as to whether, in fact, you have given enough antivenom. If you have further evidence of coagulopathy give them more antivenom, then give them more fresh frozen plasma.

Question
I wanted to address a question that Dr John Orton raised. Give sufficient antivenom to reverse the coagulopathy and then often the neuromuscular effects become fixed. With a neuro-muscular effect when do you decide you have given enough antivenom?

Dr J Orton
The coagulopathy appears to be the more easily, the reversible component envenomation of it. The neuro-muscular blockade, particularly where there had been delay in establishing antivenom treatment, may becomes, and probably will with Taipan, irreversible. Obviously with neuromuscular problems the treatment is ventilation until they wear off. We should all know that. Could Dr Sutherland comment on using the degree of coagulopathy as a method of titrating the amount of antivenom required.

Dr S Sutherland
Normally after 24 hours all the venom has been absorbed from the bite site. If one did a post-mortem then one would not find very much venom at all at the bite site as the venom has been absorbed and gone to its target areas. It reaches its target areas via the blood stream. By and large one or two doses of antivenom will saturate all the target areas. We know that when the venom peaks in the serum, a lot comes out in the urine including some of the neurotoxins.

Once I had to give a talk at a large hospital. It is one of these hospitals that you go into and when you go to the "Enquiries" there is a notice "At lunch. Back at 1". The Superintendent, when finally I found him, said, "We only asked you to come out and give a talk because you have been making such a fuss about snake bites. We never get any cases." It was really a lovely welcome. I gave the talk and I had just got back to the Lab when the registrar in casualty at this hospital, who had not been to the talk, rang up in a panic because they had a case of snake bite. You never know when a snake is going to strike.

Question
I was involved once with a gentleman who was bitten by a Brown snake on his long saphenous vein. I am pretty certain there was a degree of direct intravascular injection of the venom. The chap that was with him said that about five minutes after the bite he started fitting. I eventually went to the medical evacuation of this guy. He was fitting quite severely. If you give to your animals direct intravascular injection does fitting occur? Or was this man's fitting related to hypoxia or inter-cerebral bleeding or epilepsy?

Dr S Sutherland
There have been a number of cases in humans where epilepsy has followed snake bite. We do not know why it happens, but there is always a massive amount of venom involved. There is often no history of epilepsy before the incident. It would be very hard to design an experiment in mice to see if epilepsy occurs. They are very sensitive to venom and before you could tell that they had epilepsy, they would be dead on the table.

Question
Could you give us a brief word about the long term effects? A couple of our patients have had neurological involvement and muscle weakness for many, many months. The ones that had the severe renal failure were a bit changed mentally afterwards.

Dr A McKillop
Headache is the most common persistent symptom after envenomation. Paresis of individual limbs has been described. Over-zealous tourniquet application may have been the cause. Whether there is a direct proteolytic injury to the nerves, I am not sure. Permanent injury to patients has been described in the Australian literature. In Western Australia three patients had persistent diminished renal function, when reviewed six to twelve months after the episode. Other patients have suffered cerebral injury, presumably part of the whole syndrome of hypoxia or hypotension.

Question
Could Dr Sutherland comment on direct proteolytic damage to nerves?

Dr S Sutherland
Direct nerve damage is uncommon. You will occasionally get it locally with massive envenomation. The patient has paraesthesia around the bite site with loss of motor power.

There are multiple targets where the venom is going to attack. A severe case generally takes three or four months to get back to normal. A lot of them lose quite a lot of weight. This generally relates to delay in treatment. If they get serum sickness they could be washed out for months. For example with cases of Funnel-web spider bite that survived before antivenom the patients often were weak and "run down" for months. By and large these envenomations do not do you much good!

HYPOTHERMIA

John Knight

The title of this talk is hypothermia. However, I am not going to be dealing with the effects of core temperatures below 34.5° which is commonly accepted as hypothermia in medical terminology, but rather with the effects of cold on divers and their survival.

HEAT BALANCE

Human life is a balancing act between heat gain and heat loss. The heat content of a 70kg man, is some 9000 Kcal. This is the amount of heat needed to take 55L of water, containing a soup of fats, proteins, carbohydrates and minerals, from 0°K to 37°C.

The balance is pretty fine as a gain or loss of 200Kcal, just over 2% of the heat content, will incapacitate that 70 kg man, either by hyperpyrexial delirium or hypothermic coma.

Human heat regulation is centrally controlled, in the hypothalamus, in response to blood temperature variations and messages from the skin. There is a constant heat output from the heart, liver, respiratory muscles, kidneys and gut in the torso (the core) and from the brain. In addition, bursts of heat are generated from the use of limb muscles.

Man evolved in the tropics and it is thought that he was a hunter-gatherer from the start. This involved running in the hot sun, so one of the adaptations that developed in man was a very efficient heat losing system. Heavy, workloads can be carried out in a large variety of temperatures and the rectal temperature maintained within the normal range. Extra blood flow through the skin increases radiant heat loss and extra sweating increases evaporative cooling.

The human race spread all over the world before the days of the steamship. In most places the first Europeans discovered the inhabitants of faraway islands to be dependent on stone tools, so a primitive technology allowed mankind to live in the arctic, the tropics, and anywhere in between. How was this achieved? It was by using extra insulation and extra heat. The extra heat was provided by fire. The insulation could be animal skins, as for the Eskimos, or other sorts of clothing. The adaptations of the human to cold should be well known to those of us who do not live in Queensland. They are a decrease in heat loss due to skin and limb vasoconstriction and an increase in heat output by shivering.

The normal human response to cold is vasoconstriction in the skin, and then vasoconstriction in the arms and legs occurs. The major limb blood vessels form a counter current heat exchanger. So the arterial blood reaching the limb has been already cooled by the cold venous blood, coming back from the limb, which picks up heat, thus not cooling the core quite as much as one would expect.

Vasoconstriction effectively reduces heat loss from the torso, limbs and face. Most Australians have an insulating layer of fat over the muscles. Heat loss is mostly from the neck, axillae and groins where major arteries are near the surface. Another area of heat loss is the scalp which does not develop vasoconstriction in response to cold.

COLD ADAPTATION

Humans can adapt to cold. It is not something that happens quickly. It requires long exposures to cold. Adaptation to cold involves some changes in the body's reaction to cold, the major ones being that the basal metabolic rate is increased and the shivering threshold is decreased. The person adapted to cold produces more heat at rest and starts to shiver at a lower core temperature than the normal person. Most sports divers are not exposed to cold often enough to develop any adaptation.

There has been some dispute as to whether cold adaptation really occurred in humans, but the studies of the Korean diving women, the Ama, carried out by Professor SK Hong over many years have shown that it does occur (see p6).

In the middle 1960s, when Professor Hong started studying the Ama they were fatter than the non-diving village women and, in winter, had higher basal metabolic rates. At that time these women, dressed only in thin cotton drawers, could dive for shellfish and seaweed in up to 10m of water, in the summer for about 30 minutes twice a day before they had to get out of the water. They were too cold to carry on and their core temperatures had dropped to the region of 35°C. In winter they could only tolerate one half hour dive a day.

In 1977 wetsuits were introduced. The wetsuit works by trapping a layer of water between the suit and the body. That layer of water warms up to skin temperature. The wet suit prevents various currents that take heat away from the bare skinned swimmer. The air in the fabric of the wetsuit, which is a mass of bubbles of nitrogen or air in rubber, acts as an efficient insulator and reduces heat loss. Someone in a well fitting wetsuit, that does not allow water to slosh in and out with each movement, can stay quite warm for some time. The wetsuit is an excellent insulator on the surface, as anyone who has worn one on a hot day will know. Unfortunately, it compresses with depth and becomes a less efficient insulator.

Now the Ama can dive for up to two hours, in summer and winter, for a drop of only a degree or so in their core temperatures. Their cold stress has been very considerably reduced. In the three years after the introduction of wetsuits the raised metabolic rate that had developed as a result of the cold stress declined to the same metabolic rate as those onshore. Over the same period the time that these women could stay in the water without their wetsuits became shorter. Now they are dependant on their wetsuits for their livelihood, as they have lost their adaptation to cold.

All the women in the village are fatter now than their mothers were in the 1960s. This is probably due to better economic circumstances for the villagers.

WATER CONTACT REFLEXES

There are a number of reflexes that occur when cold water is applied to the skin. If the face, the beak area of the face

especially, is put into cold water, the response is bradycardia and apnoea. Naturally enough one does not try to breathe water. The bradycardia is known as the diving reflex, and is to be found in all mammals and birds. With bradycardia goes vasoconstriction of everything except the vessels supplying the brain and heart. This of course has survival value by restricting the supply of oxygenated blood to vital organs.

Another reflex that occurs is a gasp when cold water is applied to the skin. I have had plenty of experience of this as for four years at school I had to have a cold shower every morning, regardless of the weather. It is possible, if you know the cold water is coming, to suppress the gasp, but it can be very difficult. Some people suffer an exaggerated form of gasp when they fall into very cold water. They develop an extremely rapid and deep respiratory pattern which is quite beyond voluntary control. Under these circumstances, they must keep their faces out of the water or they will drown. The effort required to keep your head out of the water is quite high, and trying to swim when you are breathing at 30 to 40 breaths a minute and paddling hard to keep your face out of the water, is very exhausting. This phenomenon explains many mysterious deaths of good swimmers who have fallen into cold water. It was discovered quite by chance one day during cold exposure experiments at Cambridge when a champion swimmer fell into the water and developed this pattern of respiration and was unable to reach the side of the swimming pool only a few strokes away. He was rescued as his head was disappearing.

With very cold water, this hyperventilation is sometimes combined with an inability to move. I have seen one case of this when the Royal Australian Naval Reserve diving team in Melbourne was searching just below the Eildon reservoir dam for a missing person, who was not there. The water temperature was about 5°C and the shade temperature was about 35°C. The divers, united by buddy lines, dropped out of the boat. The fourth one rolled out but did not bob up as the others had. When he had been pulled up and was hanging onto the side of the boat he said “If I had not had my regulator in my mouth, I would have drowned. I was breathing as hard as I could, and I couldn’t move”. A respiratory pattern like this, of course, is immediately fatal, unless you happen to have a regulator in your mouth and a buddy by your side.

IMMERSION

Water is a much more hostile environment than air because of its thermal capacity and specific heat. It takes a thousand times more heat to heat one volume of water than it does to heat a similar volume of air. Water conducts heat 25 times faster than air does. Seventy one per cent of the body volume is within an inch (or 2.5cm) of the skin, thus reducing the distance through which heat has to be transported to the outside world. This very adaptation for losing heat is a danger to those of us who entertain ourselves by submerging. It is even more of a danger for those who involuntarily find themselves in the water. Very many of the deaths from drowning that occur after aircraft land in the water, or ships are sunk, are precipitated by hypothermia interfering with the ability of the person to think or swim.

The body in contact with water which is colder than itself, the normal situation, rapidly loses heat to the surrounding water. This sets up convection currents in the water which move the heated water away from the body, cold water moves in and the process starts again. Add to this small scale heat transfer the water movement due to swimming movements and one can see that this is a potential for very high heat loss. In fact it is much better for the submerged individual to avoid moving and huddle in to a little ball, the so-called Heat Exchange Lessening Posture (HELP) than to try and swim. Lack of movement reduces heat loss considerably.

The factors governing heat loss are the temperature of the water, the amount of heat generated in the body and the amount of insulation to slow down heat loss from the body. If one compares the heat losses of a thin person and a fat person, both while sitting still in water and swimming, one discovers that a thin person cannot sustain the swimming without a large drop in core temperature, while the fat person, thanks to his insulation keeping heat in his core, can maintain his body temperature, which is one of the things that explains why long distance swimmers on the whole are well covered people.

The human body copes with cold by vasoconstriction and increased heat output. When the human body goes into water, at first there is a high rate of heat loss. This decreases as vasoconstriction occurs. Then the rate of heat loss decreases again as increased heat production starts. The use of a wetsuit removes the first, steep part of the heat loss curve, and by increasing the insulation around the person, allows more efficient retention of the extra heat generated. However, a wetsuit does not stop heat loss. It merely reduces it.

Cold induced vasoconstriction affects both the arterial and venous sides of the vascular tree. This results in increased resistance, causing a rise in blood pressure, and an increased venous return to the heat resulting in a larger cardiac output which accentuates the rise in blood pressure. Both systolic and diastolic pressures rise in the immersed vasoconstricted human.

If the person has hypertension, this rise in blood pressure can be extremely high. This increased cardiac workload has been thought to be responsible for a number of diving deaths in those with the older age groups, that is the over 30s. This is based on American figures which do show that as divers get older, the proportion of definite cardiac deaths increases. Many of these deaths occur at the end of the dive when the person is on the surface of the water. They are not drownings.

At the beginning of cold water immersion experiments there is a rise in core temperature because the shut down of limb circulation keeps the warm blood in the core.

COLD INDUCED MALFUNCTIONS

The first symptoms of heat loss are feeling cold, followed soon after by shivering. And then, long before you have reached a dangerous temperature at your core, there are the mental changes of slowness of thought, slurred speech, with these goes inco-ordination. Cold affects the

transmission of nerve impulses down nerves, and also affects the rate of contraction of muscles. The combination results in malfunction and difficulty in fine movements, and as one grows colder, the grosser movements get affected as well. Anyone who has been exposed to low environmental temperatures knows that their fingers go numb, that the sensation of touch is impaired and their fine movements are soon impaired.

The early signs of hypothermia are very similar to those of the early signs of alcoholic intoxication, a certain sense of well being and ignoring the obvious problems around one. This is a very dangerous situation for a diver. One of my colleagues in Melbourne went diving in October some years ago. When he surfaced at the end of the dive, he was unable to climb into the boat. He had to be helped in. In his own words, he was blue with cold, and yet he was not the slightest bit upset that he was too weak to climb up into the boat, a thing he could normally do quite easily. He has no real memory of this, all he remembers is what his friends have told him. He obviously had difficulty concentrating. He did not progress through slurred speech, disorientation and hallucinations to drowsiness and coma.

Of course, somebody floating in a lifejacket who gets cold enough to go unconscious will not have the protective reflexes to avoid breathing in water when a wave gets up and slaps him in the face.

People who are becoming hypothermic often stop swimming. They just quietly sink. History is full of sad stories of wrecked sailors clinging to the mast until one by one they lost their grip and fell into the water. This is certainly due to hypothermia, a hypothermic loss of muscular control, leading to falling into the water and drowning. During the Second World War, many men were found floating dead in their life jackets. They had not drowned. Their lungs were perfectly normal. They had died from cold, from loss of body heat.

On Arctic convoys from the UK to Murmansk, the expectation of life for a downed fighter pilot in his standard flying suit was less than five minutes after he was in the water. They were often still alive when they were pulled into the escorting destroyer's whaler which reached them in five minutes or less, but they died in the boat on the way back to the destroyer. Those that did survive always had to be helped up the scrambling nets, they could not get themselves up as muscular power had gone from cold. Introduction of immersion suits that kept the man's clothes dry dramatically changed the picture. With the layer of dry clothing trapping air between him and the survival suit, he could live for half an hour or more without getting into the situation that five minutes exposure had given him before.

Under cold water it is necessary for insulation to be applied to the head because the scalp, unlike the rest of the body, does not have a cold induced vasoconstriction. The head goes on losing heat even though all the other surface vessels are severely constricted. As the head is some nine to ten per cent of the body surface area, and a good half of this is scalp, there is a large loss in heat from in the head. Mind you, it is a lot more comfortable to go diving without a hood, but in cold water it is very sensible to wear one. Without a hood there is only a very small distance between cold water and the brain and the tissues are not efficient insulation. So the brain cools locally as well as being cooled by a cooler blood flow.

Cold induced mental change is the major problem of incipient hypothermia for divers because once one's brain does not work properly, one makes wrong decisions.

Heat loss in a wet suited diver does not stop when he gets out of the water. Most wetsuits have nylon cloth on both sides of the neoprene. The water trapped in outside cloth will evaporate in the air currents that eddy round the diver as the boat moves to the next dive site. The heat for this evaporation comes from the diver's body. So he will start the second dive colder than he was when he finished the first dive unless he takes his wetsuit off and puts dry clothes on between the dives.

AFTER DROP

Once a drop in core temperature has occurred the core temperature continues to drop after a person is removed from the water and dried. When wrapped up well and no longer losing heat the core temperature continues to drop. It continues even when active rewarming is started.

This after drop can be as much as 1°C. It is due to the cold parts of the body draining heat from the core as they warm up. Whatever method of warming is employed this after drop will occur.

PREVENTION AND TREATMENT

Hypothermia only occurs when heat loss exceeds heat gain. The most important treatment is to prevent the occurrence of hypothermia. Not staying underwater too long, not diving when chilled and warming up thoroughly between dives are simple, sensible things to do. Feeling warm is no guarantee that your heat losses have been replaced. The only way to be certain that heat losses have been replaced is to start sweating. This shows that the body needs to lose heat.

Recognition of diving induced hypothermia must be clinical. The essential treatment is to stop further heat loss. So the victim must be taken out of his wetsuit and dried and covered with as much insulation as is available. Adding heat is the next step. In a diving boat the only available source of heat may be other human bodies.

Acute hypothermia should be treated by rapid rewarming. The simplest method of adding heat is to immerse the body in warm water. The difference between skin temperature and the bath temperature should not be more than a few degrees; anything more is painful. Of course there are problems associated with a reduced blood volume (both immersion and cold cause a diuresis) and the peripheral vasodilatation due to the warm water on the skin and the continuing after drop of core temperature with this method. But it is efficient and well tried.

THE RISKS OF HYPOTHERMIA TO DIVERS

The risks of hypothermia to a diver are not those of death through cardiac irregularities and arrythmias brought on

by cold. They are much more the risks of cold induced errors due to clumsiness in muscles, clumsiness in thinking, inappropriate thoughts, and if the person does go unconscious from cold, drowning through loss of protective reflexes.

FURTHER READING

Alexander J. Respiratory Heat Loss. In: *Thermal Problems in Diving, Hypothermia-Hyperthermia*. Wilmington, Commercial Diving Center, 1977; 65-70.

Beckman EL and Reeves E. Physiological Implication as to Survival During Immersion in water at 75°F. *Aerospace Medicine.* 1966: 1136-1142.

Brauer RW. Temperature Regulation in High Pressure Environments. In: *The Pharmacology of Thermoregulation*. Symposium, San Francisco, Basel Karger, 1973; 99-111.

Craig AB and Dvorak M. Thermal Regulation of Man Exercising during Water Immersion. *J Appl Physiol.* 1966; 21: 1577-1585.

Craig AB and Dvorak M. Thermal Regulation of Man Exercising during Water Immersion. *J Appl Physiol.* 1968; 25(1): 28-35.

Edmonds C, Lowry CJ and Pennefather J. *Diving and Subaquatic Medicine*. (2nd ed) Sydney: Diving Medical Centre, 1981; 289-299.

Freeman J and Pugh LGH. Hypothermia in Mountain Accidents. *International Anaesthesiology Clinics.* 1969; 7: 997-1007.

Keatinge WR. *Survival in Cold Water*. Oxford and Edinburgh: Blackwell, 1969.

LeBlanc J. Physiological Changes in Prolonged Cold Stress. In: *Proceedings of the Cold Water Symposium*. Toronto: Royal Life Saving Society, Canada, 1976; 9-14.

Pozos RS and Wittmers LE (eds). The nature and treatment of hypothermia, Minneapolis: University of Minnesota Press, 1983.

Pugh LGCE. Temperature Regulation in Swimmers. In: Rahn H (ed). *Physiology of Breathhold Diving*. Nat Acad Sc National Research Council Publication, 1965; 1341: 325-348.

Pugh LGH. Tolerance to Extreme Cold at Altitude in a Nepalese Pilgrim. *J Appl Physiol*. 1963; 18(6): 1234-1238.

Pugh LGH. Cold Stress and Muscular Exercise, with Special Reference to Accidental Hypothermia. *Br Med J*. 1967; 2: 333-337.

Pugh LGH. Isafjordur Trawler Disaster: Medical Aspects. *Br Med J*. 1968; 1: 826-829.

Raymond LW. The Mechanisms and Management of Hyperthermia and Hypothermia. In: *Thermal Problems in Diving, Hypothermia-Hyperthermia.* Wilmington: Commercial Diving Center, 1977; 35-63.

Raymond LW, Bell WH, Bondi KR and Lindberg CR. Body Temperature and Metabolism in Hyperbaric Helium Atmospheres. *J Appl Physiol*. 1968; 24(5): 678-684.

Webb P. Human Tolerances of Thermal Extremes. In: *Thermal Problems in Diving, Hypothermia-Hyperthermia.* Wilmington: Commercial Diving Center, 1977; 17-28.

Webb P. Cold Exposure. In: Bennett PB and Elliott DH (eds). *The Physiology and Medicine of Diving and Compressed Air Work.* 2nd Edition. London: Bailliere Tindall; 1975; 285-306.

Webb P. Thermal Problems in Undersea Gaseous Environments. In: *Thermal Problems in Diving, Hypothermia-Hyperthermia*. Wilmington: Commercial Diving Center, 1977; 29-33.

THE DROWNED LUNG

V Callanan

I am going to talk about some of the pathophysiology of the drowned lung. This was not always the part of drowning that received much emphasis. In the 40's and 50's more emphasis was placed on changes in serum electrolytes and blood volumes. In the 60's and 70's the lung problems and the acidosis associated with it became a subject of research. In the last 10 years or so lung problems and cardio-vascular functions, in particular, have become prominent and in more recent years, brain preservation has become a widespread topic in this area.

I intend confining myself to talking about cardio-pulmonary aspects of near drowning. I will exclude victims of dry drowning, that is those who have laryngeal spasm and do not actually have water entering the lung. So I am talking about patients who get water in their lungs. Water in the lung quickly produces profound hypoxia, hypercarbia and a profound metabolic acidosis. By that time the patient reaches hospital, some of this may be reversed slightly by cardiopulmonary resuscitation. The hypercarbia may be reversed, however the profound hypoxia and metabolic acidosis will always be present. Usually the patients present with all of this triad unless the patient has recovered sufficiently to restore his cardio-pulmonary function almost to normal.

By the time the patient reaches hospital it really doesn't matter whether the patient drowned in salt or fresh water because by that time the syndromes are fairly similar. In fresh water aspiration the patient will finish up with surfactant damage and the adult respiratory distress syndrome (ARDS). If they drown in salt water, for various reasons, they finish up with the same syndrome. If they happen to aspirate their stomach contents then the same thing can happen. I would like to highlight the importance

of the aspiration of stomach content or vomiting in the near drowned victims because, as you are aware nausea and vomiting is very common in patients who become hypoxic and that is the problem with the patient that is near drowned. Figures taken from the Surf Life Saving Association show how common vomiting and regurgitation is in patients who need resuscitation. In those needing cardiopulmonary resuscitation, the experience was that 90% of the victims had either vomiting or regurgitation and overall difficulties with the airway occurred in 50% of these patients. However, whatever the exact mechanism is, the patient finishes up with the adult respiratory stress syndrome.

Large amounts of water may be ingested. That, with the water aspirated and perhaps some fluid resuscitation along the way, leads to a hyperhydration syndrome with an expanded blood volume. This, combined with alveolar and capillary damage, leads to excess amounts of water in the lung and to pulmonary oedema, which is the basic problem in the near-drowned victim. When a person presents to hospital the pulmonary care of the patient must be the initial concern. I do not want to dwell on these various aspects because they are reasonably well known. The techniques that are needed in the pulmonary care are airway control, which depends predominantly on the level of conscious of the patient, oxygen therapy for hypoxia, positive end expired pressure (PEEP) with or without intermittent positive pressure ventilation, depending again on the cerebral status and respiratory status of the patient. These techniques and the initial state of the patient are assessed using clinical examination, blood gases, chest x-ray and in the more severely affected patient invasive haemodynamic monitoring. The cardiovascular care of the patient, which is always done in conjunction with the pulmonary care, is firstly to optimize the patient's cardiac function. That means optimizing the blood volume, perhaps using bicarbonate for the severe acidosis if that is thought to be depressing cardiac function, and the use of a vasopressor and again the assessment is done using techniques with which most people here will be familiar. Other therapies which may be necessary are the use of bronchodilators if bronchospasm is present to a significant degree using either aminophylline or a 82 stimulant. These may have other beneficial effects. Steroids have been used by some people. There is, however, no evidence that they make any difference to the outcome of the patient or to the magnitude of the pulmonary oedema. Antibiotics are in the same class as steroids. They are not necessary unless the patient happened to drown in a sewer.

If humans get water in the lungs profound hypoxia occurs. One of the main reasons for this is that the shunt through the lung can be as high as 70% of the cardiac output. This means that 70% of the blood, as it goes through the lungs, is not being oxygenated. In the patient with anything but a mild degree of hypoxia, then the use of positive end expired pressure produces a dramatic result in these patients. Now whether this is applied as continuous positive airway pressure (CPAP) via a mask in the patient who is conscious, and able to generate adequate respiratory volumes, or it is CPAP via an endotracheal tube in a patient whose level of consciousness is not good but who can still maintain, although cerebration is impaired, adequate ventilatory volumes, or whether intubation, intermittent positive pressure ventilation and PEEP is needed, does not matter. PEEP always has a dramatic effect. Some patients seem to need intermittent positive pressure ventilation especially to open the collapsed alveolar ducts and alveoli that have filled with water. When a significant amount of this occurs, the lungs become very stiff and the amount of work necessary to generate the pressures to initially open these airways in a particularly ill patient cannot be generated by the patient, so often intermittent positive pressure is needed. Sometimes only for a short time in the initial stages and then as improvement occurs, the patients can manage to ventilate themselves if they are assisted with the positive end expired pressure.

PEEP does not decrease the amount of water in the lung but probably expands the alveoli to the point where the amount of water is made a thinner layer so that gas exchange can occur across the alveolar capillary membrane. PEEP is good stuff but unfortunately it is a two-edged sword. The good edge is that it increases the functional residual capacity, in other words, it expands the lung. It certainly decreases the shunt and so allows oxygenation of blood going through the lungs to increase and therefore increases the partial pressure of oxygen in the arterial blood. However, it does have some unwanted effects. The first being an increase in physiological dead space, which means that the patient needs to move, or have moved for him, larger volumes of ventilation to eliminate carbon dioxide adequately. In most cases that is not a significant clinical problem. However the other detrimental effect of a decreased cardiac output certainly is. Whether the patient gets the decreased cardiac output from ARDS or some other cause or from near drowning the problem is the same.

Let us look at exactly why this occurs and what we might do about it. The most probable cause for the decreased cardiac output is the decreased pre-load in the heart. I would ask you to keep in mind what pre-load is. Starling, in his well known articles, did not mention preload as pressures but referred to fibre length, and fibre length is better reflected by an end diastolic volume in whichever heart chamber you are referring to, rather than pressure. While in the healthy heart volume and pressure might be linearly related, in any patient who is ill the compliance, or stiffness, of the chamber can vary enormously and therefore the pressure that one measures may not necessarily accurately reflect the pre-load of the heart. What are the mechanisms by which the pre-load is decreased? The one which we have all been familiar with for a long time, is increased mean intra-thoracic pressure, or increased pleural pressure, decreasing venous return therefore decreasing the cardiac output. There are some other mechanisms which may be important, whether the patient has ARDS or is having PEEP for near drowning or whatever cause. One is the fact that the lung volume, increased by the PEEP, squeezes the heart, and so the ventricle does not relax as well, which means that it has effectively become more stiff. At the same time PEEP and the ARDS cause an increased right ventricular after-load due to the increase in pulmonary vascular resistance, both from the disease process and the extra pressure that PEEP will apply. These can induce a degree of right heart failure with dilation of the right side of the heart. This can shift the ventricular septum across, pushing it into the left ventricle and changing the functional characteristics of the left ventricle causing

a decreased compliance. If you have a decreased end diastolic volume for the same filling pressure that is usually interpreted as a decrease in contractility and it gives a decrease in the left ventricular stroke volume. The problem that occurs from this mechanism, is that it is difficult to know how to treat it. Another mechanism which has been postulated to decrease the cardiac output is a humeral one which has been shown in cross-circulation experiments to occur, although its exact significance in any particular patient and exactly what the mediators of this humeral response are, have yet to be elucidated. So it is a complex problem.

How best to increase the cardiac output, given that you have a problem? There are three paths and they effect the determinants of the cardiac output. As we know, one is by affecting pre-load, and I have mentioned that. The second is by increasing contractility by the use of a vasopressor and the third would be to increase forward flow by decreasing the after-load of the ventricle. PEEP should be considered in the overall context as not curing the disease that is going on, but to pinch an orthopaedic term, as a splint to maintain the lungs in the position of function until recovery slowly occurs.

The message I want you to remember is that no matter how good the care once the patient reaches hospital RESUSCITATION AT THE SITE OF DROWNING IS THE MOST IMPORTANT DETERMINANT OF ULTIMATE PULMONARY AND CEREBRAL SURVIVAL.

Dr Vic Callanan is Director of Anaesthesia and intensive Care at the Townsville General Hospital.

SALT WATER ASPIRATION SYNDROME

Bart McKenzie

Navy divers have very strong macho image. When they are under training, it is very important to submit to peer group pressure and live up to this macho image. They do not complain about minor problems. When they are under training if they get some fractures in their toes or perforated eardrums, they just keep on diving as long as they can put up with the pain and vertigo. But there was one thing that really used to stop these divers in their tracks, and that was the salt water aspiration syndrome.

This was a syndrome that we often used to see in Navy divers. They would present with an acute febrile illness. It was short lived and was characterised by a high fever, by myalgia and by respiratory distress. The typical scenario was that the divers would present to the Royal Australian Navy School of Underwater Medicine which was closely associated with the Diving School, with a constellation of symptoms. The main feature was that they felt very ill and they looked very ill, often they were cyanotic. They complained of fever, rigors, malaise and dyspnoea. They usually presented in the evenings after a day's diving and often after night diving as well. They were usually breathing fast. They had some respiratory distress. They had creps and rhonchi in their chests and occasionally they were blue. They usually had a high fever, a temperature of 39° or so and they had a tachycardia. Chest x-rays showed that they had patchy pulmonary infiltrates, which looked a bit like pulmonary oedema but it could pass for respiratory tract infection as well. There was a cotton wool appearance around the lungs. When we did their blood gases they were invariably hypoxic, sometimes with oxygen tensions down to about 40 mm Hg or so and they had a slight hypocapnia as well.

They invariably gave a history of aspiration of sea water. The Navy had some fairly obsolete equipment up until about five years ago, and the demand valves that they used almost invariably leaked. Most of the divers, most of the time, were inhaling a fine aerosol of water through the leaky demand valve. Another source of aspiration of water was from buddy breathing. Buddy breathing is when the divers share a demand valve. They pass it from one to the other. When one diver takes it out of his mouth and hands it over to the other diver it usually fills up with water. This is not a great problem if you have got modern equipment, because you just exhale into the demand valve and that exhausts the water out through an exhale valve. But the Navy divers' equipment had a rather large dead space. It needed about a lung and a half full of air exhaled into the demand valve in order to clear it. Modern equipment has a purge button and if you have not got enough air in your lungs to clear the demand valve you press the button and it blasts compressed air into the demand valve and blows the water out. But the Navy equipment did not have purge buttons. So buddy breathing was a fruitful source of water aspiration. There were other ways of aspirating water as well in Navy diving training but buddy breathing was the most common one. At night in Sydney Harbour the only thing you can see is the luminous dial on your watch, everything else is done by feel and by guess work. So buddy breathing at night was another fruitful source of aspiration of sea water.

Salt water aspiration is not near-drowning or even near, near-drowning. The amount of sea water that was being aspirated was only about just 10 mls or even less. So, I am not talking about a drowning syndrome, this is something quite different. The divers would aspirate water for one reason or another, and then continue their diving. They were not incapacitated. Often they would have a history of a bit of a cough after the dive. They might even cough up a little bit of blood, and then they would be OK. They would carry on with their normal diving activities without any problem. There was a latent period before they developed the full blown syndrome. This latent period was usually about one hour to two hours, but it had a range. Sometimes if they were really bad they would present almost immediately and the outer limit was about 15 hours. Then they would present with the syndrome myalgia, shortness of breath, cyanosis and all the rest of it.

Before the salt water aspiration syndrome was described, they would present complaining of this syndrome, and the doctors thought that the divers either had an acute respiratory tract infection or viraemia or even pneumonia. The whole clinical pattern certainly fits that sort of thing. So the doctors, quite reasonably, would fill them up full of antibiotics and put them to bed. By the next morning they were completely well, absolutely asymptomatic. However without being given antibiotics, they still got better in the same time. This was a bit perplexing.

The syndrome was looked at by Carl Edmonds in the early 1970s. He closely documented about 30 cases just to see what would happen. When he did blood gases on them and found that they were cyanotic, he thought he might try 100% oxygen. Not only did 100% oxygen reverse the dyspnoea and hypoxia but it also reversed the whole syndrome. If one gives these guys 100% oxygen, within about half an hour not only is the hypoxia gone but also the fever, the myalgia - the whole shooting match goes. I find it extremely interesting that oxygen should resolve the syndrome. Carl did some other things with the divers. Being a scientist he thought he would try to reproduce the syndrome and it was easily reproducible. If he gave them a leaky demand valve to breathe from, they would get the syndrome. He also found that if he then gave them 10% oxygen to breathe that would really bring the syndrome on. However, the divers were not really keen on this and neither was the Navy Ethics Committee, so he had to give that experiment away.

There are a lot of interesting features in the syndrome. Firstly, the divers would often present in groups. There would be no cases for a month or two and then all of a sudden there would be 10 or 15 in a period of 2 or 3 days. Why this should be I am not sure. The equipment leaked all the time and these men were aspirating sea water during their training all the time yet only occasionally would they get the syndrome. It suggests that there was something in the water that was giving it to them, either an infective agent or maybe it was an allergic reaction. The other interesting feature was that there seemed to be an individual susceptibility. Sometimes you would get the same diver who would present during his training with six successive cases of aspiration and yet other divers who had been diving alongside him and even using the same equipment and inhaling the same salt water did not get it at all.

I thought originally that this was perhaps confined just to Navy divers, but it is not. We now know that civilian and sports divers get the syndrome as well. We have seen it on a couple of occasions in surfers who have aspirated a bit of water after being dumped and that kind of thing. The patho-physiology of it is of great interest to me. I do not know much about it nor does anybody else either. There has not been much research on it. Maybe it is just part of a continuum between asymptomatic aspiration of a small amount of sea water and near-drowning. Maybe it is a sort of halfway point to near drowning. Maybe it is an allergic effect due to inhalation of material from sea water like plankton or sea water proteins of some description or other. Or maybe it is just a chemical pneumonitis due to inhalation of hypertonic saline.

I have got a sneaking feeling that this syndrome is a lot more common than we think it is. I have a feeling that there are divers and swimmers who are presenting to Casualty Departments and GP's surgeries and perhaps even Intensive Care Units complaining of symptoms and signs along these lines, who are being successfully treated with antibiotics. I would remind you that the salt water aspiration syndrome is self-limiting, it gets better whether you treat it or not but it gets better much quicker if you treat it with 100% oxygen.

THE CEREBRAL SEQUELAE OF DROWNING

Peter de Buse

One of the things that has concerned people for a long time when dealing with children who drown is the likely outcome. Now a number of children will be placed on ventilators for some time, with paralysis and thiopentone infusions which make it impossible to assess their state of consciousness during treatment. The parents are naturally very anxious to know what the outcome will be. I will mention briefly some of the cerebral sequelae to drowning and then outline the Royal Children's Hospital approach to the management of drowned children and then discuss the prediction of outcome in some of the children.

The initial problem is cardiac arrest, accompanied by apnoea. Hypoxia is not necessarily all that much of a neuronal insult. The idea that 3 or 4 minutes of hypoxia is the cause of the subsequent central nervous system deficit is very much in doubt. Animals which have been ventilated with gases of very low oxygen content but with their cerebral perfusion maintained have been judged to be normal after some 60 minutes of what we would consider to be hypoxia. Following hypoxia, very often because of the effect of the hypoxia on the myocardium together with the neuronal insults there is a problem of subsequent cerebral perfusion and oedema. The cranium is only some 5% greater in volume than the brain. If there is any oedema then the rigid skull makes sure that the intracranial pressure goes up. The effect of hypoxia and drowning on the cell, must be considered as well. The magnesium dependant ATPase system, and the sodium dependent ATPase system, result in an influx of sodium with an efflux of potassium and this is accompanied later by the influx of calcium ions into the cell. There is currently interest in the calcium blocking drugs in relation to hypoxic cerebral insults. Some people would suggest that the anoxic ischaemic insult leads to a massive influx of calcium ions into the cell, that mitochondrion uncoupling occurs at that stage and again because of the entry of calcium irons into the cell, there is an activity of phospholiphase and with the release of free fatty acids, particularly arachidonic acid and that this has an effect on the subsequent perfusion of the brain. In animal experiments there is a very good correlation between the arterial supply of the brain and the boundary zones at which neuronal damage occurs. Following the continuing insult that takes place after hypoxia these may be prostaglandin release and thromboxane release which with effects on platelets and small vessel activity may give rise to an increase in cerebro-vascular resistance. The calcium damaged cells, with the release of superoxide free radicals, may also be related to the increase in myofibrillar spasm.

In the last couple of years the Royal Children's Hospital in Brisbane has admitted 38 drowned children. There is another series, with which I wish to compare our figures, from California, where the numbers are much the same. The age of drowning is very largely in the toddler age group. That is because of the number of domestic swimming pools in Brisbane and is perhaps related to the lack of fencing and safety measures in those pools. When the children are admitted initial evaluation is made. They can

be divided into groups whose general condition is good, the vital signs stable and who are neurologically normal, and by and large you can say that they are going to do well, whatever you do to them. In Group 2 the general condition is stable in terms of their cardio-vascular and other physiological signs, but they are neurologically abnormal and in Group 3 they are in or have recently had a cardiac arrest and are neurologically abnormal. The initial management of Group 1 is one of observation and minor fluid restriction only.

I will describe our approach to the situation in Groups 2 and 3. The children's condition on discovery, by that I mean when they were actually fished out of the water, was established by asking the older children or adults, who were concerned with their recovery, what they were like when rescued. Two of them were alert, though they had been underwater for some time. Two were considered to be blunted in their sensorium and 33 of them were comatose. The neurological condition on admission to the Royal Children's Hospital, that is the initial assessment when they came into the Casualty Department, is classified as awake (Group A) or they have some blunting of their sensorium (Group B), and in Group C they are comatose. Group C1 is when they are decorticate, in other words, they have some increase in tone but with a flexor attitude. Group C2 are decerebrate and in Group C3 they are flaccid or dead.

We would like to think that we did blood gases on most of the children but that is not so. Of the ones who had it done the moment they arrived some were not really very abnormal at all, and some are quite abnormal and still survived. Looking at the length of witnessed apnoea, from the time of discovery by the rescuers to the onset of spontaneous respiration, which may be just a gasp we can see that the majority of them have taken some sort of gasp within 5 minutes. But there are a few where there have been very prolonged periods before there has been any spontaneous effort at all, but is not necessarily associated with fatal outcome. Comparing the neurological status on admission with the period of witnessed apnoea shows that those children who had had prolonged periods of witnessed apnoea were in a rather worse neurological state but they do not correlate all that well.

The results of the experiments in animals that I mentioned earlier do not necessarily parallel the human state. So we have adopted the attitude that the damaged brain needs both oxygen and glucose and that we will try to minimize the needs of oxygen and glucose and will try to maximize the delivery. We have adopted the HYPER protocol that was made popular through the Pediatric Clinics of North America in 1979. We seek to normalize hyperhydration, hyper-ventilation, hyperpyrexia, hyperexcitibility and hyper-rigidity.

Most of the children who have been admitted have been hypothermic. There was only one who had a normal temperature. The average temperature has been about 35°C. That is probably related to the sea temperature and swimming pool temperatures in Brisbane which never, even in winter, are very low. We have not maintained hypothermia, as some people have felt should be done, because we feel that the effect of prolonged hypothermia below about 34° or so is very much bound up with suppression of the immune functions of the child. Our attitude has been to make no active efforts to warm the child but allow it to rewarm spontaneously. Extreme hyperthermia has sometimes been a problem and we have used chlorpromazine to deal with it.

We have maintained them at between one-third to half their normal fluid requirements. We aim at a serum osmolality of 305-310 in response to that fluid restriction. It is important when considering cerebral oedema and raised intracranial pressure to try to decide whether one is specifically trying to deal with oedematous tissues, and that probably is not all that easy to do, or whether one is non-specifically trying to lower intracranial pressure, by shrinking the blood volume and reducing brain water content, which is what we commonly do with diuretics and dehydration or whether one is attempting to prevent or mitigate the formation of oedema. Those people who give steroids in a dose of 0.1 mg per kg., four hourly, are presumably using that particular principle. Cerebral pressure monitoring has been part of our regime in the last few children that we have had. Our observations have been in keeping in with the work published from California. Other workers have shown that a cerebral perfusion pressure, that is the mean systolic blood pressure less the intra-cranial pressure, of less than 50 mm Hg was always accompanied by a poor outcome and the same was true for an intracranial pressure that was over 20.

We have used barbiturates. Our regime has used an ariaesthetic initial dose of 5 mg per kg and then between 1 and 4 mg per kg an hour. There are plenty of people who would use between 5 and 10 mg per kg an hour. It has been suggested that some of the neuronal effects of calcium influx may have been ameliorated by barbiturates. A recent report in the Annals of Emergency Medicine gave results of 10 dogs who were put into cardiac arrest and apnoea. During resuscitation, five of them received no calcium channel blockers and five of them were given an effective dose. The neurological status of the second group was definitely superior to that of the ones who were not given calcium channel blockers.

In our series all those children who are awake or had minor blunting of their sensorium did well and the children who did badly were predominantly in the decerebrate or flaccid group. The Californians had a rather more scattered group in the comatose group and they had rather more deaths and with a rather larger number with persistent deficit. There is a very wide variation between the various reported series. Of course some of them come from places where the water is cold and some where the water is quite warm, but it is still very, very difficult to predict the outcome of children. Very few factors are significant for prediction. Age and sex are not significant. pH on presentation does not seem to have all that much bearing on survival, nor does the temperature on presentation. If the child was virtually dead on presentation, that was very strongly significant. So was the presence of fixed dilated pupils. Although it is always put forward that if they have been fished out pretty quickly, you can say to the parents that the child is going to be alright, that is not necessarily true at all.

Other things are not significant either. In our hospital, we have taken the time to the first gasp being below 20 minutes as a predictor. I have my doubts about that being a very valuable predictive factor. We elected to ventilate one, a child who at the initial evaluation was on the border line between being blunted in sensorium and being comatose. The child had a cardiac arrest from its endotracheal tube becoming blocked during the middle of the night. That child subsequently was a really very grossly physically handicapped and mentally damaged child. I wonder if we had not intubated her and had left her alone, whether she would have been alright.

Dr Peter de Buse is the Director of Intensive Care at the Royal Children's Hospital, Brisbane.

DISCUSSION

Chairman, Dr C Acott
I would like to comment on Dr Knight's introductory remarks. People who come from the higher latitudes and far south seem to think that it is always warmer up here. I did. When we had a conference here in July everybody came up in their shorts and shirts and they sat around the fires all night. The water temperature at which people can maintain their body temperature in water, is 72°F which is about 24°C. The water temperature at Yeppoon in July is 21.5°C. The map issued by the Department of Transport in one of their safety education articles shows Townsville as being the latitude at which people can maintain their body temperature in the water without aids such as extra clothing in winter, for 12 hours. That means in practical terms that if you fall into the drink just before or just after sundown, which is about the most likely time, and if you get found after daylight next day which is about when you can hope to be found, if you are lucky, you are likely to be either dead or fairly close to it in this latitude in winter time.

Dr J Knight
I would not disagree at all. It is a fallacy, and a widely believed one, that warm water does not induce hypothermia. There are people in this room who would undoubtedly survive much longer than others. It depends on your insulation and your heat output. There are obvious survival differences in shape. This is shown in fell walkers, people out getting hypothermic from exposure to wet windy conditions, as well as in people falling in the water. It really means that you have got to choose your genes and your cook very well if you are going to spend a long time in the water unexpectedly.

Dr Vic Callanan
How much water do you need to aspirate to get the salt water aspiration syndrome? I suspect that if they are obtaining their water in a nebulised form, and these people are in the water for some time, they could get into their lung quite considerable amounts of water. I wonder how much hyperhydration may play a part in this giving them combined cerebral and pulmonary effects.

Dr B McKenzie
I do not know how much water you need to inhale. We have seen cases of aspiration syndrome where people have developed it after one inhalation event. With buddy breathing for instance, they have received a demand valve full of water and they have inhaled a bit of it. The demand valve does not hold much water even if you inhale the whole lot. No research has been done on this so we do not know, but I suspect that the volume of water can be quite small to provoke the syndrome although it can be large as well.

Dr J Knight
As somebody who has had this syndrome using Navy equipment, buddy breathing, it is not much water. We were passing the regulators flowing, with them upside down so the water would not get in. We had a lecture about salt water aspiration syndrome that morning and we did our buddy breathing in the afternoon. We did occasionally fail to have it flowing. I shivered all night and came down next morning and announced that I had the salt water aspiration syndrome. Funnily enough two other doctors who had been to the same lecture turned up and said "I had a bloody awful night, I was shivering all night, I feel sore as anything. I wonder what is the matter with me?"

Dr A Holloway
Did the Navy do water samples for bacterial count and did you do microscopy of what they coughed up, looking for things like E coli in water and algae in the lungs? Around the coast of Southern Africa, chills rigors and infection in near-drowned children correlated with currents, sewerage flow and bacterial colonisation of water.

Dr B McKenzie
I would believe the contamination theory, especially in Sydney because they dump raw sewage into the water. It is probably on the cards. We never analysed the water. Very little research has been done into it, we just do not know. I was thinking of doing a project making some artificial sea water or ultrafiltering sea water and getting people to breathe that and see if that provoked the syndrome. Sometimes they had eosinophils in their sputum but we did not do cultures on their sputum. The thing against the bacterial contamination theory is that we have seen it in other parts of the world and in other parts of Australia where there is clean water. If it was just confined to Sydney I would say that it was sewage inhalation.

THE ISRAELI NAVAL HYPERBARIC INSTITUTE

Yehuda Melamed and Dan Kerem

HISTORY AND DEVELOPMENT

Since its foundation in 1973, the Israeli Naval Hyperbaric Institute (INHI) has seen a period of steady growth and development, becoming what is now virtually a national institute of diving and hyperbaric medicine. Although the factors which have contributed to this continuous expansion in services and facilities are many and varied, the basic reasons behind it are those encountered the world over. There was an initial stage, with a demand for meeting immediate needs, with a subsequent, and developing, interest in investigating the possibilities of further applications and innovation.

It was in the early 1960's that the Israeli Navy installed the country's first multiplace hyperbaric chamber at one of its bases. At this stage, the unit's primary function was to treat Navy diving casualties. On occasions it was also used for treating civilians involved in similar incidents and for indications other than diving accidents.

The early 1970's witnessed a vast expansion in civilian diving activity in the waters around Israel. The number of active divers increased over a short period of time from a few hundred to many thousands. Unfortunately, this was accompanied by a steep rise in the number of civilian diving accidents. During this period too, there was a move on the part of major oil companies to extend and expand their activities in the Suez area. It became evident that to cope with such a situation, steps would have to be taken towards concentrating, expanding and coordinating expertise and facilities in the fields of diving medicine and physiology and diving safety. The necessity was also seen of attaining a level of research which would make it possible to answer specific questions and solve basic problems in these spheres.

It was the general consensus of opinion at the time that, since it already had the equipment and manpower required to initiate such a scheme, the Israeli Navy was in the best position to confront the problem. Seeing in the project a matter of national importance, the Navy undertook to organise all activity in the field.

During the early period of its development, the INHI operated the two-lock hyperbaric chamber which had been installed by the Navy ten years previously. The Institute was occupied mainly with the medical selection of diving personnel for the Navy, as well as with treating diving accidents from both the Navy and the civilian population. There was, in addition, a rather limited amount of research, conducted mainly on animals in a converted monoplace chamber.

The INHI's aims and goals were defined as

1. The medical selection and periodical examination of Navy personnel, including submarines, involved professionally in underwater activities.
2. To serve as a national treatment centre for diving casualties and medical emergencies requiring hyperbaric treatment.
3. To serve as an information and professional advice centre for all persons in Israel involved in the field of diving and hyperbaric medicine.
4. To conduct research in the field of diving and hyperbaric medicine.
5. The training of medical staff (doctors, corpsmen, technicians, etc.) to provide diving and hyperbaric medical treatment in the Navy and for civilians.

THE PRESENT

In the years since 1973, the INHI has been engaged in a gradual expansion programme designed to enable it to pursue its stated aims to the full. These efforts are reflected in the growth and change in the two fundamental resources available to the Institute, its manpower and its basic facilities. The present staff of the INHI includes physicians, senior physiologists, laboratory and chamber technicians, corpsmen, a librarian and a specialist in speech and hearing disorders. The Hyperbaric Physiology Research Unit, fulfilling a major aim of the INHI, functions as what is in essence a separate entity at the Israel Oceanographic and Limnological Research Institute, while the basic facilities at the Institute have also undergone modification and development.

TABLE 1

STAFFING LEVELS

INHI PERSONNEL

Physicians	3
Administration Officer, (Senior Corpsman)	1
Medical Secretary	1
Director, *Library & Information Centre*	1
Secretary	1
Audiology Officer	1
Technicians	3
Corpsmen	2

HPRU PERSONNEL

Physiologists (Ph.D.)	3
(One physiologist at present on one year's unpaid leave)	
Technician	1
Laboratory Technicians	2

FUTURE DEVELOPMENT PLANS

Additions to be made in the near future:

Physicians	2
(One for the HPRU)	
Technicians	2
Laboratory Technician	1
Corpsman	1

The Hyperbaric Chamber

The INHI now has a Drager three-compartment hyperbaric chamber, capable of accommodating six patients at a time on beds, or up to twenty occupants in a sitting position. The chamber is equipped with a mechanical respirator, suction apparatus and temperature control, while CO_2 and O_2 analysers complete the atmosphere monitoring system. Patient monitoring capabilities include closed-circuit TV, ECG, EEG and respiration. Oxygen is supplied to occupants via oronasal masks, with overboard dumping of expired gas. The chamber has a working pressure of 100 metres of seawater (MSW). It can be compressed with either air or nitrogen/air mixture. As it has a CO_2 scrubber, the chamber is capable of nitrox saturation dives. Air compressors, compressed gas banks and an Electricity generator make the chamber entirely self-sufficient.

The Audiology Laboratory

The INHI's audiology laboratory is equipped with an audiometer, a tympanometer and noise dosimeters. More sophisticated examinations such as brain stem auditory evoked response are carried out by the audiologist at the Rambam Medical Centre nearby, or in the Behavioural Biology Laboratory at the Technion (the Israel Institute of Technology).

The hyperbaric chamber and the audiology laboratory are the units which serve the two primary and most basic aims of the INHI, those of diver selection and medical treatment.

Diver selection

After the initial selection process, all divers in the Israeli Navy undergo periodical medical examinations. These are carried out by the INHI staff with the assistance of ENT and Ophthalmology specialists, who are permanently assigned to the Institute as part of their reserve duty.

Medical treatment

INHI staff and facilities are on call 24 hours a day, 365 days a year. One physician is available immediately for telephone consultation, and the chamber can be in operation within 30 minutes.

A system of rapid transfer-under-pressure has been developed for the treatment of diving accidents, and has been successfully employed since 1975. There are monoplace recompression chambers situated at the more important diving locations all along the coasts of Israel, both at Naval bases and civilian diving clubs, mainly at those in the Red Sea area. The two multiplace hyperbaric chambers at present available, one at the INHI and another at Yoseftal Hospital in Eilat in the south, are shortly to be supplemented by a third, two-compartment chamber which will be installed at the INHI. All of the monoplace chambers, manufactured by Drager of Germany, are fitted with oxygen masks and equipped with a mating collar fitting the multiplace chambers. In this way, the patient may easily be transferred under pressure from monoplace to multiplace chamber.

Once a suspected diving accident victim reaches one of the monoplace chambers, he is quickly examined by the person in charge (physician/corpsman at Naval bases, or diving instructor at civilian establishments). In severe cases treatment may be commenced at once. At the same time, the duty physician at the INHI is contacted and expert advice obtained as to treatment guidelines.

The monoplace chambers are employed as a means of achieving rapid recompression and transferring the victim under pressure. As a rule, no attempt is made to complete the therapy in the monoplace chamber, which is transported instead by helicopter or vehicle to the nearest multiplace facility for continuation of treatment.

Patients who require hyperbaric oxygen (HBO) therapy for indications other than diving accidents are transferred to Rambam Medical Centre, and receive HBO treatment as required at the INHI, where up to four patients may easily be accommodated at one time. The most common indications for which HBO therapy is administered are clostridial infections, combined synergistic infections, cerebral air embolism due to surgical, traumatic or iatrogenic causes, CO intoxication, traumatic peripheral vascular ischaemia, crush injury, etc. The INHI was recently officially recognised by the Ministry of Health as a National Clinical Hyperbaric Unit associated with Rambam Medical Centre in Haifa.

The three remaining aims are served by what are essentially three self-contained and self-sufficient units, the library, the Hyperbaric Physiology Research Unit and the educational programme.

Library and Information Centre

The INHI's modern library and reading room is entirely devoted to diving and hyperbaric medicine and physiology. It serves both military and civilian institutions, and is administered by a full-time librarian. Access to computerised information retrieval systems in Israel and the USA, as well as to large libraries in Israel and abroad, enables it to keep permanently up to date and to conduct literature searches on request. This centre, which incorporates lecture theatre facilities, provides the academic and information services so vital to the Institute's everyday medical and scientific work.

The Hyperbaric Physiology Research Unit

Background

The INHI's Hyperbaric Physiology Research Unit (HPRU) operates at the nearby Israel Oceanographic and Limnological Research (IOLR), which is the national Oceanographic Institute. This situation is the result of efforts to merge national research facilities and manpower in order to achieve the "critical mass" required for a viable research program. Apart from these institutions, the Faculty of Medicine at the Technion (the Israel Institute of Technology), serves as an academic authority. It supplements the research team by providing students studying for various degrees, contributes research equipment and services such as animal care, computing, photography, etc., and most important, provides a scientific milieu for the research team.

Research policy

Being a national multi-institutional unit, the HPRU serves as a research facility answering the needs of the national diving community according to a long range program prepared and periodically re-evaluated by a scientific committee. The unit cannot and does not profess to compete with the leading research laboratories in the world, but holds that in order to receive, interpret and apply research results from abroad, it should have a diverse and active program of its own.

At the same time, the unit recognises the Navy as the current main user of its research efforts and the major contributor of funds. Therefore most of the research, as well as routine and short-range training and evaluation activities, is conducted in response to specific Navy requirements.

Facilities

The unit operates a two-compartment research chamber (fitted at Duke University Medical Center Hyperbaric Laboratory) equipped with a wet compartment and rated to 10 ATA. An electrical bicycle ergometer, a breathing simulator and extensive physiological monitoring equipment allows thorough investigation of diver response, as well as diving gear performance, in actual diving conditions.

In addition, the unit has three animal chambers rated to 10, 25 and 120 ATA (the latter being dry/wet) offering a wide range of pressures for hyperbaric testing. It has modest animal operating facilities and a fairly well equipped respiratory laboratory.

Areas of research

In the past, in order to acquire some skill in the many areas of hyperbaric research, diverse topics such as experimental decompression sickness, theoretical models of bubble resolution in tissues, hydrostatic pressure effects on isolated tissue and on breathholding vertebrates, various aspects of pulmonary and central nervous system oxygen toxicity and pulmonary function in the hyperbaric environment have all been subjects of research projects. More recently, a long-range research plan has been formulated which puts emphasis on two main research topics, ie. oxygen toxicity and the respiratory system in the hyperbaric environment. In the first, animal models are used to test various approaches to the prevention or delay of the toxic symptoms. In the latter, alterations in pulmonary mechanics, gas exchange and regulation of respiration are studied with the aim of coaching divers to use, intentionally, an optimal breathing pattern under a specific set of conditions.

Physiological tests for divers

These tests are intended for purposes of large-scale screening, selection for specific diving activities, periodic checkups and accident investigations. The tests, most of which are run inside the wet compartment of the experimental chamber, include measurements of central responsiveness and ventilatory reaction to CO_2 and to graded exercise, as well as steady-state body CO_2 levels during hyperbaric exercise.

Diver training

This activity is intended to have divers experience, under well controlled conditions, various stress situations which they may encounter during actual diving. The training allows the diver to develop early recognition of these situations, to realise his personal ability to cope with them and his tolerance limits, and finally to learn how to avoid them or how to arrest them once they have arisen.

Equipment evaluation

Tests are carried out on new, modified and post-accident diver breathing apparatus. The evaluation includes physiological monitoring of parameters such as breathing resistance and work of breathing under standard conditions created by a breathing simulator, as well as human tests for objective and subjective assessment of equipment-diver combinations.

Instruction of diving and medical personnel

The INHI provides regular training in diving medicine, physiology and safety for Navy corpsmen and divers and civilian diving instructors. At least once a year, a two-week course in diving medicine is held for physicians, and being the only such course available in Israel, it is also open to civilian doctors. Graduates of the course are recognised as diving medical examiners by the Federation for Underwater Activities in Israel. Students at the Technion Medical School in Haifa may take an elective course in hyperbaric physiology and medicine at the Institute. They may also prepare their theses for the degrees of MD, BSc, MSc and PhD at the Hyperbaric Physiology Research Unit belonging to the INHI.

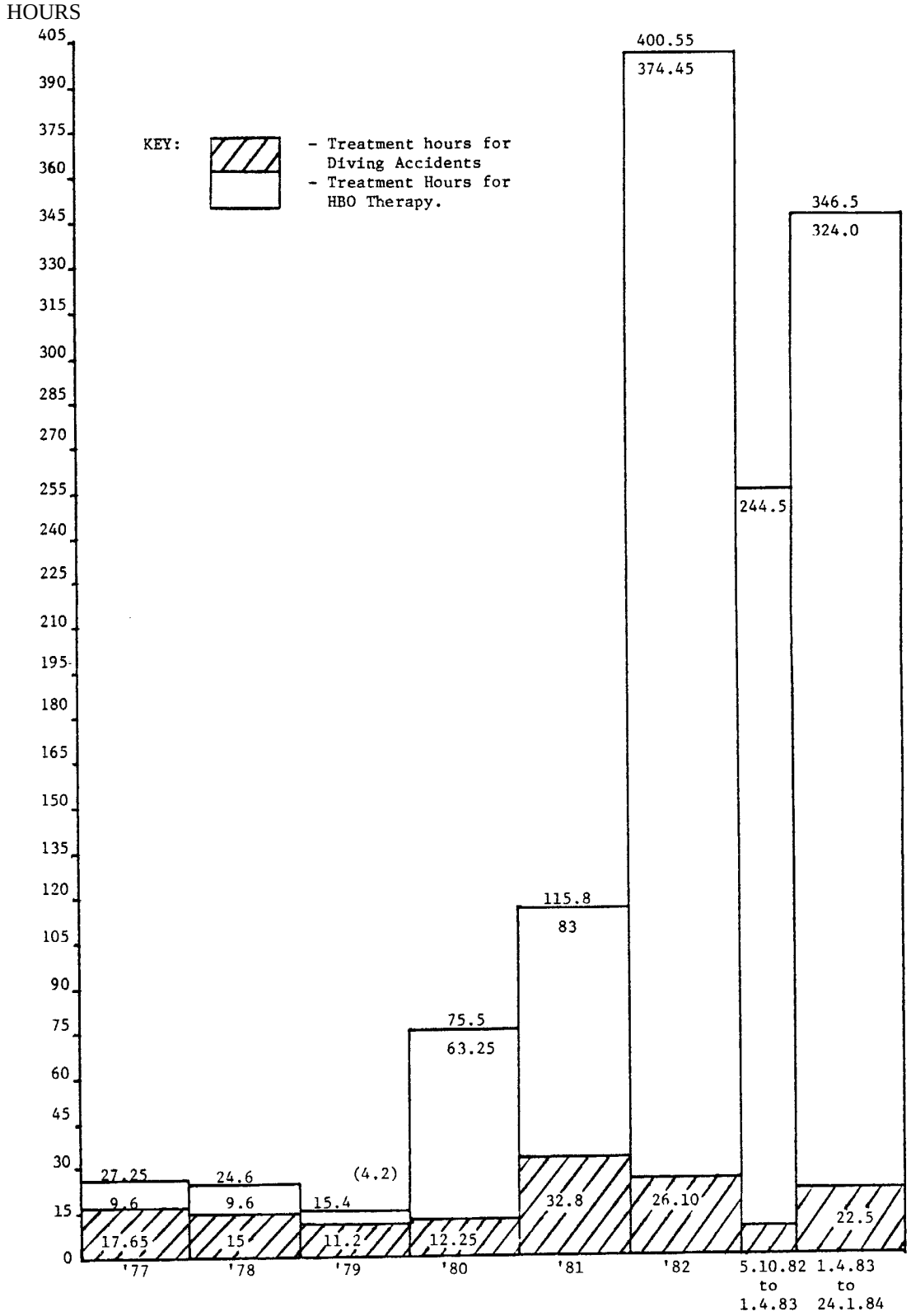
The INHI's future is one of continuing growth and development. It is expected that a further dramatic expansion in the sphere of clinical hyperbaric medicine will occur when the Institute's additional two-lock chamber is installed in a specially constructed extension to the present building. This will enable a broader range of indications to be considered for treatment, and make it possible to provide this treatment as a matter of routine. As hospitals throughout the country become increasingly aware of the applications of hyperbaric therapy, more of their patients will be referred to the Institute and be able to receive the treatment they require.

As new research workers join the team presently working at the HPRU, it will become possible to engage in more academic research rather than the specific and more practical work being done at the moment. This is important both for the researchers themselves and for maintaining high academic standards.

This increase in manpower will also facilitate expansion in the educational services provided by the Institute. The impetus of this educational effort will be directed towards medical students from Jerusalem, Tel Aviv and Be'er Sheva, who will join the Haifa medical students already participating in the program. A greater number of civilian doctors will also be able to take part in these courses.

TABLE 2

TREATMENT HOURS FOR HYPERBARIC OXYGEN THERAPY AND FOR DIVING ACCIDENTS 1977 TO 24.1.84



This table illustrates the increase in the number of treatment hours devoted to hyperbaric oxygen therapy, particularly during 1982 and 1983, in contrast to the almost unchanged treatment hours devoted to diving accidents. This is due to an increased awareness on the part of physicians and hospitals of the possibilities of HBO treatment.

Consequently we expect that in the near future the Institute's hyperbaric chambers will be in operation on a routine basis, giving hyperbaric oxygen therapy to patients from all over the country, while the number of treatment hours for diving accidents will remain low.

The Israeli Naval Hyperbaric Institute is in a constant state of development, broadening the field of its activities in clinical medicine, research, and all the support facilities required to keep the Institute up to date with the latest developments in diving and hyperbaric medicine, and to push it forward into the future as an innovator in its own right.

The postal address of the Israeli Naval Hyperbaric institute is

PO Box 8040,
Haifa 31080,
ISRAEL.

Department of Energy,
Petroleum Engineering Division,
Thames House South,
Millbank, London, SW1P 4QJ

18 April 1984.

DIVING SAFETY MEMORANDUM NO 3/1984
DIVING SAFETY INTERLOCKS

The Norwegian Petroleum Directorate Safety Notice issued in Diving Safety Memo 2/1984 drew attention to the need for safety interlocks in diving systems. This memo draws attention to regulation 12(4)(a) of the Diving Operations at Work Regulation 1981 with particular emphasis on the need for interlocks on pressurised systems.

In addition to company safety procedures diving systems are to be fitted with safety interlocks where necessary to prevent any unintentional or uncontrolled loss of pressure. Particular attention should be directed to chamber/bell mating systems, diver evacuation mating systems, food and equipment locks. (Where interlocks are not fitted at present this should be done before July 1st 1984).

Internal communicating doors in the TUP chamber should be shut and a seal obtained when bell mating or unmating or transfer of personnel or equipment is taking place. These doors should not be opened again until such time as the internal door between the transfer chamber and the transfer trunk has been shut.

18 April, 1984.

DIVING SAFETY MEMORANDUM NO 4/1984
DIVER CERTIFICATION: TRANSITIONAL CERTIFICATES

Some doubt appears to exist as to the validity of transitional certificates issued by the diving contractors in accordance with regulation 15 of the Diving Operations at Work Regulations 1981 and the length of time that these certificates remain valid.

Transitional certificates remain valid for as long as the owner wishes to use them unless, of course, they are revoked by the Health and Safety Executive. There is no intention at present to cancel transitional certificates.

Some contractors prefer divers to hold an HSE certificate. Divers who hold transitional certificates who wish to obtain an HSE certificate should apply in writing to the Health and Safety Executive, SP1 SC2, Regina House, 259 Old Marylebone Road, London, NW15RR.

The diver may be asked to provide evidence of his competence.

SA WARNER
Chief Inspector of Diving

REFRACTIVE ERRORS AND THEIR CORRECTIONS FOR DIVERS

FJ Geddes

All of us have had the experience, when diving without a face mask, of blurred vision underwater. With the use of a mask, everything becomes clear again due to the refractive difference of air and water. That is, for most of us. But what about the diver who has an optical correction? The mask is a help, but only to a certain distance, and then all becomes blurred again.

What can be done? The choices are to use either contact lenses, or stick-on spectacle frames with lenses inside the mask, or ready-made prescription lenses to fit masks such as the Tabata mini, or full prescription lenses laminated to the inside of the face plate glass. There are advantages and disadvantages with each method.

Contact lenses

These, whether hard or soft, are an excellent way of correcting vision underwater. Soft lenses seem to be more resistant to “float out” and are usually easier to wear. Peripheral vision, while the lenses are in, is first class.

The disadvantage is cost. They usually cost about $140-$180 per pair, and in the event of the mask flooding, “float out” is just one more problem for the diver who may have his or her hands full at the time.

Stick-ons

This method is quite cheap. Providing the rubber suction cups and/or glue hold, it is a good aid to vision. An old, or spare, pair of spectacles can be utilised by this method.

The disadvantages are that three glass surfaces must be kept clean and droplet free, and the field of vision can be restricted by the spectacle frame.

Ready made lenses

These lenses are really only suitable for basic low-power, non-astigmatic prescriptions. The top power used should be not more than -3.00 dioptres. Beyond this power, distance, pupil centres, and lens centres become more critical. Ready made lenses, being set into the mask at a standard distance apart may not correspond with the divers

inter-pupillary distance. The range of masks available is quite small with little chance of fitting the non-standard diver who has difficulty getting a good seal on some masks. Cost is not too bad, averaging $40-$50, plus the cost of a new mask.

Lenses laminated to the facemask glass

Finally, purpose made lenses offer the widest range of any of the four methods. Positive and negative, as well as astigmatic correction can be made. Bifocals are also available. Lens centres can be controlled precisely and the diver has a much larger range of masks to select from, the only requirement being that the glass face plate must be of good quality and readily removable from the rubber surround.

Lenses are ground flat on the front with all the prescription on the back surface. They are then cut to the required shape and laminated to the face plate with a special adhesive. With the correct adhesive, the bond is permanent even though some slight yellowing may take place if the mask is left in strong sunlight for long periods of time. Lens delamination does not seem to be a problem, as, in 20 years my firm has had only one mask returned to us for re-fitting. The cost of these lenses averages $60 per pair, plus the face mask.

NEW PRODUCT INFORMATION

DECUPAD THERMAL RECOVERY CAPSULE

The following information has been provided by the agents for the Decupad Thermal Recovery Capsule (TRC). Three test reports (three volunteers) issued by the Textile Department at Leeds University were used and two letters from users were provided as evidence of the effectiveness of the use of the TRC.

It was developed in conjunction with the safety and medical officers of virtually all the major offshore companies, air/sea rescue, diving companies and other interested parties working out of Aberdeen, Scotland. It has been Home Office tested and is used by Coneco, Texaco, Total, Mobil, British National Oil Corporation, Shell, BP, etc as well as off Alaska, Nova Scotia, Newfoundland and other areas. Land based UK services also have TRC's available.

The TRC is designed for the prevention and treatment of hypothermia in a cold or wet environment. It is claimed to be the one single piece of equipment most likely to ensure survival of a hypothermic patient in an isolated situation, and is convenient to transport and use. The principle behind it is that if the heat produced by the metabolism of the body, even in a hypothermic patient, can be retained, rewarming will occur. This will occur in all except the most severe cases, and even in these its use will be protective and improve survival chances during transport to hospital. Many severe cases may appear to be dead but be revivable through such management. Total packed weight is approximately 10lbs and it measures 30" x 12", so is easily transportable. A weather resistant, bright orange carrying case is provided. The unit is reusable after cleaning.

In appearance it is like a sleeping bag which opens down one side and totally encloses the patient except for an opening for the face to show. It can be opened to allow access to small desired areas as needed. It is effective even with a wet patient, there being no need to first strip the victim. The depth of the pile and the material used ensures that there is no loss of insulation due to the water.

The cost of $A575 seems to be high but for expeditions, including diving, where hypothermia is a significant risk factor, it is cheaper than a funeral. Local agents are:

Superior Sportswear Pty Ltd,
PO Box 40,
TOONGABBIE, NSW 2146.

TWO "CROOK" TANKS

In Moline, Illinios, this May a scuba tank "took off just like a rocket", ripping through three walls and injuring two employees of the dive shop. The tank had been brought in for a hydro test and an employee had opened the valve to allow the tank to empty. Two days later two of the employees began to unscrew the valve to begin an inspection. When the valve had been partially unscrewed, air started to escape and the tank took off like a jet-propelled rocket. The valve went one way, knocking a heavy metal door from its hinges, and the tank went another. It travelled 35 feet, passing through three walls. The tank had not been hydro tested since 1976. It was concluded that the tank had been stored full of air and had contained some water, the resulting rust having fallen into and blocked the valve while the tank was upended to drain for the two days. This had prevented the venting of air when the valve was turned to the open position. Had the tank been stored with roughly 500 psi air pressure remaining in it, as is usually recommended, it is unlikely that the accident would have happened.

In Dublin recently the explosion of a tank under test resulted in a shower of cannabis resin all over the room, sticking to the floor, walls, ceiling and an employee. It also alerted the police to a hitherto ignored theft's importance. The dive shop operator had been suspicious about this tank, one of a batch left for testing at one of the shop's branches, as it seemed to be too small to qualify as an 80 cu ft cylinder despite its USA markings. Its inside measurements seemed to be less than expected. It was handed over to another test shop for a second opinion on this apparent discrepancy. There it was connected to a compressor, and exploded a few minutes later. It was one of a pair and the second cylinder, on checking, revealed that at some previous time the tank had been cut and threaded and the cannabis resin then packed like a doughnut around an inner cylinder which contained the air. Evidently the resealed cylinder had sufficient strength to stand an ordinary "fill" but insufficient to take the pressure on this occasion. It is not known whether the owner had used it much since purchasing it.

Investigation revealed that the police had staked out Dublin airport two years ago in response to a tip that cannabis was being smuggled into Eire in scuba tanks, but they failed to realise that the empty tanks had false internal capacity. They also, correctly, regarded the theft of the tanks as an ordinary theft, typical of what happens to unattended goods at an airport. The tanks were later sold to an unsuspecting diver. The thief was therefore now sought by the police with more zeal than is usual for those who purloin scuba tanks, and the original owner of the tanks is being offered the chance to assist the investigations. One word of warning, the Eire police and customs know this trick now and it reduces the tank's capacity cruelly.

Information Source: *UNDERCURRENT*, May and August 1983.

UNUSUAL INCIDENTS FROM THE PAST

Number 5 1963

A Reserve Ascent

Readers may be interested to read of an ascent made from 135 feet off Point Perpendicular, Jervis Bay, recently by a member. I think it is a trick worth knowing and I am sure everyone will commend the presence of mind of the third-class diver concerned, Robert Bennet of East Roseville.

Six men went straight down to bottom and gathered for a check before moving off. RB's demand valve started to blow. Quick breaths failed to reseat the valve and in a short while he was down to his reserve.

He gave the 'out of air' signal (instead of the 'on reserve' signal) and started for the surface fast. The dive leader, Gunter Grafenauer, well known as a crack diver both in Sydney and in Wollongon (sic), had him back on the bottom quick as lightning and, in view of his signal offered him his mouthpiece. RB refused this - he had, in the moment of danger evolved the answer. When he wanted air he pulled his reserve, then shut it off. The whole party made a controlled 25 feet per minute ascent without further trouble, RB with air to spare.

Australian Skindivers Magazine. July/August 1963

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

BLACKIE THE CROOK CROC AND OTHER SAURIANS

Taking the police sergeant's dog was a mistake, but when you are 4.5m long and carry your tonne weight about on 3 and a half legs people usually pass over minor character imperfections. Initial attempts to catch him failed because younger, faster crocodiles reached the bait first, but after 12 days he was caught and carried back by ten good men and true to Mamaga and Justice.

Until he could be flown out to the Cairns "Australian Bird Park" (well, he was to be flown there!) he had to be lodged in the jail, the only building large enough and strong enough for this purpose. Until he arrived the Bamaga jail had only two "guests". One was an habitual drunk. He sobered up pretty quickly when Blackie arrived. The other was a voluntary mental patient. He was moved to a different part of the Jail and thoroughly enjoyed waving to the stream of visitors coming in to see Blackie. "The place is like a circus" the police sergeant stated.

Since his capture the crocodile experts in Cairns have been deluged by reports of monster crocs devouring cattle. Which makes a change from the "Top End" stories of the mozzies carrying them off. Blackie, of course, has an unbreakable alibi for such present troubles.

Meanwhile, up in Cape York Peninsula, Professor Gordon Grigg of Sydney University is trying to find out what crocs do when alone. Apparently the brutes are impossible to view without their being aware of the observers. He has 40 crocs in a tank at the University and "they don't act natural". He has an instrument pack to measure their breathing habits and dive times and a problem with getting co-operation. Custom made (well, the market is rather limited) neoprene suits were procured and twelve 3m crocodiles were captured and dressed. When eight were recaptured eight days later all but one had managed to wriggle out of their jackets. Evidently crocs prefer to dive in their birthday suits. Back to the drawing board!

His other research interest was the tolerance levels of crocodile eggs to flooding. They are laid in nests near the river banks during the wet season and suffer a high mortality rate, too high under present circumstances. Professor Grigg finds crocs fascinating and claims "they've got everything going for them. They are very interesting creatures". When it comes to understanding them, it seems that crocs in a tank are less good than a croc in the bush.

PORPOISES DO IT AGAIN!

A fisherman whose four-metre boat was swamped and sunk by the waves in the Atlantic claimed later that he owed his life to porpoises. "There were sharks in the water but the porpoises were all around me and stayed right beside me." He swam and floated for 12 hours before being rescued and taken to the Memorial Medical Centre, Jacksonville, Florida to be treated for exposure.

Reprinted from the Daily Telegraph, 27 May 1983.

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.

The subscription for Full Members is $30.00 and for Associate Members is $20.00.

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,
Queensland, 4700.

NOTES FOR 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 photo reduction direct. Books, journals, notices for symposia etc., will be given consideration for notice in this journal.

DON'T ALWAYS FOLLOW THE INSTRUCTIONS!

A notice in the Warrenton Democrat in Virginia read: "If you are one of the hundreds of parachuting enthusiasts who bought our 'Easy Skydiving' booklet, please make the following correction: on page 8, line 7, the words 'State zip code' should read 'Pull rip cord'."

One presumes they added a hope that nobody was inconvenienced by the error.

SPUMS ANNUAL SCIENTIFIC MEETING 1985

The venue will be the Maldives Islands. In order to get the best diving weather the meeting will be held in late April. The monsoon makes the weather unpredictable, and wet, after that.

There is an international airport in the Maldives. Regular flights from Australia to the Maldives, via Singapore, have recently started cutting out the need for an overnight stop in Columbo. It is proposed to use these direct flights for SPUMS.

As further details come to hand you will be kept informed.

CONFERENCES

THE 4TH WORLD CONGRESS ON INTENSIVE AND CRITICAL CARE MEDICINE

Will be held in Jerusalem from 23rd to 28th June 1985.

It will be followed by:

AN INTERNATIONAL SATELLITE SYMPOSIUM
ON HYPERBARIC OXYGEN
IN CRITICAL CARE MEDICINE
to be held in Eilat from 30th June to 2nd July 1985.

The Chairman, Scientific Programme, will be:

Dr Yehuda Melamed,
Director, Israeli Naval Hyperbaric Institute,
PO Box 8040,
31080 Haifa,
Israel.

TOPICS

MECHANISM OF HYPERBARIC MEDICINE
CRITICAL CARE IN THE MULTIPLACE AND MONOPLACE CHAMBER
GAS GANGRENE
BRAIN INJURY, ISCHAEMIC AND TRAUMATIC CARBON MONOXIDE AND OTHER TOXICITIES
CURRENT USES OF HYPERBARIC OXYGEN IN EMERGENCY MEDICINE
ACUTE PERIPHERAL ISCHAEMIA
POLYTRAUMA
THE CRITICAL CARE OF SEVERE DIVING ACCIDENTS
SURGICAL AIR EMBOLI
FUTURE PROSPECTS

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