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CONTENTS

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1 Anderson T, RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15 (2): 19-22
2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: J.L.Publications, 1985

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EDITORIAL

There was a tradition in ancient Rome of granting to a successful general and his army a Triumph, a march through the city complete with evidence of their success, samples of their prisoners and of their booty. There was only one minor condition, that standing in close proximity to the general in his chariot there was to be a person who frequently spoke into his ear saying "Remember, you too are mortal". In some small part the letter from Peter Horne printed in this issue (page 55) serves a similar function for all those involved in the controlling positions in diving. Undoubtedly his is not the only voice questioning the Accepted Wisdom proclaimed by those whose opinions are sought by coroners.

Such include all the "brand leaders" in diving instruction who have strictly defined training protocols and prohibit not only omissions but also additions to the course content. There are the diving medicine authorities who apply rigid standards to others and forget the slim basis for some of their beliefs. There is no formal appeal mechanism in Australia or New Zealand, unlike in the UK where medical decisions can be arbitrated. And there are the authorities, who quote the small print of regulations and ignore the totality of the events when reporting on some incident which they have investigated. It is timely for such groups to realise that not everyone everywhere and always applauds every decision they make "in the best interests of diver safety".

There is nothing to suggest that the basic factors influencing underwater safety, and indeed survival itself, are either unknown or are being misinterpreted but there is some justification for the criticism that too simplistic a view is being taken, that all recreational divers are being treated as if they were equal both in experience and ability. Despite the multitude of certificates which a keen diver can acquire by attending courses the first one is the most important in many respects because the skills which a pupil acquires in the basic course can decide later survival. Now is possibly an opportune time to consider whether these courses are adequately preparing pupils for what they will encounter. It has been demonstrated that the inexperienced are at greatest risk of suffering a diving-related death, that most fatalities occur at or close to the surface or associated with no/low air situations, and the reason could lie in the content of the training courses, the standard the divers are expected to reach, and the accuracy of their understanding of their diving ability and of their limitations.

History relates that the ancient Medes and Persians treated their laws as being immutable. This rigidity prevented them from making alterations to suit changed circumstances but did not deflect the tide of change. The diving instructor organisations, in an attempt to present an image of being responsible corporate citizens and to impress both the general public, and the litiginously inclined, with their devotion to safety, developed rigid protocols to which their members must swear allegiance or face expulsion. This is hardly conducive to developing a critical review of the results of their training. There is still, even in medical circles, a very natural caution when members of any group are asked to assess the beliefs and practices which are taken as articles of faith, so it is little wonder that there has been so little research to find out what problems divers face nowadays and to adjust training to better prepare novices to manage such problems.

Naturally this should not be taken to imply The Establishment is always wrong in what it declares to be inalienably true and that critics are always models of rectitude. It means that from time to time shibboleths need to be examined to evaluate which views are most nearly correct on some specific detail at that time.

Answering the call for a re-assessment of current diving practices and teachings will require facing two major obstacles. First is the low level of awareness of the need to question current dogma. Second, there is the task of developing an awareness of the value and importance to intelligent decision making of gathering more information about diving problems, of how deplorably small is the data base (case records) for many present day beliefs. However it is the diving instructor organisations which can best help remedy the situation as they alone have a direct access to those best in a position to supply such reports, the instructors.

It is time to question the belief that a safe recreational diver can be created by a course lasting a few days and involving even fewer dives in open water. There is need to question the value of training which stresses escaping from avoidable problems (running out of air) in contrast to courses which stress awareness (and avoidance) of all such situations. Indeed it is time to question the true dangers of having a medical history of childhood wheezing, of diabetes and of many other medical conditions. This is being undertaken in the UK but not apparently in other countries. A Pandora's Box of questions lies waiting and it is hoped to cautiously examine several during the coming year. The task is daunting and those who advise divers of the dangers of "doing their own thing" are unlikely to win any popularity awards. But the task is both worthwhile and necessary and readers are invited to provide their views. One such view appears on page 56 and another on page 61.

The papers on carbon monoxide poisoning from the Hobart Meeting make interesting reading. views against and for hyperbaric treatment of patients with this condition appear on page 71. The papers from the 1989 Annual Scientific Meeting draw attention to the dangers, which can be lethal, of running out of air and panic underwater with two case reports of very different presentations of cerebral arterial gas embolism.

SCUBA DIVING FATALITIES in AUSTRALIA AND NEW ZEALAND

2. THE ENVIRONMENTAL FACTOR

Carl Edmonds and Douglas Walker

Background

In a previous report[1] it was determined that amongst recreational Australian and New Zealand diving fatalities during the 1980s, the environmental factors contributed to 62% of the deaths. One hundred consecutive deaths, which complied with strict requirements as regards data acquisition, were assessed. The figures therefore represent both actual numbers and percentages of the total.

In 52% of the cases the environment was a probable contributing factor, and in 18% it was considered likely. In some instances there was more than one environmental contributing factor.

The environmental factors included both natural hazards (eg. tidal currents, sharks, etc.) as well as man made hazards (boats, dam outlets, etc.).

In the ANZ series we only considered the factors which materially contributed to the divers death. Because of this, such situations as fresh water diving, altitude exposure, etc. are not recorded in this series, as they *per se* do not contribute to the death.

In 47% the diver either had no experience of the diving environment being encountered, or an inadequate experience to cope with that environment. In less than 50% of cases was there sufficient training or experience for the planned dive.

Results

In the 62% of cases the following environments contributed to the deaths. The total of percentages (each 1% is one death) in table 1 is more than 62 as in some cases more than one environmental factor was present.

EXCESSIVE WATER MOVEMENTS

This was by far the major environmental problem (36 %) contributing to diving deaths.

In 15 % the tidal current was too great for the diver to negotiate. In 15 % there was rough surface conditions contributing, often involving “white water” and surging water around rocks. One of these cases involved exposure to surf. In 2 % it was a normal to and fro surge which caused the problem. In 3 % there was a sudden unexpected underwater surge which put the divers into the difficulty. Dam outlets trapped two divers (2 %).

DEPTH

Depth, which contributed to 12 % of the deaths, did not always equate with nitrogen narcosis. Sometimes the depth itself was simply too great for that person. In four cases it was considered a major factor, and in eight others, likely. In most of these cases it was the greatest depth to which that diver had dived

Table 1

ENVIRONMENTAL FACTORS

Environment	Percentage
Water movement	36
Excessive depth	12
Poor visibility	6
Cold	5
Marine Animals	5
Caves	5
Entanglement	4
Exit and Entry Problems	5
Boats	3
Diving under a ledge or boat	3
Night Diving	2
Total	**86**

MARINE ANIMAL INJURY

Marine animals contributed to the death in 5 - 8 % of the cases. In one case there was a shark attack. In two others there was obvious evidence of shark attack, but it was not clear whether this occurred before or after death. In another episode a shark bite, probably from a wobbygong, initiated the incident but did not of itself cause the death.

In one instance the presumed attack was possibly from a eel. In one well recorded instance, a squid was caught by the diver on his spear and during its attempted removal it damaged the diver and initiated, very rapidly, a series of events which lead to his death. This is the only recorded case of a death from a cuttlefish injury. In another episode there was a Crown of Thorns injury probably responsible for causing vomiting underwater and the initiation of the fatal sequence of events. In another case, which was considered too indefinite to be included, there were multiple coelenterate stings on the legs,

CAVES

Of the six deaths (6 %) in caves, two involved double fatalities.

In four of these deaths the caves were in the ocean, and one of these involved only a small entrance through which the diver could only enter after removing all breathing equipment, and trying to breathe from a very long octopus regulator, the equipment being pushed in front. In another two cases the caves were of the fresh water type.

DECREASED VISIBILITY

Six divers (6 %) died in conditions of decreased visiblity.

ENTRY AND EXIT PROBLEMS

Five divers (5 %) died because of promblems with either entry or exit. One of the deaths occurred because of poorly planned entry and others because of difficult or unavailable exits.

COLD ENVIRONMENT

Although the cold environment was considered to be a contributing factor in five cases (5 %), none were involved in diving under ice, and they occurred because of inadequate protection or excessive exposure.

ENTANGLEMENT

Environmental hazards caused entanglement in only four cases (4 %). In three of these it involved lines used by divers and in one it involved kelp. Not included in this figure is entanglement in harnesses or diving equipment.

BOATS (N = 2-3 %)

Boats were involved in two or three cases (2-3 %). In one case the boat caused apprehension on the part of the deceased, but was not a physical threat. In another, the boat definitely caused the death. The third diver was possibly run over by a boat.

TRAPPED UNDER LEDGE OR BOAT

Three divers (3 %) died after being trapped under a ledge or boat.

NIGHT DIVING

Two divers (2 %) died during night dives.

Discussion

It is important to realise the difference between these figures and those given in the United States by the National Underwater Accident Data Center (NUADC)[2,3] and Australia by Project Stickybeak.[4] In both those surveys the figures referred to the incidence of the particular environment, i.e. all the cases who dived in caves were classified as cave divers. In this Australian and New Zealand (ANZ) series, only when the environment actively contributed to the death was it included.

Thus a cave diver, who happened to have a coincidental dissecting aneurysm of the aorta, is not counted as a cave diving death in the ANZ series.

In some cases there have been more than one contributing factor. Thus two fresh water cave divers who got hopelessly tangled in their own lines, at 59 metres depth, were recorded as only two cases in the 62 affected by the environment. Nevertheless they were recorded 3 times within this part of the survey, as cave diving, as entrapment and as depth. The reason they would be included in cave diving is that, in the open water there is every likelihood they would have reached the surface and not died. The reason the depth was included was because of the almost certain narcosis and resulting poor judgement contributing to the incident and because depth increased their air consumption. The reason the entanglement was included was that it took so long for the rescuers to disentangle them, even with knives and good lighting, that the divers themselves would have found it particularly difficult. They would not have been included in the poor visibility section, as they both had adequate lighting available.

By far the most significant environmental factor was that of water movement with which the diver could not cope. In almost half these cases there was a tidal current. It was evident that divers were not able to cope with strong currents, probably because of the excessive drag on equipment. In many cases the current had the effect of either separating the diver from his safe environment (boat, shore, etc.) or of inducing him to swim very strenuously in an attempt to regain that safety. Fatigue, panic, cardiac dysrhythmias, salt water aspiration and asthma provocation may result.[5]

In just under half the cases of excessive water movement, there was rough surface conditions. It was evident that a diver in “white water” is a diver in trouble. The reasons for this might have been related to the fact that “white water” is usually fast flowing and turbulent, having the same effect as tidal currents, or it might have made the diver less buoyant and therefore requiring more effort to remain on the surface. The interference with visibility associated with “white water” might also have been a factor.

In those cases in which there was unexpected underwater surge, the deaths were due to trauma, with the divers

being battered against rocks and losing or damaging equipment.

There were 2 who died because they were trapped and drawn into a **pressure outlet** (a valve) in a fresh water dam. It was not appreciated that the outlets in dams, although not very deep, nevertheless cause a considerable pressure difference. In both cases the body was drawn into the outlet pipe. Although the flow of water was not great in either case, the pressures were excessive once the divers body had been drawn onto and had obstructed the orifice.

The effect of depth in contributing to 12% of the deaths, is probably an underestimate. In many other cases it may have contributed, because of the influence on nitrogen narcosis, consumption of air supply, resistance to breathing, ascent problems, panic, etc. In no case in this series was depth related to decompression sickness, as the latter disease was not a cause of fatalities.

There is a tendency to belittle the importance of marine animals, and in many such injuries the pathologist would be unlikely to observe even a fatal injury (e.g. from a cone shell or blue ringed octopus bite).

Fresh water cave diving was not particularly common in this geographical area, and in 4% of the deaths the caves were ocean caves, in which the customary cave diving procedures were not followed.

It was clear from the lack of experience in the cases in which the environment contributed to the death, that training in that environment was usually inadequate. It was not readily appreciated by many of the divers who succumbed, or by many of their companions, that training for one diving environment does not necessarily translate to others.

These cases confirmed, again, that diving is carried out in a potentially hazardous environment, and this can be unforgiving when adequate precautions for safety are not taken.

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DIVING ACCIDENT CASES TREATED AT THE H.M.Z.N.S. PHILOMEL RECOMPRESSION CHAMBER IN 1988

Allan Sutherland

Summary

This report on diving accident cases treated at the Philomel recompression chamber during 1988 shows that the cerebral effects are more refractory than previously thought when assessed by general practitioner observation, family observation and scientific assessment at a Post-Concussion Clinic.

Cases treated

A group of six doctors, the Philomel Recompression Chamber Roster, provided the Diver Emergency telephone advice and treatment for decompression sickness (DCS) and arterial gas embolism (AGE) during this period. Thirty cases required treatment in the recompression chamber. One was an Indonesian aviator under training with the Royal New Zealand Air Force and the remainder resulted from sport diving accidents. Of the thirty cases, twenty-three have been contacted subsequent to discharge, and of those 23, eight are permanently and significantly damaged as a result of their diving accident, and five are unable to return to normal employment.

Two follow ups were conducted, one at six months and the second at two years. Three of the cases are still consulting me on a regular basis, but all are functioning at a cerebral level much below that before their diving accident. The usual symptoms occur intermittently, headaches one day per week or two, thought blocks, poor concentration, muddled thinking, dropping things, falling, altered sensation, limb weaknesses, getting lost, etc. The presentations have marked similarities to post viral, or chronic fatigue/ ME syndromes.

I arranged for the post-concussion clinic at the Auckland Public Hospital, run by neurophysiologist Dr Dorothy Gronwell, to assess these cases. Regrettably the follow up information is not available on all cases, but three were assessed at thirty per cent deficit for higher cerebral function by this clinic, and a similar assessment would probably apply to the other three more severely affected patients who were not tested. By the very nature of their disease these patients become unreliable. Appointments are made with neurologists, post-concussion clinics and clinical psychologists, but the patients do not turn up, or turn up at the wrong time; further, as they are geographically distributed through the North Island, follow up is particularly difficult.

The worst cases

W had a Skipper's ticket and was an entrepreneurial gentleman until his diving accident. He now works intermittently relieving as a taxi driver, frequently getting lost and confused whilst working. W was assessed as having a 30% deficit. He appears punch drunk and he is certainly functioning at a much lower level than before the diving accident and his enjoyment of life is much impaired. He has been treated with tricyclic anti-depressants and this seems to have improved his mood and adaptation to life.

M, a welder, took six months to return to work, changed jobs three times, had much domestic difficulty, and was assessed by the Accident Compensation Commission medical insurance (ACC) as having a permanent disability of 30% after one year. He was treated with tricyclic anti-depressants for that year but is no longer on any treatment. Now, after two years, he has returned to welding and is much improved, but is not as he was before the diving accident. Both he and his wife descibe a personality change.

E, a diabetic, hypertensive Maori, who would dive six times a day to 100 feet, possibly taking illegal shellfish. His lifestyle, after his diving accident, has markedly changed to his own distress. He is not getting on with his family, is giddy and has joint pains. and his whole personality has changed. Attempts to get this man to attend for a neurological assessment or head injury assessment have been unfruitful. He was encouraged to make contact with the ACC, and follow up attempts have been unsuccessful.

T sustained two decompression accidents. He suffered with severe back pain and intermittent "head problems". This man clinically had spinal and cerebral bends. He presents as punch drunk and came into conflict with the ACC and received no final assessment or payment. He has recently had three lumbar vertebrae fused by an orthopaedic surgeon. This man believes that his personality has changed and that he is permanently disabled from his diving accident. This is confirmed by the neuropsychological report.

D suffered with dyslexia as well as having a diving accident and this certainly accentuated the symptoms. This encourages me to believe that people with a major cerebral deficit should be advised against diving, or be made aware of the slightly greater risks should a diving accident occur. This person lost two jobs and has become considerably depressed, with difficulties adjusting to a less responsible job, taken since the diving accident.

B, the aviator, who was decompressed rapidly and sustained an air embolus, was deemed unfit to continue his flying training. Follow up has not been possible, but there is little doubt that he is permanently cerebrally damaged.

It has not been possible to contact two other patients at two years, but they were contacted at six months and neither was in the same employment as they had been before the accident and both were having domestic and work problems which they attributed to the diving accident.

These were eight of the 23 patients followed up to 6 months and of 30 patients in all.

Less serious cases

On assessment after treatment ten patients had persisting minor symptoms which had mostly resolved before the six month follow up. The remaining twelve cases claimed to be totally normal at discharge.

Conclusions

This interim report demonstrates the great problems involved in following up this particular group of people, who may well have been difficult before the diving accident, but who were certainly very difficult to find and follow up following the diving accidents. This confirms the statement by Curley et al [1] that the cerebral recovery from DCS and AGE is more refractory than previously thought.

A full study of this group would make a good research project for someone with plenty of time and detective skills.

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DIVE COMPUTERS AND FLYING AFTER A DIVE TO 39 M FOR 10 MINUTES

John Lippmann

An interesting report by Balldin[1] describes an experiment in which 10 healthy, male divers or military helicopter pilots simulated a rectangular profile dive to a maximum depth 39 m for a bottom time of 10 minutes in a hyper/hypobaric chamber. The ascent rate was 18 m/minute and no decompression stops were performed, in accordance with the US Navy Tables. After an interval of 3 hours at 1 ATA, the pressure in the chamber was reduced to simulate an altitude of 3,000 m which is similar to the maximum cabin pressure that occurs in some commercial airliners. A Doppler ultrasonic bubble detector was used to monitor the divers in the pre-cordial position. Bubbles were detected in 30% of the divers, however no cases of decompression sickness (DCS) were diagnosed.

Many dive computers indicate the surface interval required before flying in a commercial aircraft after a dive or series of dives. These surface intervals vary greatly between computers, depending on the decompression model on which the computer is based and the parameters and criteria used within the model. Curious to discover what surface intervals various dive computers would require before flying after a dive to 39 m for 10 minutes bottom time, I collected a variety of units and tested them in a pressure chamber.

The chamber was pressurised to 4.9 ATA over one minute, maintained at 4.9 ATA for 9 minutes and then gradually released over 4 minutes to simulate an average ascent rate of approximately 10 m/minute, as specified by most of the computers. The only computer that indicated the need for a decompression stop was the "Micro Brain Pro Plus" which required a stop of 45 seconds at 3 m. Table 1 shows the results.

The results caused me some concern. Three of the five computers indicated surface intervals considerably shorter than the 3 hours after which Balldin found substantial gas phase formation.

One would hope that the slower ascent rate recommended by the computers would reduce the degree of gas phase formation below the level that occurred in Balldin's subjects, but, to my knowledge, this has still not been proven. In any case, despite the very useful ascent rate indicators incorporated in most dive computers, many divers still, at times, find it difficult to maintain such a slow ascent rate, especially if no ascent line is available. If ascent is too rapid, substantial bubbling may occur, and this will very likely slow down gas elimination. The current computers do not adequately account for any delayed off-gassing due to a rapid ascent and will give exactly the same surface intervals before flying (as well as identical repetitive dive times) as when the ascent rate has been adhered to. In a similar experiment to the above, an ascent rate of 18 m/minute gave almost identical results on the dive computers despite their recommended ascent rates having been greatly exceeded. The only difference was that the "Micro Brain Pro Plus" required a decompression stop of 70 seconds at 3 m. It still indicated that it was safe to fly immediately after the dive.

Table 1

INTERVALS BEFORE FLYING FOR VARIOUS DIVE COMPUTERS

Dive computer	Interval
Aladin Pro (US Divers Monitor 2)	36 min
Datamax Sport	3 hr 25 min
Micro Brain Pro Plus	0 hr
Skinnydipper	5 hr
Suunto SME-ML (R1)	2 hr

We can never really be sure exactly when it becomes "safe to fly" after a dive as it will depend on the degree of gas phase formation and for how long it persists. There is a gradual reduction in risk with time. Many authorities now recommend that a diver waits at least 24 hours before flying after any air dive, but a substantial number of divers have suffered from DCS after having waited far longer than 24 hours before flying. Flying as long as 5 days after extensive diving has resulted in symptoms.

Most dive computers are continuing to improve as the manufacturers realise their shortcomings and modify their programs accordingly. However, it appears that they still have some way to go. By the end of 1988, 121 cases of bends in divers using computers had been reported to the US Diver Alert Network (DAN,) 77 of these occurring in 1988 alone.[2] In the USA, computer-related DCS increased from 14% of the total DCS cases in 1987, to 36.6% in 1988.[3] Divers should not blindly follow their computers but should add substantial safety margins to the times allowed by their units.[4] This advice seems very relevant to flying after diving.

Interestingly enough, despite the worrying short intervals often given by the "Aladin Pro" and "Micro Brain Pro Plus" for flying after diving, these two dive computers are generally more conservative than the other brands for most diving situations and are, in my opinion, the best dive computers currently available.

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SCUBA TRAINING FOR THE OUT-OF-AIR SITUATION

Douglas Walker

By far the most contentious subject to raise with those who instruct scuba divers is whether practice in making an out-of-air ascent should be prohibited, permitted if performed under very strictly controlled conditions, or be made an integral part of the training course. This paper discusses dispassionately some of the background to the controversy and records the manner in which the main instructor organisations in Australia have chosen to resolve the conflict between the risk factors of the alternative possible courses of action. Tables 2 to9 are constructed from information in the Divedata Databank (Project Stickybeak) files. These files are accessible to any interested person or organisation.

Historical Background

Until pumps had been developed capable of supplying air in sufficient volume to maintain a man underwater the only way to dive was in the breath-hold mode, although some records state that Greek divers in ancient times may sometimes have used air lowered to them in cauldrons by people in boats.[1] The problem of supplying air to keep the water from rising too high with within diving bells as they descended was solved by sending air down in weighted barrels on a line, a method described by Halley[2] which remained in use till a sufficiently effective air pump was developed and users changed to this method of replenishing the bell air. He later designed a hookah system which was supplied by the pressured air within the bell. The mortality and morbidity among the workers is unknown but it is probable that accidents occurred when the workers were faced with a necessity to reach the surface or drown, with some suffering air embolism. In those rough, tough days their deaths would be accepted as due to drowning as the very existence of such a condition as air embolism had yet to be recognised.

As soon as it was realised that a man could be supplied with a continuous flow of air while wearing a helmet, which was in essence a mini-bell, the commercial possibilities were recognised. Now workers were free to move on the sea bed, no longer limited to the area immediately below the bell. The occupation of commercial diver came into existence, hardy, brave labourers who seemingly had a philosophical (or resigned) attitude to the dangers of the work they performed. The early suits were of a helmet and jerkin type, so filled with water if the wearer bent too far forwards, a design soon replaced by the standard suit (except among pearl and sponge divers). The open suits could be discarded by the wearer should necessity or panic make him desire an emergency surfacing.[3] It was such untrained divers who first made "free ascents", luck deciding the outcome of the ascents, although the reason why the fatalities occurred was not discovered until the US Navy introduced practice out-of-air ascents for submariners and some died.

The first recorded instance of a "for real" free ascent from a submarine was that of Corporal Bauer and his two crew from 18 msw depth in 1851. William Bauer must have been an exceptional man because while trapped in the damaged "Sea-Diver" listening to anchors trying to engage themselves in the submarine's structure, intending then to pull it to the surface but more likely to break one of its portholes, and watching water seep through the damaged joints on the hull plates, he managed to think clearly. In this he was helped by the remarkable composure of his crew. His knowledge of physics enabled him to recognise that the hatch was kept firmly closed because the water pressure was greater outside than in the submarine but that he could equalise the pressures by opening the sea-cocks to allow water to enter. That he persuaded his crew that this course of action was safe says much for his personality. All three ascended successfully despite each first taking a last deep breath before opening the hatch. The success of their response to the life or death situation was without benefit of prior training or, as far as is known, planning for such an emergency.[4]

That their survival was not a unique event can be given credence by the findings of the British Submarine Escape Mission, set up by the Admiralty to investigate known cases of escape from actually sunk submarines of all nations.[5] This noted that most of the successful escapes were made without the use of the equipment designed for use in such circumstances "but rather by free ascent or buoyant-assisted free ascent (buoyant ascent)... a result of the malfunctioning (equipment) and/or poorly-trained crews". There is relevance in this comment to the subject of the training of scuba divers and especially to attempts to perform a buddy-breathing ascent unless both divers are well trained and have sufficient air available.

The desire to reduce the toll, and danger to morale, from both peacetime and war sinkings of submarines prompted the design of apparatus to assist the safe escape and ascent of survivors as well as attempts to develop methods of rescuing them by use of a diving bell or a submersible. It was recognised that men would be reluctant to trust themselves to the open sea at depth by exiting from the environment they knew, the submarine, unless they had some reason to trust the apparatus they were to use. For this reason a rule was made that all submariners had to pass a course involving making ascents in a test tower. The United States Navy (USN) appears to have been the first to build a Submarine Escape Test Tower (SETT), in 1930, although it is known that from the 1920s there had been testing of escape and ascent methods in the mine test tank at the Naval Gun Factory and in the open sea from diving bells and it was from these tests that the US Navy developed the Momsen "lung". It was only after the visit of Rear Admiral Ruck-Keene to the New London SETT that the Royal Navy (RN) constructed a similar tower in Gosport at HMS Dolphin.[5]

It became apparent that the SETT training was occasionally associated with fatalities despite the vigilance of the SETT instructors and for this reason a recompression chamber was first placed close to the tower and later installed at the top close to the area where those who had just ascended were watched for signs of sudden illness, now recognised as due to arterial air embolism. Indeed it was observations on such cases which led in 1931 to the realisation that such persons were not suffering from decompression sickness occurring after a remarkably short and shallow dive.[6-8] These occasional deaths also showed the need to improve equipment so that it would become more nearly natural for the person making the ascent to breath in a normal continuous rhythm.

Although it can be correctly argued that SETT situation differs significantly from that existing in the training of scuba divers because the participants start from an air environment and have no desire to be divers, rather they would never wish to enter the sea "for real" unless their submarine was trapped on the sea bed. However the close observations made of them as they ascended and the close medical backup create a source of information which cannot be matched by any diving sources. The rarity of the deaths and the fact that they still occur despite the careful management of such training makes the SETT records of interest to both sides of any discussion of the safety of practicing out-of-air ascents.

The use of a body count of fatalities is a rather crude method of determining the safety of any activity because it fails to take into account the morbidity which may be resulting in some unstated proportion of those at risk. However there are naturally many reasons why those who operate a procedure are very reluctant to investigate for possible adverse effects, particularly if there is no demand that they do so and they believe the procedure which is involved is cost effective and therefore necessary. There have however been two significant papers which indicate that there may be a hidden degree of morbidity even with carefully conducted and monitored SETT escape procedures as evidence of both cerebral air embolism and pulmonary barotrauma has been found to occur in some who have made a successful ascent and are clinically normal.[9,10]

None of this was of interest outside the small group of persons involved in the training of submarine personnel until the development of the Cousteau-Gagnan demand valve and its clones in the 1940s made it possible for recreational diving to develop, as it had early been recognised that the use of rebreathing apparatus was far too dangerous for general use. In contrast to the wartime situation, where training and operational fatalities were accepted and could be kept secret, any recreational diver who died received newspaper publicity, was the subject of coronial investigation, and had the details discussed within the then small diving community. Recreational divers very rapidly concluded that they should avoid such equipment and use only systems which supplied air on demand, the open circuit as contrasted with the rebreathing mode.

Naturally the diving community, which till then had been a small number of tough, skilled, breath-hold spearfishermen, was at once keen to learn more about using the equipment which would greatly expand their underwater time. They had no sources of instruction save those who had taught themselves (and survived to teach others), no instruction manuals, and no awareness on the part of anyone that there were any special risks, and naturally assumed breath-hold experience, usually gained in the rough water close to the rocks, prepared them to manage any situation even if their air supply apparatus failed them. Nobody then suspected the existence of conditions now detailed to every trainee diver, or rather there was no awareness of their importance. Word of this new and easier way to venture underwater soon spread, attracting a very different type of person, people who had no experience of breath-hold diving and desired formal instruction but were without diver friends who could tell them the safe way to use an "Aqualung". This was at first the generic name for all such apparatus but because this was the registered trade name of the Cousteau Gagnan apparatus there was developed the generic alternative term self contained underwater breathing apparatus or SCUBA.

Naturally this new market attracted rival manufacturers who attempted to develop apparatus which by-passed the patents of the original developers. In Australia some keen breath-hold spear fishermen with engineering skills constructed scuba apparatus for their own use, then were approached by friends to supply them also with sets and from such beginnings several small firms developed. These flourished for a time and then ceased trading when faced by the market power of large overseas manufacturers. In Aus-

tralia it had been necessary for most spearfishermen to construct their own equipment so local manufacturing of this rather more complex scuba equipment was fully accepted. It is not known whether there was a similar make-your-own phase in Europe but the larger market there possibly tempted small commercial manufacturers to start up. These were able to undercut the prices of the authentic Cousteau-Gagnan apparatus and therefore enjoyed a period of success in the market. However the quality of some of these products was so poor that the users soon became aware that risk of a regulator failing was significant. In the UK this led the Kingston Branch of the BS-AC to institute the survey of some of their members in 1962 which is sometimes quoted as proving the safety of practicing out of air ascents.[11]

The survey is of particular interest because it was the only recorded such one in the UK. The original report cannot now be found but the resume shows that it was based on the replies of 36 active members of the branch. They had a total of 170 years of diving, an average of 4.73 each. The total membership of the club, adding the yearly totals for the six years 1956-1961 together, was 20,597, an average of 3433, so the sample was small and there is no information as to how representative it was of the general diving experience of the members. The report states that they had made a total of 461 practice free ascents, 25 necessary free ascents, plus 11 rapid ascents (from loss of weight belt or life jacket firing). A calculation was made to show that the club members would have made something like 30,131 rapid ascents during the six years if their diving was similar. But as a concession to any possible criticism that the sample divers were more active than the others the total was given as 15,000. It was noted that there had been no fatality in the club although SETT figures would predict such would occur, and mentioned that members only practiced a free ascent when they felt they wanted to, not in response to an order, that they did not ascend so rapidly, and were used to swimming underwater so adapted to pressure changes automatically. It is not stated whether there was any training or whether members performed a free ascent where and when and under whatever circumstances they thought fit. There is no statement that the club actually required or supervised the free ascents. This report by Hume Wallace failed to deflect Royal Navy advice that the BS-AC prohibit training involving practice of out-of-air ascents (free and buoyant ascents) “unless there was a recompression chamber ready for use at the site”. Buddy-breathing training was commended, to be performed swimming horizontally with no actual ascent to be made. This was stated before the assembled BS-AC Diving Officers by Stanley Miles, David Elliott[12,13], and others on various occasions. As a concession each year 100 BS-AC divers have been allowed to attend the Royal Navy SETT establishment to be instructed in how the RN manages ascents by submariners. The reason for this offer, other than as a public relations exercise, is very difficult to identify but may indicate an understanding of the desire, which many divers appear to harbour, to practice such ascents.

As Table 1 indicates, the BS-AC has now departed from its initial acceptance of the advice it received and now includes the practicing of buddy breathing ascents in training courses. But in its defence is the fact that BS-AC courses may occupy three months rather than the five days most commercial courses take to “train” recreational scuba divers. The BS-AC courses seek to progressively increase the difficulty of the skill being taught, with monitoring of pupils to ensure that they have sufficient skills before being set more difficult tests. Whether this is understood and approved by the RN is not known, and it is debatable whether it is the time spent under supervised training rather than the actual content of the training which is to be credited as being important to diving safety of BS-AC divers. There are two additional factors which are helpful to safety, the club system and club diving officers. These may not be perfect but they have some personal knowledge of their fellow divers’ abilities. This complete change in BS-AC policy has not apparently been commented upon by any RN liaison officer, which is strange. Indeed the BS-AC’s Principal National Coach[14], addressing the 1987 Diving Officers Conference, stated “No one challenges the advice that assisted ascent and rescue ascent should be practiced so that divers are better trained to cope with a true emergency, and there is no question of dropping this training requirement”. He added that “to get maximum benefit from the exercise it should be repeated by doing as many ascents as possible”.

In other countries it continued to be accepted than all scuba pupils should demonstrate an ability to perform one or more emergency ascent in an out-of-air situation, a decision based more on doctrine than facts because there has never been any survey of European diving fatalities and in the USA the diving data appears to have been regarded as of less importance than the gut feelings of many instructors and diving medicine experts.[15] One factor which has mitigated against an impartial discussion of whether or not a practical experience of out-of-air ascents had greater benefit to the diver than the possible danger has been the semantic disputes which have surrounded and intermixed with such discussions as the original term employed, free ascent, was later used to describe a variety of methods. In this paper the term “emergency response to an out-of-air situation” has been chosen to describe what type of problem the training seeks to address.

Present Australian Training Protocols

There was a commonly agreed standard to which Australian Instructors had to bring their students so they can be given a NQAS certification in addition to that awarded on course completion by the organisation of which the instructor is a member. However the different interpretations of these course parameters has resulted in significant differences between the training programs detailed for use by instructors in different organisations and reflects in large degree the different philosophies of training which are now

TABLE 1

TRAINING PROGRAMS FOR OUT-OF-AIR SITUATIONS

Ascent	Emergency Swimming training	Buddy Breathing training	Shared Air (Octopus)
FAUI	No	Pool or calm sea horizontal at 2-3 m depth for 25 m then open sea at depth >5 m	Pool or calm sea horizontal at 5 m depth for 25 m then in open water
PADI (Aus)	Pool, horizontal in shallow then diagonal from deep, >30 ft exhaling continually. Ascent Open water Instructor on line Regulator in mouth from depth 20-30 ft.	Pool, stationary then swim >50 ft in shallow end. Open water get breathing rhythm then one 20-30 ft buddy breathing ascent.	Pool or calm water stationary then swim both as the donor and recipient >1 minute then donor and recipient ascents.
SSIA	20 ft ascent ** up line with instructor	Pool floor	One ascent from 20 ft depth
NAUI (Aus)	Pool, horizontal depth 2-3 metres.	Pool or calm sea 2-3 m depth then sea >5 m horizontal for 2 minutes.	Pool or calm sea 2-3 m depth then open water >5 m ascent as donor then as recipient.
NAUI (USA)	Diagonal 40 ft line in pool.	As above	As above
BSAC	No * but pool ditch, ascend, descend, don scuba, <3 m.	Graduate from land to pool to sea, increasing depth, problems sharing ascent.	as above then in sea gradually to 25 m (advanced divers), stop 6 m.
ASC (unspecified)	Requires “training in buddy system techniques” and in “out-of-air emergency alternatives”		

* See Appendix A
** This test may soon be eliminated from training

Abbreviations

PADI Professional Association of Diving Instructors
NAUI National Association of Underwater Instructors
FAUI Federation of Australian Underwater Instructors
SSIA Scuba Schools International Australia

TABLE 1 APPENDIX A

DOFF AND DON AND FREE-FLOW TRAINING

	Doff and Don	Free-flow
NAUI	Yes, in pool, keep regulator in mouth	*
PADI	*	Yes, kneeling in shallow end of pool 30 seconds.
FAUI	*	*
BSAC	Yes (voluntary), in pool, <3m, ditch, surface, descend and don. Also ditch as fast as possible.	Yes, during mask clearing and buddy breathing lessons, kneeling in pool in shallow water
SSIA	*	*

dominant in the USA and the UK. In essence the dispute is between whether in the limited time available for basic training there is to be stress on avoiding problems or on escaping the consequences of mistakes or misadventures.

There are only two basic reasons for a diver to be in a situation where he or she is unable to ascend using the original air supply: the equipment may be entangled but the wearer can ditch it and get free, or the air supply has become inadequate or failed, as occurs when the scuba tank is functionally empty or there is some malfunction of the equipment. Considered thus, the submariner has to abandon the submarine trapped on the sea bed but is unlike the diver in that he now has a secondary air supply, an escape suit, to enable a breathing ascent to be made after discarding the primary equipment (the submarine). Similarly, hose-supply divers should be wearing a bail-out bottle to avoid the consequences of out-of-air situations due to equipment failures. The out of air or low air(no/low) status scuba diver has by definition no reserves of air, this being the essence of the problem. Though not all hose-supply divers wear a bail-out bottle the majority appear to quickly learn to survive occasional compressor failure situations by making an emergency ascent as soon as their air supply ceases. They learn by having to face for-real out-of-air situations. Fatalities usually, but not invariably, being in the inexperienced users of such apparatus.

The situation is therefore that a scuba diver will only need to exercise an ability to make an emergency ascent if either the equipment becomes irretrievably entangled, fails suddenly or he or she runs out of air. The latter is by far the commonest and should be totally avoidable by following the accepted safe diving rules. That so many diving-related fatalities are associated with divers who are in a low or out-of-air situation is a matter which deserves urgent attention (Table 2). Possibly there is inadequate stress during training on adopting a “defensive diving” attitude, the recognition of and response to potentially adverse situations before the crisis stage, and too much time is spent on showing how to respond after an out-of-air crisis has occurred. There is only a limited time available for instructing pupils so course content is necessarily a matter of compromises. These are much influenced by the prejudices of those involved and their attitudes to the competing philosophies for survival in an adverse environment. One can learn to identify problem-breeding situations and act in a manner which avoids their development to a critical degree, or accept that such critical situations are unavoidable and concentrate training time on the management options of such crises. In an ideal world there would be time to address both options, the prophylaxis and therapy approaches to diving safety.

It is possible that the initial source of the belief in Australia that practicing of some form of out-of-air ascent ought to be part of scuba diver training was the UK, an importation of the initial BS-AC training which was not modified after the RN advice to omit such training practices was reluctantly accepted by that organisation. In recent years the influence of US beliefs has been a significant factor due to the dominant position of the Professional Association of Diving Instructors (PADI) and the National Association of Underwater Instructors (NAUI) in scuba diver training in Australia, with the Federation of Australian Underwater Instructors (FAUI) alone continuing with its acceptance of the advice from both RN and Royal Australian Navy (RAN) to avoid practicing emergency type ascents. Despite the significant differences which exist over this matter between each of the training organisations (Table 1) they have agreed to recognise each other’s certificates of scuba training,

TABLE 2

SCUBA DIVING FATALITIES 1980-1988

	Total	adequate air	no/low air	No Air	Low Air	N/s
Australia	62	18	39	23	16	5
New Zealand	56	17	28	20	8	11

TABLE 3

SCUBA DIVER FATALITIES 1980-1988

CAGE V LOW/NO AIR STATE

	Number	Adequate	No or low	Not Stated
Australia				
Total	8	-	7	1
Had gauge	7	-	6	1
New Zealand				
Total	12	3	6	3
Had gauge	11	3	5	3

TABLE 4

SCUBA DIVER FATALITIES 1980-1988

TRAINING STATUS V NO/LOW AIR STATUS

		Number	No training	In class or some training	Trained sometime, somewhere	Not Stated
Australia						
Total	62	13	9		38	2
No/low	39	7	6		25	1
New Zealand						
Total	56	15	4		21	16
No/low	28	8	2		10	8

though obviously there will be some instructors who will regard divers trained by another organisation as having received a less good training than that necessary for safety.

There are two factors to consider when seeking to reach a decision as to whether any of these differing teaching programs can be considered the one of choice. The scuba divers produced by the course should have a safety record at least as good as divers taking alternative courses, and those elements in the course which differ from alternative course schedules and have a potential for causing morbidity should be clearly demonstrated to produce safer divers better fitted to recognise, and manage, all probable diving-related adverse situations. All courses should produce divers who are aware of the limitation of their skills, that they are novices on completion of any course until experience has resulted in them reaching the stage where the new knowledge has been so integrated into their cerebral databank that it is now part of the automatic response pattern in any diving-related emergency situation.

This requires that the training exercises have not only been practiced sufficiently to make their repetition accurate and automatic, sometimes called "over-learning", but also be safe enough to be repeated by divers to maintain and improve the skills after leaving the instructor-controlled situation of a class. Naturally the relevance of taught skills can only be established through an examination of the apparent critical factors revealed by analysis of actual diving-related incidents, and as changes occur in diving practices and equipment there will be changes noticed in the kind of problems which most significantly effect divers. Training will need to take account of such changes by adding to or amending the content of courses.

The out-of-air situation in scuba diving fatalities

An examination of the adverse factors found on analysis of scuba diving fatalities indicates that a low-air or out-of-air situation has been present at the critical time in far too high a proportion of the incidents to be other than a significant factor which has influenced the course and utcome. It disadvantages the victim, reducing his or her options in devising a successful response to whatever was the immediate problem. In only one instance did a catastrophic failure of a scuba air supply system occur and there were additional adverse factors involved here, for this victim was totally untrained and using scuba for the very first time. In one other case a low air situation was tragically terminated when the diver's buoyancy vest's venting valve failed during ascent and as an additional complication one weight had moved, jamming the quick release of the weight belt. The discussion of factors relevant to diver safety is limited in this paper to avoidance of the low air and out-of-air situations and assessment of probable outcome, risk versus benefits, for different teaching protocols which presumably instil in trainees different patterns of response should they be faced with such a situation. As it is inevitable that no training course in recreational scuba diving can afford to allocate enough time to produce divers who are more than novices on completion of their training, all courses must be a compromise and true learning comes from the repetition of what they have performed only a very limited number of times during training.

Possibly the first significant finding is that while a no/low air status was present in the majority of cases of cerebral arterial gas embolism (CAGE) such cases form a relatively small proportion of the instances where air shortage had occurred. This is shown by comparing tables 2, and 3. The tables are all based on the case reports in the Project Stickybeak Divedata Databank, with the diagnostic label of CAGE being applied where a description of the incident made this the probable critical factor. In addition, in most instances, the autopsy findings were confirmatory. One can therefore deduce that a no/low air situation is not inevitably an event which is followed by a CAGE. In the majority of instances a combination of additional factors influence the course of events, and although the cases considered here ended fatally it is likely that the majority of low air situations were successfully managed by those involved. Diver safety will naturally be enhanced by the strict avoidance of a low air status although an understanding of the correct response to such a situation is undoubtedly of value. Logic suggests that the low-air state should be less serious than to be without an adequate air supply as in the latter situation a necessity to reach an air source will severely limit the response options, an assumption supported by the data (Table 2) which shows that out-of-air fatalities outnumber those with low-air status.

The next important finding (see Tables 4 and 5) is that the no/low air situation occurs with significant frequency across the full spectrum of training and experience. This can be taken as an indication that neither initial training nor diving experience is teaching scuba divers the need to monitor their remaining air and to ascend while still having sufficient air to meet any emergency situation. It is also apparent (Table 6) that such situations are developing proportionally more frequently in those furnished with a contents gauge than those without such an aid. As the gauges in these cases are not faulty the problem must lie with the users, an indictment of their diving techniques. The majority of no/low air situations, on such figures, appear to be avoidable.

The importance of the buddy diving principle is the one matter taught in all courses of instruction, although it is common to observe that many divers follow this precept somewhat loosely after release from the class situation. There has probably never been a formal investigation concerning the correctness of this advice to divers. However it is noticeable that solo or separated divers are far too frequently the victims of fatal incidents to support a change in opinion concerning the apparent adverse effect on diver

TABLE 5

SCUBA DIVER FATALITIES 1980-1988

STATED EXPERIENCE V NO/LOW AIR STATE

	Number	No or slight experience	"Some" experience	Experienced	Very experienced	Not stated
Australia						
Total	62	23	16	19	4	-
No/low	39	10	15	11	3	-
New Zealand						
Total	56	15	17	14	3	7
No/low	28	8	9	7	-	4

TABLE 6

SCUBA DIVING FATALITIES 1980-1988

CONTENTS GAUGE AND NO/LOW AIR STATE

	Number	No Gauge		Gauge		Not Stated	
		Total	no/low air	Total	no/low air	Total	no/low air
Australia	62	14	8	48	31	-	-
New Zealand	56	6	3	36	21	14	4

TABLE 7

SCUBA DIVER FATALITIES 1980-1988

DIVE GROUP AND NO/LOW AIR STATUS

	Total	Solo	Diving with buddy			Diving in group		
			no sepn	separation before	separation during	no sepn	separation before	separation during
Australia								
Total	62	6	10	20	11	4	11	-
No/low	39	4	8	13	7	1	6	-
New Zealand								
Total	56	15	3	27	7	-	4	-
No/low	28	7	1	14	2	-	4	-

safety related to a solo situation (Table 7). It cannot be claimed as statistically certain because the proportion of divers at risk who are similarly solo or separated is unknown. One thing however can be stated, they are as frequently in a no/low air situation as are the generality of scuba diver fatalities and certainly cannot seek emergency air from a buddy.

The role of the buoyancy vest may be debated, whether it is for surface support, to permit buoyancy control at depth, or for its lift capacity when the wearer wishes to ascend but has excess weight. It is now common for the buoyancy vest to be of the backpack type which uses the scuba tank as its air source. One result of this is that when the wearer is in a no/low air situation this has the effect of making the inflation system inoperative because in an emergency situation no diver will have the breath to orally inflate the vest. The congruence of no/low air and the wearing of a tank supplied buoyancy vest is shown in Table 8.

It has been shown that the majority of fatalities occur at the surface or at relatively shallow depths and this holds for the no/low air fatalities also (Table 9). If the case reports are examined it will be seen that inexperience, water conditions, being solo or separated, retention of the weight belt, failure to inflate the buoyancy vest, and medical conditions (most commonly these are cardiac) are frequently significant additional adverse factors. A singularity in the data from New Zealand is the comparatively few surface fatalities where the air supply was deficient. The reason for this has not been determined.

Discussion

There are a number of aspects of this problem which may be considered as requiring consideration:

1. Is the no/low air situation adverse?

The response options of a scuba diver to adverse events will inevitably be reduced should there be a need to conserve air because the remaining supply is running low, and become even fewer as the supply fails. Study of case histories indicates that there are usually several adverse factors present in incidents in which the dead diver was later found to have a low-air problem and they all influence the course of events. Naturally training and diving experience are assets but they do not guarantee survival where an out-of-air or low air state occurs (Table 4, 5). It has been shown that although cerebral arterial gas embolism victims are commonly found to have been out of air, the majority of fatalities where an out-of-air state has been present do not suffer this ending, which may plausibly be taken to indicate that it is the presence of one or more such additional adverse factors which convert a situation from a potential to an acute crisis for the diver. By implication the air status is not the single critical factor, though one which may play a crucial part in the progress of events.

There are several ways in which the no/low air state in a dive may be a danger. It will cause a degree of anxiety, have an influence on decision making, affect the inflation of the buoyancy vest where this is supplied from the scuba tank, make exiting more dangerous and a surface swim back to the dive platform mandatory. There will be the additional factors that no precautionary "deco" (decompression), or more properly, "safety" stop will be possible and a severely reduced ability to provide a buddy with air. Without an air supply a diver cannot submerge and continue to breathe, an action which may be life saving in rough sea conditions. Panic is more likely to occur if the diver is without an adequate supply of air.

2. Is the no/low air situation avoidable?

There is only one responsible reason for running out of air while scuba diving and that is where there has been some kind of sudden equipment failure. Such events appear to be rare, though a free-flowing regulator or freeze-up could prove equally serious to an unprepared diver. In all other diving situations the divers should monitor their air, and if necessary their buddy's, to ensure that surfacing and return to the dive platform is commenced while adequate air still remains. As shown (Table 6) it is not only the divers who are without a contents gauge who become low on air. In fact those who have a contents gauge appear to be proportionately more represented in the no/low air category.

Although there have been some minor inaccuracies in the contents gauges in some instances, none of these was a sole reason for a fatality. There appears to have been no pressing reason for victims to have failed to heed their gauges. Had they done so the probability is that they would not have died.

3. What is the best response option?

Naturally the best response is to become aware that the air supply is becoming low and commence ascending immediately. If correct dive procedures have been followed the buddy is close by, but if the buddy is some distance away it will be a waste of very valuable air to swim horizontally to the buddy rather than making a solo ascent. Case reports suggest that a buddy-breathing ascent in such circumstances is liable to fail disastrously, either when the donor also runs out of air or the rhythm of exchanging of the regulator is not honoured. Unless the buddy pair have established, through practice with each other, a mastery of the buddy breathing technique they will find it difficult to perform in the emergency they now face. If they have been so safety motivated to have made the effort to train with each other they are unlikely to have got into such a low air predicament. The use of the buddy's "octopus" regulator eliminates the dangerous consequences of any failure to exchange the regulator equitably. However there still remains the probability that the buddy's remaining air is becoming low and if subjected to a doubling of demand will run out rapidly, which will necessitate both divers having to perform a "free ascent" for the final portion of their ascent,

TABLE 8

SCUBA DIVER FATALITIES 1980-1988

BUOYANCY VEST TYPE AND NO/LOW AIR STATUS

	Number	No vest	Vest inflation method				Not stated
			oral	CO2	LP hose	Fenzy type	
Australia							
Total	62	13	-	7	36	3	3
No/low	39	5	-	3	27	2	2
New Zealand							
Total	56	15	6	3	18	-	14
No/low	28	6	3	1	11	-	7

TABLE 9

SCUBA DIVER FATALITIES 1980-1988

INCIDENT DEPTH V NO/LOW AIR STATUS

		Depths in metres							
	Number	**Surface**	**Ascent**	< 10	10-20	20-30	30-60	>60	**Not stated**
Australia									
Total	63	22	6	9	5	1	6	2	12
No/low	38	12	4	6	4	-	3	2	7
New Zealand									
Total	56	17	7	6	5	3	2	-	16
No/low	28	4	5	5	2	3	-	-	9

risking air embolism.

There are some diving situations where direct ascent is not possible, as in cave diving, and here it may be necessary for a diver to buddy breathe for a horizontal distance but for this type of diving special training is mandatory and management of the air supply is one of the most stressed safety factors.

It is not possible to discuss instances where there has been a successful ascent using buddy breathing or shared air (the use of an Octopus regulator) because reports of such are lacking, a regrettable consequence of poor information gathering. However it is likely that such ascents became necessary due to one of the divers failing to monitor his or her contents gauge, which should not occur and is a self induced situation.

It has been suggested that no/low air situations can be expected to become rare with the increasing habit of scuba divers to have an octopus rig, two cylinders with independent regulators, or to carry a small emergency (pony) cylinder. Human nature being what it is, the most likely outcome will be that the diver will, in most instances, treat the additional cylinder air as available for the dive rather than an emergency reserve. There would still be a need for the diver to respond correctly to the warning of the low air status which was developing. In the early days of scuba reliance was placed on the diver recognising the onset of some difficulty in obtaining the air. The first attempt to solve this problem was by developing the J valve to create an air reserve. Later the sonic warning systems were introduced. None proved reliable. It could be held that a diver who fails to take cognisance of the information which his contents gauge provides has failed to learn correctly and that greater time should have been spent on this during the training course.

It may be claimed, though without documentation, that the training courses which include ascents after simulated out-of-air situations have saved lives and that such divers are not shown in fatality records. Against this is the

fact that for many years in the UK the BS-AC has prohibited the inclusion of any actual ascent training in the out-of-air procedures and there is no evidence of this having been an established adverse factor in such fatalities as have involved their members. The primary reason for setting up the Project Stickybeak Divedata Databank was to establish a basic data resource to enable an examination and evaluation of the many factors which influence the evolution of any "incident". For this reason it has always been recognised that reports about non-fatal incidents would be highly valuable. Unfortunately, and world wide, the diving community has failed to recognise the vital importance of such reports. The reasons presented for this reporting failure are several but generally based on the claim that confidentiality promises could be dishonoured and the reports used as a basis for legal actions. Such fears are rarely justified and the risks have been minimised by designing the scheme so that the control of all reports is medical. It is often forgotten that the basic facts of incidents are not so much unknown as undocumented so there is not really any protection from non-reporting if some aggrieved person is desirous of instituting an action for damages. However failure to report makes it harder to identify problems and their possible remedies before a serious misadventure occurs.

In the de facto no/low-air situation the diver involved is likely to unexpectedly receive an inadequate supply of air and need to respond urgently. It is here that training and experience provide protection against panic, and BS-AC experience supports the premise that such training need not include practice of emergency procedure ascents. It has not been shown by follow-up assessments that the few practice ascents which are made during training courses have inculcated an ability to perform shared air ascents in those so trained which is panic-proof in a crisis situation.

The ability to make a successful ascent after becoming aware of being in a no/low air is probably a function of having confidence that it is possible and an understanding of the manner in which it should be performed. Training which extends to reach the stage of over-learning is not practical when courses must be designed with tight cost and duration constraints. So recreational divers are likely to find their emergency-ascent skills will very rapidly degrade after they graduate, but in an emergency situation they can probably ascend safely and without apparent morbidity despite the less than perfect performance of ascent procedures. This would be a reflection of the body's "redundancy principle" which ensures a continuation of functions despite trauma unless the damage occurs in a vital position which is not protected by a back-up. There are many instances of untrained divers making successful ascents, particularly those using unreliable hose supply equipment.

4. Does emergency ascent practice train or only demonstrate?

Numerous repetitions of a new skill are necessary if it is to become integrated so that it can be performed without any conscious thought in response to the relevant stimulus. There can be no such over-learning of emergency ascents because even the proponents of this procedure admit that there is a minimal risk associated with it (but balance this against their claim that the benefits outweigh the risks). In order to maintain and even improve their skills some divers will practice what they have been taught in the class situation but without an instructor in attendance to ensure an accurate performance. This unavoidably introduces a potential long term risk element into the history of such divers and also may reinforce the attitude, which they may have, that entering a no/low air situation is safe as they know how to reach the surface. However the records indicate that the surface itself gives no guarantee of safety when some additional adverse factors are present. The no/low air state has a danger potential which is additional to the obvious ones of either drowning or suffering an air embolism in seeking to reach the surface and air, so avoidance of such a situation has potential benefits in comparison with the attempt to become skilled in performing emergency ascents.

Some speakers at the UMS Workshop[15] recorded the opinions of their organisations on the question of how many repetitions of a new skill were necessary before the pupil was likely to perform it in an emergency situation. Smith (YMCA) suggested 17 exposures to buddy breathing were necessary, Egstrom (UCLA) quoted the views of diving officers from various institutions that a diver was not trained until he had performed 8-12 successful open water dives, a standard not achieved by their courses, while Miller reported that NOAA divers had to make 15 open water dives before attaining the category of "limited diver" and 100 before "unlimited diver". The view of Dr Lanphier was that he "did not feel very confident that ascent training in the open water adds a great deal to what could be accomplished with optimal classroom and pool training, although there are no numbers available on the subject", that such training should be a thoroughly voluntary procedure on the part of pupils, not required for certification of any group.

The BS-AC courses extend for far longer than those which are run by the commercial organisations and continue beyond basic training to include the "optional but recommended" section called Confidence Building Exercises. In a pool the equipment is removed and the pupil exhales while ascending, takes a breath, descends, and dons the equipment. There is a second exercise where equipment is ditched as rapidly as possible, remembering to disconnect a direct feed hose from the buoyancy vest. Table 10 shows the requirements for such exercises among the various diver training organisations.

5. What are the risks?

There have been no known fatalities in either Australia or New Zealand associated with the present training methods but a reference to the SETT experience shows that "hits" can occur even where stringent supervision is maintained. It has also been shown that subclinical pulmo-

nary barotrauma and cerebral air embolism occur in the SETT situation. It is likely that similar events are happening in those practicing shared air ascents. These ascents suffer from the serious disadvantage, inseparable from their basic design, that a minor mistake in performance may be followed by the rapid and possibly irreversible occurrence of morbidity. Although probably the majority of diving-related fatalities are identified and their critical factors analysed in Australia and New Zealand, such is far from being the case in other areas of the world where investigations may be less complete and documentation less easily accessible. By chance there has been one instance in Australia in recent times where two divers were apparently practicing a buddy-breathing ascent, though not under supervision, and one died during the ascent. Both were experienced and were on an Advanced Divers' course at the time. Hardy (NAUI) reported to the UMS Workshop[15] the fact that in the period 1970-76 the 5 major training agencies in the U.S.A had lost a total of 80 people during training, 20 during ascent training. DAN data has note of 11 instances where diving instructors had suffered injury after running out of air and/or when making rapid, panic ascents.[16]

One aspect of the ascent procedures taught is that pupils have to exhale in a forcible and continuous manner and this introduces the possibility of creating some areas of air trapping due to the collapse of some small air passages. It is far more physiological to make some inhalation efforts during ascent, even if there is no air available, as this will tend to open up any collapsed passages and allow the distal alveoli to vent. By keeping the regulator in the diver's mouth there is also a chance that there will be air available as depth lessens and ambient pressure falls so that the pressure of the air remaining in the tank again becomes sufficient to activate the regulator.[17]

Although the ascent methods as taught may not appear to cause any fatalities directly they may be inculcating in divers a dangerous attitude to air conservation, an unstated belief than to continue diving until almost all the air has been consumed is the clever way to dive as one is not paying for more air than is used on the dive. While it might be counterproductive to teach a pupil to regard running out of air as being an irredeemable error there should be greater stress on informing pupils that scuba diving is safe only as long as the diver remains capable of making suitable adjustments to the environment. This includes having a sufficient supply of air to inflate the buoyancy vest, perform decompression, being able to accept being washed back off rocks by water power when attempting to exit, and having the option of swimming underwater when surface conditions are rough.

Because there is no certain information concerning what proportion of scuba divers habitually, frequently, or occasionally, use most of their air supply before commencing their ascent, there can be no firm conclusions drawn from the data in Table 1. But in considering the matter it should be remembered that their chances of survival would almost certainly have been considerably greater if they had to contend with even one fewer problem. There is need for a survey of scuba divers to ascertain their response patterns to air monitoring, and a confidential survey of scuba divers about occasions when they recognised a serious adverse problem might be overtaking them would be of enormous value because their survival would provide information not only concerning the problem but the successful response they employed or others provided.

Summary

The problem facing Instructor organisations in devising training protocols was accurately defined by Dr Nemiroff in 1977, at the UMS Workshop:[15]

"One of the difficulties is that we are trying to train a skill for an emergency context that requires either a high degree of skill, or extensive reinforcement, or over-learning, or all three. In a true emergency, where the mind is not working and the body is not functioning the way it should, the emergency technique that would be best would be one requiring absolutely zero skill, a zero memory, and zero reinforcement. Therefore I have no answer to what the best emergency technique is, but it seems to me that we should strive for those that require minimum skill, minimum reinforcement and yet can be considered valid exercises under the conditions."

Although not so identified by Nemiroff, such a "zero skill" exists and is the option which every diver has to avoid continuing until he or she enters the no/low air state.

Readers should consider their response to the following questions which bear on the options open to those who train scuba divers. It is recognised that instructors are required to conform to the training program set forth by the organisation which gives them authority to graduate their pupils. However no rules need be regarded as beyond critical examination and the following list of heads of discussion may serve to stimulate such an examination.

1 Is the no/low air situation to be regarded as adverse?

2 Is it avoidable by taking simple prudent care?

3 Is knowledge of emergency ascent procedures sufficient or is actual practice necessary (consider case reports)?

4 Does the usual course practice teach or only demonstrate emergency ascent methods (is over-learning achieved)?

5 Is the possible danger of air embolism during training outweighed by the benefits (note SETT experi-

ence and the absence of proof that such training has been beneficial to scuba divers who follow advised rules for safe diving)?

6 Course time is limited. Is time better spent in thorough indoctrination in safe diving practice or in practicing emergency ascents, i.e. accepting that no/low air situations are inevitable?[18]

7 Are pupils warned of the potential dangers in practicing such ascents when unsupervised by a trained instructor and of the likely decay of their skills if they are not practiced?

8 Are pupils to be advised that their buddy is likely to have a reduced air supply when theirs is no/low status and that a tank supplied buoyancy vest fails to operate when the tank is near empty?

9 Is there any evidence that there is justification for the differences in the scuba training courses defined by each major Instructor organisation?

This paper does not provide a definitive answer to such questions because there is inadequate data available (i.e. incident reports) to form a firm basis for a definitive analysis. There is a need for instructor organisations to recognise the value of the Divedata Databank concept of confidential management of reporting of all types and severities of diving-related misadventures and to take a more active stance in supporting efforts to obtain such reports.

Acknowledgements

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SCUBA DIVING AND PREGNANCY

Catherine M Leslie

Introduction

Diving during pregnancy is a relatively new concept and consideration in obstetrics. Over recent years with the influx of improved equipment and the accessibility to the equipment necessary for diving, the number of divers has greatly increased. The percentage of women divers has also dramatically increased. As few women consistently dived in the 1960s not much thought was given to the effects upon them and their unborn child whilst diving. As the number of women divers increased during the late 1970s and on into the 1980s, this is increasingly becoming an area of concern for many women. 20% of the diving population are women and they are mostly of the child bearing age.

What is worrying is that women do not know that they are pregnant until they miss a period when they are probably at least two weeks pregnant. The questions that are continually asked are "Should I dive when I am pregnant ?" and "If it is alright to dive, to what depths and time limits ?". In the following paper I will look at the information available and attempt to answer these questions according to the current recommendations.

Potential Problems of the Pregnant Diver[1]

MATERNAL FACTORS

- Morning sickness and motion sickness
- Reduced respiratory function
- Circulatory competition with placenta
- Altered sympathetic response
- Reduced fitness and endurance: unusual fatigue
- Size: fit of wetsuit, harness, etc.; clumsiness with the possibility of injury
- Effects of lifting
- Increased fat and fluid leading to increased susceptibility to DCS
- Mucous membrane swelling causing difficulty clearing ears

FETAL FACTORS

General

- Hypoxia from various mishaps
- Hyperoxia which might cause blindness, closure of ductus arteriosus, haemoglobin breakdown or consumptive coagulopathy
- Exercise hyperglycemia possibly followed by post-exercise hypoglycemia
- Exercise hyperthermia
- Physical injury
- Leaking membranes allowing infection
- Maternal envenomation causing direct or indirect damage
- Decompression producing bubbles and perhaps altered placental flow

Early Pregnancy

- Malformations related to maternal decompression sickness (DCS)
- Possible teratogenic effects of pressure, oxygen, nitrogen, dive-related medications, bubble formation and other problems
- Recompression treatment leads to exceptional exposure to O_2 and N_2
- Decompression causes bubbles and may cause birth defects

Late Pregnancy

- Prematurity (Ama diving)[2]
- Decompression bubbles may cause stillbirth

Maternal Problems

In the first trimester, especially between the sixth and twelfth weeks, there is a very definite increased incidence of nausea, vomiting and sea sickness potential. This is so even without the more obvious "morning sickness". In view of the harmful effects on the fetus of the various anti-motion sickness medications, there is no justification for taking these drugs just to go diving.

During pregnancy, probably due to the effect of progesterone, there is a progressive interference of respiratory function with difficulty with oxygenation of the blood flow through the lungs, and also an increase in the resistance in the airways to air flow. The results of this may be to reduce the woman's ability to cope with strenuous activity and perhaps also to increase the likelihood of pulmonary barotrauma.

From the fourth month onwards there tends to be fluid retention and mucosal swelling, thereby making the middle ear equalisation process more difficult, and predisposing to sinus and middle ear barotrauma.

The change in shape of a pregnant woman has unfortunate side effects. Wetsuits no longer fit. Weight belts cause backache and heavy and awkward equipment is uncomfortable. The weight gain and the change in posture results in the woman being less nimble physically and more likely to lose her balance, therefore at a higher risk of getting injured. There is also increased fatigue and the practical aspects of entries and exits.

During the last trimester some women experience a "leak" through the membranes without being aware of it. If sea water should enter the uterus because of this leak, there is a high risk of infection and/or premature labour.

The possibility of decompression sickness during pregnancy may be increased by the increased blood flow, the increased fluid retention, and the increase in blood clotting mechanisms. Many women also store increased amounts of body fat. As nitrogen is more soluble in fat than lean tissue, this increased amount of body fat might lead to the intake of more nitrogen.

Dr A Bove[3] noted that in pregnancy, supine hypotension, including syncope, may occur if the gravid uterus falls back against the inferior vena cava, reducing venous return. He suggested that assuming an equivalent position in the water could result in syncope after the first trimester. This raises the question whether a tight-fitting wet suit could produce the same pressure.

In general the Underwater Medical Society Workshop 1978[4] concluded that while mild exercise associated with diving would be beneficial to cardiovascular condi-

tioning, it is not clear how strongly the uterine artery blood flow responds to emotional stresses, hypoxia, hyperoxia, hypercapnia, or to many drugs. The well-being of the healthy pregnant woman (without consideration of the fetus) would not seem to be compromised by non-strenuous diving to depths less than 4 ATA (40 metres).

Post partum diving should not be undertaken until after the six week check up. There is a possibility of uterine infections if the os is not closed. Exercise fitness may take longer in returning so one should start with relaxed dives.

Fetal Problems

HYPOXIA

Unfortunately, when diving there are many causes for a reduction in oxygen levels of the blood and one of the major possible problems faced by the fetus is hypoxia. This is thought to increase the possibility of both miscarriage and birth abnormalities. There are several ways in which a diver can suffer hypoxia or asphyxia. The most common is drowning and near-drowning, with or without aspiration of water. Hypoxia may also develop as a result of carbon monoxide contamination of the breathing gas. The chance of a pregnant diver becoming hypoxic because of exercising oxygen debt is unlikely.

Although it is true that the fetus is equipped to survive a level of hypoxia that might kill the mother, serious intra-uterine hypoxia or asphyxia may have catastrophic consequences. Sundell[5] believes that nearly all infants who present with hyaline membrane disease have previously suffered intra-uterine asphyxia. He noted the many instances in which full term babies exposed to massive asphyxia develop cerebral oedema and multiple organ damage. He stated that his studies with sheep have confirmed these observations. Sundell reported that when the uterine artery blood flow was significantly lowered, it produced hypoxia and acidaemia in the fetus. Further, at the end of gestation, nearly 50% of the lambs so insulted in utero developed hyaline membrane disease, although none of the controls presented with this disorder.

OTHER GASES

Other gas concentrations may be altered in divers, e.g. an increase in the carbon dioxide levels due to the resistance of breathing with scuba, and also a rise in the nitrogen level, which happens with all compressed air diving. The harmful effects on the fetus which is experienced with theatre staff who inhale small quantities of anaesthetic gases in operating theatres may well be similar to the effect of breathing nitrogen under higher pressure than normal.

DECOMPRESSION SICKNESS

It has been concluded by some that the fetus is more susceptible than the mother to decompression sickness if circulating bubbles in the fetus are compared to clinical symptoms in the mother. This comparison is reasonable since the adult lung normally acts as a bubble filter, preventing gas bubbles from reaching the arterial side. On the other hand, any bubble found in the fetus' venous system has a clear pathway to the arterial side via the patent foramen ovale. Thus, circulating gas bubbles in the fetus must be considered prima facie evidence of arterial gas emboli. If this argument is valid, this suggests that, the fetus is more vulnerable to decompression sickness than the mother.

OXYGEN TOXICITY

What effect does an increased maternal PIO_2 have on fetal arterial PO_2 and therefore on possible fetal oxygen poisoning? This question was studied by Assali and his associates in pregnant sheep.[6] They found there was a significant rise in the amount of oxygen supplied to the fetus under the test conditions. On the other hand, Rankin[7] reported that the fetus will not be exposed to a high PO_2 if the mother does not breathe oxygen to a partial pressure above about 700 mm Hg, equivalent to an air dive of about 100 fsw. These findings indicate that the fetus may not become hyperoxic during a normal air dive. However, a problem may arise if the mother is exposed to pure oxygen at 3 ATA, as is common during hyperbaric oxygen therapy. In one study in sheep, the average umbilical vein PO_2 was raised from an air breathing level of 31 mm Hg to 458 mm Hg during oxygen breathing at 3 ATA[6]. This remarkable elevation of fetal arterial PO_2 raises the possibility of oxygen toxicity. Fujikura[8] has shown in rabbits that maternal exposure at these levels resulted in retrolental fibroplasia in the newborn fetuses.

There is a lack of experimental data upon which to assess the potential damage to the unborn fetus from oxygen toxicity. The rabbit and rat studies did not expose the experimental subjects to oxygen levels and times similar to those used in human treatments. Fetal rabbits are known to be especially susceptible to retrolental fibroplasia.

TERATOGENS

The fetus can be damaged by extraneous stimuli. It is this influence on the fetus, disrupting its development, that has persuaded so many women to avoid other toxic agents, such as smoking, alcohol, stimulants, etc, during their pregnancy. Other drugs which can aggravate this are decongestants and anti-seasickness tablets. These are often used during diving activities.

Little information is available on the effects of marine animal envenomation on the unborn. Even though there is no reason to believe that the Portugese Man of War stings or other envenomations, per se, are dangerous to the fetus, the injuries can cause generalized reaction in the mother which might be dangerous to the fetus.

Animal Experiments

The first known work on the effects of diving during pregnancy was published by Boycott, Damant and Haldane in 1908[9] and consisted of a series of observations noting that pregnant ewes presented intravascular bubbles at autopsy after dives to 168 fsw (6.1 ATA) for periods ranging from 15 to 240 minutes. The authors concluded that a young fetus four inches or less in length had too active a circulation and too small a bulk to develop bubbles. Further, there were no bubbles even in an advanced fetus if the exposure lasted for 15 minutes or less. This part of the study was overlooked until 1979 when attention was drawn to it by Bolton[10].

In 1968, McIver[11] exposed 28 anaesthetised pregnant dogs to a simulated depth of 165 fsw. Thirteen were exposed for a bottom time of 60 minutes, and fifteen for 120 minutes. All of the 28 adult animals presented marked disseminating intravascular bubbles at autopsy. Two of the 94 fetuses of the first group and two of the 99 fetuses of the second group had bubbles in their coronary arteries, but no bubbles were seen in any other vessels. The amniotic fluid surrounding all 193 fetuses contained numerous bubbles, but all fetuses survived the dives. Also 23 newborn pups were exposed to air at a simulated depth of 165 fsw for 60 minutes, and eight showed marked bubbling throughout the vascular system.[11]

Chen[12] observed another instance of fetal resistance to decompression sickness in 1974. While using rats to study another problem, he noted that the fetuses of anaesthetised pregnant rats did not show intravascular bubbles even though the mother developed fatal decompression sickness. Both of these studies suggested that the fetus was more resistant to decompression sickness than the mother, which caused many workers to conclude that as long as a pregnant diver did not herself develop decompression sickness, her fetus was safe.

In 1978 Fife et al.[13] called attention to the fact that the placentas of both the dog and the rat have a countercurrent arrangement between the maternal and fetal microcirculations, while the human placenta has a concurrent arrangement of the two microcirculatory systems. Since the concurrent arrangement is less efficient for the exchange of substances between mother and fetus than a countercurrent system, data derived from dogs and rats should probably not be considered applicable for humans. This concern led to a search for an animal whose fetal/maternal mirocirculatory dynamics more closely resembled that of humans. The sheep was suggested as a model.

In 1978, Fife and his co-workers[13] instrumented seven pregnant sheep by implanting Doppler ultrasonic transducers around one maternal jugular vein and one of the fetal umbilical arteries. Seventeen simulated air dives were made. It was found that a dive considered safe for humans (100 fsw for 25 minutes) produced circulating air bubbles in the maternal jugular vein. In fact, although circulating bubbles were detected in the fetus after dives to depths as shallow as 60 fsw, no bubbles were detected in the maternal jugular vein of any of the animals. Further, no clinical symptoms of decompression sickness were noted in any of the mothers after any of these dives, although it should be noted that in most cases as soon as bubbles were confirmed in the fetal circulation, the mother was recompressed immediately. It is possible that on some dives she might later have developed symptoms. These observations suggested that the fetus of an animal having placental microcirculatory dynamics similar to those of humans might be at greater risk of decompression than its mother.

It is believed that bubbles are generally created when a diver surfaces even after a no-decompression dive to less than 60 fsw. Initially these bubbles may remain in or near the peripheral capillaries, particularly in muscle tissue. They can easily be forced into circulation by muscle action, including shivering. Normally, however, in the adult these venous bubbles are filtered out by the lungs and thus prevented from becoming arterial gas emboli. Contrary to earlier views, the mother may actually develop circulating bubbles before her fetus does.[14] However, on some dives the fetus may develop circulating bubbles before the mother presents clinical symptoms of decompression sickness. Because the fetus has a patent foramen ovale, its lungs are not able to serve as a bubble trap, and umbilical artery bubbles quickly become arterial gas emboli with potentially serious consequences. Thus umbilical artery bubbles should be regarded as a grave threat to the fetus, in contrast to the usually benign consequences of a modest number of venous bubbles in the adult.

It appears that the age of the fetus may affect its susceptibility to decompression sickness. The Boycott, Damant, and Haldane 1908[9] study in sheep showed that fetuses 100 mm (4 inches) or less in length did not present with bubbles, while those over 100 mm (4 inches) often did. Studies by Fife et al[13] also suggest that fetuses in the first trimester may be more resistant to decompression sickness than those in the third trimester. However, in early embryonic development destruction of even a single cell may result in serious or lethal abnormalities when growth and differentiation have taken place. For this reason, the consequences of decompression sickness in the embryo or early fetus may be worse than in the fetus near term.

Human Data

The Ama are the free diving women of Korea. These breath-holding Ama divers, who dive up until a few days before childbirth, have a 44.6% incidence of prematurity with an infant of less than 2.5 kg; compared to 15.8% in the non-diving females of the same district[2].

SUSAN BANGASSER[15]

In a survey where 72 women who dived whilst preg-

nant were questioned, one third stopped in the first trimester when they found out they were pregnant. More than a third stopped during the second trimester mainly due to increased size, and the rest continued on. 39.4% claimed they maintained the same level of diving activity during pregnancy.

Most were seasoned and competent divers. The deepest dive was to 54 m (180 ft) and the average depth was 18 m (60 ft). The average age was 30 years. Nausea in the first trimester sometimes kept a diver out of the water. There was no decompression sickness in any of the mothers. 5 decompression dives were made.

The babies delivered by pregnant divers were all normal according to their mothers. One babe was underweight at birth. The complications that occurred during pregnancy include; one premature birth, one septic abortion (caused by an accident, not diving related), two miscarriages (one woman had four miscarriages in all, 2 prior to learning to dive and 2 after). All but one woman dived after pregnancy. Fifteen women dived during more than one pregnancy.

MARGARET BOLTON[10]

Information from 208 women of whom 109 dived during pregnancy and 69 did not. This showed a raised incidence of abortion, stillbirth, low birthweight, neonatal death and congenital abnormalities. Twenty women dived to 30 metres or more, during the first trimester, 3 had babies with congenital abnormalities. The incidence is normally 1:50 of pregnancies. This figure is 6 times higher. Four others diving to lesser depths had babies with other congenital malformations (2 with congenital heart disease and 2 with minor abnormalities). There are no recorded malformations in the babies of mothers who did not dive during pregnancy. More than 6% of the babies in the diving group were small for gestational age compared with only 1.4% in the control group.

CASE HISTORY BY G TURNER AND I UNSWORTH[16]

A report on a baby born with arthrogryposis and some dysgenic features whose mother went scuba diving in early pregnancy. She was a 22 year old primi gravida. Both parents went on holiday from the 40th-55th day after last normal menstrual period. The mother dived at least once daily to a total of 20 dives in the 15 days. Most dives were to a depth of 18 m or less but 3 were to 30 m and 1 to 33 m. The ascent rate used by the mother and her husband was 18 m per minute, though this was usually estimated rather than actually timed. When decompression was required, a modified version of the USN tables was used. All the dives except one were without complications, the exception involved an “equipment failure” of the husband whom she was buddying, at the end of a strenuous 15 minute bottom time dive at 18 m. The rate of ascent of both was described as “very rapid”. She felt well but tired after this dive.

No medications were used apart from oral Sudafed 60 mg on 2 or 3 occasions to aid ear clearing, early in the holiday.

The rest of the pregnancy was uneventful. The abnormalities noted in the baby were unilateral ptosis, small tongue, micrognathia and short neck. The penis was adherent to the scrotum. The upper limb joint movements were all normal except for the hands. The fingers were in fixed flexion with some webbing between 3rd, 4th, and 5th fingers, the thumb was digitalised but had two phalanges. The hip joints were dysplastic with reduced range of movement and one hip was dislocated. There was fixed flexion deformity of the knees and bilateral equino-varus deformity of the feet. The head circumference was normal and motor development was appropriate for the baby’s age at 3 months. Karyotype, electromyogram (EMG) and muscle biopsy were normal.

The embryopathic timetable of thalidomide affected the upper limbs around the 40th day and the lower limbs between the 41st and 45th day. Thalidomide specifically affects the migration of cells destined to form the posterior root ganglia. Arthrogryposis is presumed to result from either muscle disease or abnormalities of the cells forming the anterior root ganglion so the same timetable may be applicable.

Recommendations

UNDERSEA MEDICAL SOCIETY WORKSHOP[4]

1 There is no contraindication to diving for the normal, healthy non-pregnant female. The same general health criteria should apply to both male and female divers.

2 The fetus may be at greater risk than the diving mother. The potential risk primarily consists of decompression sickness, but hyperoxia, hypoxia, hypercapnia and asphyxia may also be involved.

3 There is insufficient experimental evidence at this time to establish diving depth and time profiles that are definitely not hazardous to the human fetus. Although a large number of women have dived while pregnant, results of epidemiological studies on the fetal effects of these dives on the fetuses have not yet been thoroughly analysed.

4 The number of unanswered questions about the effects of diving on the human fetus should encourage physicians to inform their patients of the potential risks and to advise them to act in the most conservative manner.

5 Pregnant women who choose to dive against medical advice should be informed that the potential risk to the fetus apparently increases as the no-decompression limits are approached, as the oxygen tension of the inspired gas

increases, and perhaps also a function of other factors that remain to be identified.

6 Until further studies are made, we recommend that women who are or maybe pregnant not dive.

DR JOHN BETTS[17]

Advises pregnant divers not to dive deeper than 20 metres and to cut their dives short by taking five minutes off the no-decompression time for their dive. Also in addition any woman trying to start a family should do likewise. He feels that those who follow the rules would seem to have little cause for worry.

SUSAN BANGASSER[15]

She recommends that for the pregnant diver considering diving that this is not the time to learn to dive. For the experienced diver, it is recommended that she takes things a little easier, making one dive a day, avoiding difficult surf, and using common sense. That the pregnant diver stay in less than 10 msw (33 ft). She feels that scuba diving is a great physical and mental conditioner, but common sense must always prevail. If a woman does not feel well, for example nauseated, cramps or whatever, she should not go diving.

There is a problem for the woman, who dives the first six weeks of pregnancy, because she may dive deeper than desirable, not realising that she is pregnant. The only guide here, is if a woman has discontinued birth control and plans to have a family, to begin diving shallower. This may mean a lot of shallow diving for some, but a few deeper dives can be fitted into the schedule. Once she has had her period, and it is over (usually this means she is not pregnant but not always) would be the time for any deep dives she may wish to make. She should dive shallow once ovulation or conception could be possible.

ANONYMOUS AUTHOR IN SKINDIVER MAGAZINE[18]

Dive to 9 m (30 ft) and snorkel only on the surface. For the less conservative, dive to 18 m (60 ft) to half the time in the US Navy tables. Avoid exertion and stay warm.

TURNER AND UNSWORTH[16]

It would be wise for women to refrain from diving below 9 m if conception is a possibility or if they are pregnant. A course of perfection would be to abandon diving for the duration of the pregnancy.

DR KEN KIZER[19]

Until further studies establish the safety of scuba diving during pregnancy, it is recommended that all diving activity be stopped as soon as a woman thinks that she might be pregnant and that no diving is done during the duration of the pregnancy.

Similarly, women who are trying to become pregnant should stop scuba diving before attempting to become pregnant. However if an unplanned pregnancy does occur, there is no reason ,at this time, to recommend having an abortion just because diving was done after the beginning of pregnancy.

For the inveterate divers who cannot keep out of the water no matter what the risks, he recommended that these women limit their dives to less than 9 m (30 feet), avoid repetitive dives or getting chilled or unduly fatigued, do not dive when size becomes a problem (usually after the sixth month) and otherwise dive very cautiously and conservatively.

DR CARL EDMONDS[20]

The question as to whether a pregnant woman should dive has answers that are very complex. However the final decision must be left to the diver.

There may be an issue between personal liberty and a conservative safe attitude. It is likely that those people who insist on the former, will continue to dive no matter what is said to them. It is also likely that the latter will not dive because it could never be proved 100 per cent safe.

E H LANPHIER[1]

"A birth defect, with the possibility that it was caused by diving, would be a very high price for any benefit that diving in pregnancy could possibly confer."

Conclusions

From the evidence displayed by the animal experiments especially those carried out on sheep (whose placental microcirculatory dynamics are similar to humans) the fetus is more likely to develop DCS than its mother. The workers involved with these experiments have concluded that at this stage it is not advisable for pregnant women to dive.

In the studies done on humans we see some evidence in Bolton's study[10] that there are more defects in the babies born to mothers who dived while pregnant. Bangasser's study[15] appeared to show little detrimental effect on the babies. However the case history presented by Turner and Unsworth[16] was quite frightening. I feel that more studies on the actual effects of diving during pregnancy on humans need to be done, but how can this be done? How many women will continue to dive for the benefit of a study if they are aware of the risks involved to their unborn child. Also the women who have experienced some form of problem during their pregnancy or given birth to a child who has some form of birth defect is more likely to have responded to the questionnaires used by Bolton[10] and Bangasser[15]. So it may be possible that the percentage of birth defects is not a true reflection of the number of babies with birth defects from mothers who dived during pregnancy.

There is now the legal aspect of advising a woman to dive or not to dive during pregnancy. In the 1985 workshop on Women in Diving, Innes[21] remarked ".....for those of you who are here who are instructors or practicing obstetrics or gynecology, you would be well advised from your attorneys to advise your clients not to dive. All you have to do is put one of these studies before a jury and you are sunk if your client had birth defects, ...nothing is foolproof when you have got a baby with a missing hand in front of the jury". This seems to well cover the legal aspect.

For some women, as there is no absolute proof that diving is any more dangerous when they are pregnant, they will continue to dive. As with smoking and alcohol many women will not give these up although it has been shown to be detrimental to the baby's growth and health. It really is a matter for personal choice, as long as women have been informed about the pros and cons of diving in pregnancy.

The time, place and amount of diving done by these pregnant women may also depend on where she lives and her local dive areas and where she may holiday. I think that most women probably give up diving either when they know that they are pregnant or because of morning sickness or sea sickness. In Melbourne a wetsuit is needed for all diving even during summer. As with increasing size the wetsuit gets too small and the discomfort of wearing a weight belt will make most women still diving give up at this stage. However for women living in warmer, tropical areas or visiting these places whilst on holidays, the lure of diving may be greater as it is not necessary to wear a wetsuit. The rationale that may be used by some women, especially while on holiday, is that this is a new and exciting dive location and that they will not do many dives and will limit their bottom time, after all this is a holiday and a few dives cannot hurt. Again this is a personal choice, providing that it has been made as an informed decision.

Following the research that I have done regarding scuba diving and pregnancy, at this time I personally will not dive during pregnancy. In agreement with Lanphier[1], I think that the risk of having a child with a birth defect outweighs any pleasures gained from diving. Women generally believe that they will have a "normal" baby with no problems and most will avoid doing things that may place their baby in jeopardy.

I also think that a non-diving approach would be advisable while trying to get pregnant. The theory used by Bangasser[15] for the time for deep diving could be used for the time of any diving following menstruation and the anticipated time for ovulation thus the possibility for conception. I would be happy to settle for surface snorkeling if holidaying in the tropics or the like whilst pregnant.

A conservative approach is needed when no-one is really sure what the effects of diving are on pregnancy. This attitude naturally can be changed if new information or evidence is found showing that there is little effect on the mother or the fetus. However, at present the old adage still holds. It is better to be safe than sorry !

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PAPERS FROM THE JOINT SPUMS AND ROYAL HOBART HOSPITAL MEETING NOVEMBER 1988

CARBON MONOXIDE POISONING

Alan Wood

Here is an outline of the problem of carbon monoxide (CO) poisoning in Tasmania. These figures do not cover accidental deaths.[1,2] It should be noted that a lethal concentration of CO can be reached in a closed garage in ten minutes.[3]

	1981	**1984**
Total Suicides	63	52
Gassing and other Vapours	14	N/A
Male to Female	5 to 1	3 to 1

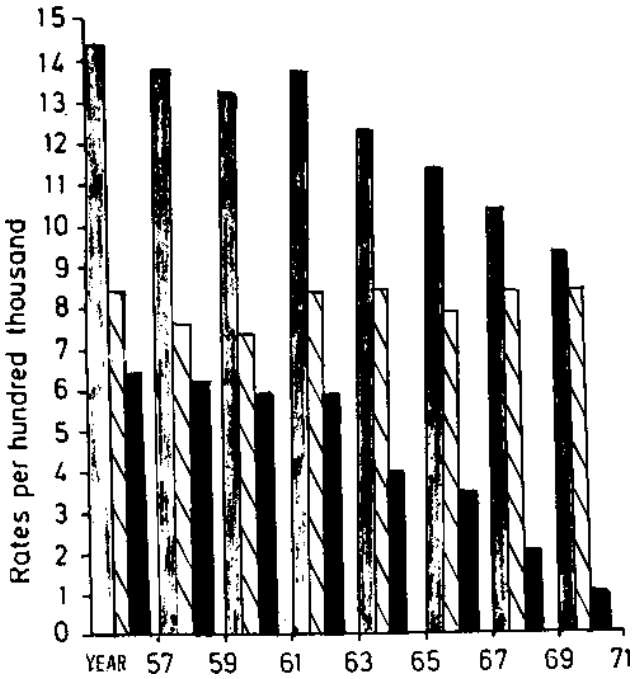
Figure 1 shows the reduction of the number of people using gassing as a means of suicide in Britain since the introduction of North Sea gas[4]. It makes the point that the availability and lethality of methods of suicide are important determinants of the numbers dying by these means.

More surprising is that when one looks through the older literature to determine outcome of survivors of poisoning with carbon monoxide it is very difficult to find good articles. This poses a problem as there is not a good yardstick by which the outcome of populations treated by hyperbaric oxygen can be measured. A commonly quoted article[5] states that of 63 survivors (no hyperbaric treatment) followed up at three years, 43 % had memory impairment, 33 % had deteriorated personality, 11 % had what was described as "Gross damage".

Reviewing more recent articles serveral themes emerged as I went through the data. The first issue that arises is the usefulness of carboxyhaemoglobin levels. It seems clear from the literature that there is a lack of correlation between carboxyhaemoglobin levels and clinical status[6]. Hence these levels should not override a full clinical assessment, and it may be quite dangerous to allow this one parameter to dictate treatment. Tissue levels of carbon monoxide are what needs to be measured and this cannot be performed in a clinical laboratory. Higher cognitive functions (such as memory, attention, concentration, calculation, etc.) may be more easily assessed and reflect impairment of the high metabolic rate tissue of the central nervous system (CNS).

FIGURE 1

UK SUICIDE RATES BY MODE OF DEATH 1957-1971



Solid columns are carbon monoxide deaths, hatched columns are non-carbon monoxide deaths and stippled columns are total deaths from suicide. The graph has been constructed from data in reference 4.

A second point raised in the literature is the great usefulness of hyperbaric oxygen both for acute and delayed presentation. The ethical considerations of a control group comparison of such an effective treatment have been addressed to some extent.[7]. In 1985, 215 poisoned patients were divided into good and poor groups. Those in the good group were conscious, with lower carboxyhaemoglobin levels and well enough to be psychometrically tested. They were given normobaric oxygen. The poor group were unconscious with high carboxyhaemoglobin levels and were treated with hyperbaric oxygen.

As a medical student I recall being taught Haldane's view that the toxicity of carbon monoxide was due entirely to its power to combine with haemoglobin in the red blood cells thus putting them out of action as oxygen carriers. This could be called the simple hypoxia theory. However it does not fully explain all of the clinical findings. A key point in the literature is the work of Goldbaum.[9] He transfused carboxyhaemoglobin into dogs with little ill effect. This highlights the point that tissue toxicity is probably one of the major determining factors in outcome and there is no easy way of measuring that.

TABLE 1

	No sequelae	**Died**	**Sequelae**	**Sequelae after delayed hyperbaric therapy**
Poor group (Treated with hyperbaric oxygen)	89 %	9 %	2 %	Not applicable
Good group (Treated with normobaric oxygen)	88 %	0	*12 %	0

* This group of the good population was then given delayed hypoerbaric oxygen and all recovered

Two points should be made. Firstly giving "mildly" poisoned cases normobaric oxygen produces a large number of sequelae. Secondly, the sequelae so produced can be successfully treated by hyperbaric oxygen therapy.

A similar article[8] said the same sort of thing, pointing out that long delays in starting treatment were not crucial although they are not advantageous. Purely psychiatric presentations of carbon monoxide poisoning offer a wide range of symptoms. Reviewing the literature revealed a lobe by lobe picture of problems. More frequently listed were dementia, psychosis and Parkinsonian syndromes. More rarely Korsacoff like syndromes (an amnestic disorder usually associated with alcoholism, with profound short term memory disturbance) were mentioned while other presentations included cortical blindness and multiple sclerosis-like pictures. The list is quite extensive and often these patients present in quite a bizzare fashion. For instance an elderly man was twice taken to casualty by neighbours complaining of visual hallucinations and disorganised behaviour. He was sent home on two consecutive evenings, and then on the third evening he was admitted. It transpired that he was being poisoned each night by his caravan's gas water heater.

The picture of chronic or sub-acute poisoning seems to be quite complex. The relatively slow induction would seem to be the mode of poisoning most likely to result in neuro-psychiatric presentations and again carboxyhamoglobin levels are not useful (Table 2).

A complex point is that the relative affinity of tissue cytochrome systems for oxygen is higher than for carbon monoxide. Hence tissue hypoxia plus carbon monoxide are required to poison the tissue cellular system. Simply having carboxyhaemoglobin bound tightly to haemoglobin in red blood cells may not necessarily mean that the tissues become poisoned. This may explain the complexity of clinical pictures encountered. So simple hypoxia due to carbon monoxide and tissue toxicity acting together is better able to explain the clinical findings including the value of hyperbaric oxygen in treatment. This provides the most rapid

TABLE 2

DIFFERENCE BETWEEN AVERAGE CARBOXYHAEMOGLOBIN LEVELS IN UNCONSCIOUS PATIENTS WHO DIED AND THOSE WHO SURVIVED[6]

		Average COHb	**Range of COHb**
Died	11	30.8 %*	19.7 - 49 %
Survived	33	29.3 %*	1.1 - 53 %

*****The difference does not reach statistical significance.

and logical method to displace carbon monoxide from the tissues as well as from haemoglobin. This will minimise tissue damage to high metobolic rate tissues. Clinically most cases present with damage to the high metabolic rate tissues of the CNS or the myocardium.

Case history

I would like now to present a case history of a twenty seven year old male with no past psychiatric history, physically very fit and in good health. My involvement started 24 hours after admission when consultation was requested with Liaison Psychiatry. The history, which was pieced together from police reports and contacts with friends and relatives, revealed that he was last seen at 9 a.m. and was found unconscious in his car in an isolated place at 5 p.m. He was slumped in the car with the ignition turned on, but the engine not running and the petrol tank half full.

There was a vacuum cleaner hose from the exhaust into the car. The rescuers did not note a strong smell of exhaust fumes in the car interior which is most unusual in these cases. When one opens a car door in this situation usually the fumes are so overpowering that one almost faints at the first breath. So the question was raised as to the duration and intensity of exposure to exhaust fumes.

In casualty he was found to be agitated, irritable and combatitive when examined or interferred with. He was lapsing in and out of consciousness but always capable of responding purposefully to pain. The carboxyhaemoglobin level was low at 2.5 %. This did not tie in with the CNS clinical state. He was placed on normobaric oxygen. Psychiatric management the next day focussed on the request to manage this serious suicide attempt. His mental state had not improved, and now he was not responding purposefully to pain and had decorticate posturing with extensor plantar responses. The confusion at this time centered around the question of poisoning with other substances and perhaps it was considered by non-psychiatrists that he may have a dissociative disorder of some sort. Hyperbaric therapy was suggested. However because of the clinical confusion which was hoped to be resolved by investigation, as well as problems with the family who initially rejected the idea of hyperbaric therapy because it was unproven, it took eight days before he came to treatment. Logistical problems arose because this man had been agitated in casualty and had to be restrained so the question of safety of patient and attendant in our small tank arose.

As a result of these and other problems the time lag between what we would have liked to happen and what did happen was eight days. In response to treatment his level of alertness and arousal improved a little, however there was no return of cortical function. Further problems with the family resulted in a further six days elapsing before the next treatment. There was no significant change after this treatment. He was eventually placed in a rehabilitation hospital. The outcome five months after the poisoning was that he is able to vocalise unintelligable sounds. He is said by the staff to recall, very infrequently, their names. He can not communicate any of his needs nor care for himself. He is violent occasionally, if uncomfortable with a full bowel or bladder, and hits out at the staff. Previously his CAT scan was normal but now it reveals marked cortical atrophy and increased ventricle to brain ratios and infarction of the basal ganglia.

This man has profound dementia and long term placement out of the rehabilitation setting in a nursing home is being looked for. So despite hyperbaric treatment (which was delayed) this patient did not do at all well. It is a very interesting example of how the very clear literature often does not seem to reflect how things are in real life.

Summary

The diagnosis of carbon monoxide poisoning requires a fairly high index of suspicion particularly with neuropsychiatric presentations. However most cases have organic features of cognitive impairment, impaired attention, concentration and memory. Carboxyhaemoglobin levels do not indicate the severity of the poisoning as this case shows well. Carbon monoxide levels in plasma might be more important but cannot be measured, but dysfunction in high metabolic rate areas such as the brain and myocardium can be measured[10], but by different means, such as cognitive testing and mental state examination. Poor emergency room state does not preclude full recovery and it is claimed that minor delays in instituting treatment are not crucial, although I have my doubts about that. Hyperbaric therapy produces fewer end point defects and is also effective on defects left by normobaric treatment.

Perhaps the final point is that a crucial part of assessment of a patient is the assessment of higher mental functions. These may be one of the most sensitive indicators of tissue toxicity that we have available. The other indicator, the ECG, seems less sensitive.

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TREATMENT OF CARBON MONOXIDE POISONING

Chris Lourey

Introduction.

The subject of carbon monoxide poisoning really has a rich history and its toxic affects were actually first noted by one of the founding fathers of modern medicine, Claude Bernard, in 1857.[1,2] Most of what I say this morning is really a compilation of evidence and recommendations that have been progressively made over the last sixty years which probably indicates that medicos are pretty slow learners.

My personal interest in carbon monoxide was enhanced when the Victorian section of the South Pacific Underwater Medicine Society gathered all the necessary data to make the ministerial submission to get the hyperbaric facilities for the State of Victoria established at the Alfred Hospital. This required an assessment of projected case load, clinical efficacy and cost effectiveness of hyperbaric oxygen (HBO) therapy pertinent to the State of Victoria. The clinical indications were category 1 indications as recognized by most authorities.[1]

The analysis of the data revealed a significant disparity in the incidence of carbon monoxide poisoning in Victoria when compared with other demographic centres, and unless Victoria is very different (which it is, if you listen to New South Welshmen) at best carbon monoxide poisoning in the State of Victoria was being inadequately treated. But what I think was that it was not even being recognized. A conservative estimate depending on the literature source and areas is a case load of somewhere between 20-50 cases per million people per year. In 1985 in Victoria there were 3 reported cases. In addition accruing clinical evidence challenges the clinical efficacy of treatment with normobaric oxygen as is recommended in standard texts. So in this context I will present a brief overview.

The disease

Carbon monoxide is a colourless, odourless, non irritating gas produced by the incomplete combustion of carbonaceous materials. The commonest sources are fire, automobile exhaust, petrol or propane engines, especially when operating in confined spaces, and generally, the colder the climate the greater the danger of carbon monoxide poisoning when operating engines in confined spaces, charcoal burners and faulty furnaces. Natural gas does not contain carbon monoxide, however if combustion is incomplete because of a bird's nest or a dead possum or accrued rubbish in the flue, carbon monoxide production will occur. Most of the commercial paint strippers contain methylene chloride which is readily absorbed through the skin and mucus membranes and metabolised into carbon monoxide.

The classical text book description of carbon monoxide poisoning, cherry red mucus membranes and skin, is not commonly seen and therefore is a most unreliable clinical aid. Because the initial symptoms of toxicity mimic other disorders such as influenza, acute confusional states and coma, a very high index of suspicion is necessary for diagnosis.

The strong possibility of dual pathology such as cardiac disease also must be considered. What is very interesting is that the highest percentage of deaths in fires (which the general population think is due to burns) is in fact due to carbon monoxide poisoning, and carbon monoxide poisoning in combination with coronary artery disease and burns. I ask the question rhetorically "In how many patients when admitted to a hospital for burns is a carboxyhaemoglobin level done ?"

Pathology

The primary toxic effects of carbon monoxide are those of tissue hypoxia. The biochemical lesion involves a preferential binding of carbon monoxide with haemoglobin, myoglobin and cytochrome oxidase A3, all are pyrole iron complexes.

Evidence also suggests that, in the brain, altered mitochondrial activity is not immediately reversible with re-perfusion and re-oxygenation.[3] This explains some of the symptoma-

tology one sees. In addition the oxygen dissociation curve is pushed to the left, further exacerbating the physiology of hypoxia.

The organ systems most affected are those with a high metabolic rate and oxygen utilisation, especially the myocardium and central nervous tissue. The affinity of carbon monoxide for haemoglobin is approximately 200 times that of oxygen. The severity of the symptoms will depend upon the concentration of the carbon monoxide, the duration of the exposure, the haemoglobin level and the level of metabolic and physical activity. For example, in a given time frame, one expects a greater concentration in the fire fighter exposed, or the worker exposed, than one would in the individual who has taken a combined overdose of barbiturates and then placed himself in the car. The metabolic rate of the latter is lower.

The previous health or accompanying disease of the affected individual also affects the outcome. Generally, and again I stress generally, such is the disease, no symptoms will develop if the concentration of carboxyhaemoglobin is 10% or less with acute exposure. I stress acute exposure as chronic exposures have been shown to impair mental function.

Exposure to 0.05% carbon monoxide for 1 hour with light physical activity will produce carboxyhaemoglobin levels of up to 20%. This will produce "flu like" symptoms, malaise, headache, loss of concentration, which are all very vague. Greater physical activity or longer exposures to that concentration will produce saturations of between 30-50% of carboxyhaemoglobin. Symptoms at these levels will vary between weakness and headache, to that of acute confusional states. Exposure to 0.1% for 1 hour will produce 50-80% carboxyhaemoglobin levels which left untreated will result in coma, respiratory failure and death.

The histological findings in cases where death was immediate shows the picture of hypoxia, with petechial hemorrhages in both brain and myocardium and areas of necrosis in the myocardium. In cases where death occurs 24 hours to some days later, one sees necrosis in the globus pallidus and the substantia nigra with generalised oedema. There is cellular disintegration in both nerve and glial cells. In deaths days to weeks post intoxication, one sees substantial brain softening with necrosis. Phagocytosis occurs in extensive areas, again particularly the globus pallidus and substantia nigra. There is accompanying demylination of white matter. All are evident with CT scanning.

The latent or lucid period of the inadequately treated victim is extremely variable, anywhere between 1 to 21 days. Symptoms vary from cloudy thinking, malaise to frank psychosis and a development of a Parkinson-like syndrome. In the absence of hyperbaric oxygen therapy the reported incidence of progressive neurological sequelae is up to 40%.

Treatment

Firstly the treatment is removal of the patient from the toxic environment, establishment of an adequate airway, correction of acidosis and treatment of associated pathology such as arrhythmias, control of fitting and burns etc. The specific therapy is oxygen. The generally held belief that one atmosphere oxygen is satisfactory is no longer tenable in the light of accrued evidence. It stands to reason that if an FIO_2 of 760 mm Hg is preferable to 160 mm Hg for the removal of carbon monoxide and increasing the available oxygen, then 1200 mm or 1800 mm would be better within the limits of oxygen toxicity.[2] This together with the high incidence of neurological sequelae should have laid to rest the normobaric regime, but sadly this is not the case. The latest edition of Harrison[4], the standard text for medical students, concentrates on normobaric therapy, supportive measures such as packed cells and the correction of acidosis. The role of hyperbaric oxygen therapy is only mentioned as being useful in seriously poisoned cases. How does one define useful or serious? As I said this is the standard text for medical students. Ignorance has become an art form!

Hyperbaric treatment

In 1985 Roy Myers[5] from Baltimore did a prospective study of carbon monoxide poisoning. This demonstrated both the subtle and the sinister nature of this disease and the high incidence of relapse in the normobarically treated group. In his group, 131 patients were treated with hyperbaric oxygen therapy. His criteria for HBO was a carboxyhaemoglobin level of 30% or greater and/or neurological signs or symptoms and/or abnormalities on psychometric testing. There were no relapses in this group. The criteria for treatment with normobaric oxygen was carboxyhaemoglobin of less than 30% and no neurological signs and no demonstrated psychometric abnormalities. There was a 12.1% relapse in this particular group. The relapses occurred in 1 to 21 days, the average was 5. They were then treated with hyperbaric oxygen therapy. There were no further sequelae subsequent to this.

Norkool and Kilpatrick[6] from the Virginia Mason Institute in Seattle reviewed their cases between 1978 and 1984. There was a mortality of 9.6% and a major morbidity of 1.9%. Of this 1.9% all were comatose on arrival. The major morbidity and mortality occurred in those who were found 48 hours after exposure or who had a delay of 48 hours before hyperbaric oxygen therapy was initiated. Full recovery occurred in 88.5% of cases.

Mathieu[7] and his colleagues in Lille, Northern France, reported 230 patients in 9 months with carbon monoxide poisoning. When one drives to the area where Mathieu works it is surprising the whole population is not suffering from carbon monoxide poisoning. The air is very polluted. 203 cases were treated with hyperbaric oxygen therapy and there were no chronic sequelae. The mortality was 1.9% and again

the mortality was in the group where there was a long delay before presentation to a hyperbaric unit.

Timchuk[8], from Moscow, reported 33 patients with carbon monoxide poisoning, all were treated with hyperbaric oxygen therapy. Recovery was 100% with no chronic sequelae.

Recommended treatment

Carboxyhaemoglobin levels of 15% and greater and/or neurological abnormalities and/or cardiac irregularities as demonstrated on the ECG and/or a general feeling of being unwell. all require HBO therapy. Wide indications but untreated it is a very nasty disease.

Australia is fortunate in that it possesses an emergency hyperbaric retrieval service nationwide and a level of cooperation and coordination between hyperbaric units not experienced in other parts of the world. The deleterious implication of impaired CNS mitochondrial function in carbon monoxide poisoning, I think offers sound reasons for providing clinically affected individuals with hyperbaric oxygen therapy regardless of their presenting carboxyhaemoglobin level.[3] In the absence of hyperbaric oxygen therapy there is a significant incidence of chronic progressive encephalopathy of up to 40%.

The cost to both the individual and to the community of a progressive encephalopathy is very expensive indeed. Legal settlements in excess of $2,000,000 have been awarded by the courts in the USA against hospitals and individual practitioners who have failed to consult and/or refer cases of carbon monoxide poisoning to a hyperbaric unit.[2] Will it take a similar impetus in Australia for this treatable disease to be recognize and adequately treated ?

It is interesting that the potential benefit of hyperbaric therapy in this disease was first noted by Haldane in 1895.[9] The first reported clinical applications were by Smith and Sharpe in Glasgow in 1960.[10] The number of studies increase and accrue each year, yet traditional thinking and practice still views hyperbaric oxygen therapy with a jaundiced eye. I think what is needed is an educational programme targeted particularly to the emergency services area, police, firemen, ambulance, rescue services, our colleagues in the emergency services area and importantly medical students. I think often the most difficult area is to educate and convince our medical colleagues. In this regard I am reminded of the tortoise, who only advances by sticking his neck out.

Resources are always a problem, but I think it can be demonstrated, certainly in the areas of decompression sickness, carbon monoxide poisoning and anaerobic infection, that HBO is a very cost effective means of treatment. It was really the cost effectiveness of the treatment that convinced the Minister for Health in Victoria. He also raised his eyebrows slightly when it was noted to him that there had been legal settlements of in excess of $2,000,000 awarded against hospitals in the USA. In our health system it would be public hospitals. When the NSCA unit closed down, and the unit has been offered to the State it was a very compelling argument.

Conclusions

The conclusion we can draw is that the outcome does not necessarily correlate with the carboxyhaemoglobin levels and the clinical state on admission. Patients must be observed closely, preferably in a hospital environment. I think that high index of suspicion is probably the most important thing and we will only achieve that when we educate people. But the area which surprised me was the information being given to medical students. That just continues a level of ignorance down the track and it is very difficult to change people's ideas when they have been given the wrong information in their training.

Upon discharge, the relatives must be given clear, firm instructions to report back to the hospital post haste if there is any change in a patient's behaviour or well-being.

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Comments on the two papers

Dr Mike Martyn

We put this subject on the programme was for exactly the reasons that have been explained by Dr Chris Lourey. In the six years that I have been in Hobart we have only treated two cases of carbon monoxide poisoning. At the Royal Hobart Hospital for the whole of 1983 there were only 3 cases admitted with the diagnosis of carbon monoxide poisoning. Firstly people just do not recognise the disease and secondly, they are not providing any active treatment. A lot of the survivors do have major problems afterwards and they are just being referred on to the psychiatrist to sort out the reason they took the carbon monoxide in the first place.

There are cases who have had carboxyhaemoglobin levels in the region of 1 and 2% who have profound neuropsychiatric sequelae. Dr Wood's case had a level of 2% and yet he clearly had carbon monoxide poisoning. The neurologist's comment was that this is the classic finding of carbon monoxide poisoning. The etiology of his disease can not be argued and yet he only had a level of 2%. So one has to be guided by the clinical condition of the patient, the higher quality functions particularly.

About 2 years ago in the Journal of Occupational Health that there was a report on people who had been exposed to chronic low levels of carbon monoxide in the workplace and certainly that there was evidence there that they had impaired cognitive function. Unfdrtunately I cannot remember the reference.

Dr. Chris Lourey

An interesting comment was made by one of the doyens of hyperbaric radiology in Britain when asked about carbon monoxide poisoning in the United Kingdom. He said "We do not see very much of it these days since we got North Sea gas". In fact carbon monoxide poisoning is still probably the commonest cause of poisoning.

Dr Ian Unsworth

The comment about the British scene, which used to have about 2,000 deaths a year from carbon monoxide poisoning, is accurate.

I think a lot of cases are occurring in Australia and they are not being referred for adequate management. I think the way we may have to go is the way of proposed or potential litigation. If we bring to the attention of our medical colleagues the case in the United States where a few million dollars were awarded because of the perhaps inappropriate management, I think our colleagues might sit up and take notice.

We do need to educate our colleagues very much and the areas Dr Lourey picked out were absolutely appropriate, accident and emergency, the police and the ambulance personnel, because the long term sequelae can be averted by appropriate use of hyperbaric oxygen.

Dr Lourey's net was wide but I think it was very very appropriate. At the moment the level of carboxyhaemoglobin that is usually quoted above which hyperbaric oxygen should be given in the perfectly normal patient is usually 25%. You brought it down to 15%. I certainly would not argue with that because of the difficulty of accepting any level of carboxyhaemoglobin is in any way always related to the CNS manifestations. I believe in the future we may in fact have to drop any investigation of carboxyhaemoglobin because it puts people off treating patients. When one can do cytochrome A3 measurements, which may involve taking a brain biopsy we may have better correlation with the clinical presentation. I heartily agree with everything that both speakers have said so eloquently.

Commenting on Dr Wood's case I think that he was probably too late in getting the initial hyperbaric oxygen therapy. After re-perfusion and re-oxygenation cellular activity in the brain does not return to normal straight away, which explains why individuals who are treated hyperbarically and improve significantly or return to normal after their first treatment, may relapse some days later. Often they may need 2, 3, 4 or 5 treatments before full recovery.

Dr Mike Martyn

I do not think one can specifically state that he would have made a 100% improvement and joined his brother in journalism, but what one can say is that there is a chance that he could have been in a better clinical state than he is now.

COLD AND THE DIVER
Physiology and First Aid of Hypothermia

Ian Millar

In deep commercial diving, with long in-water times, and especially if breathing helium/oxygen mixtures, cold exposure is a major problem that has been extensively studied. Hot water suits and insulated helmets are the dress now most often used off shore to prevent heat loss to the water. For deeper dives however the heat loss from the lungs to cold, dense breathing gas can be so large that gas heating becomes necessary If not adequate pulmonary problems can arise and hypothermia can occur despite maximum hot water suit heating.

This article however is aimed more at those involved with recreational scuba activities and other forms of air-breathing diving. In these situations, the increased risks of diving that results from cold exposure are most important. All who venture onto or under our Southern Australian waters are aware that cold can be an important factor in their activity. The scope of the increased risks that cold exposure can cause the average diver is not, however, widely appreciated. Hypothermia is only one of these risks of cold stress, however should it occur appropriate first aid and medical treatment are important. Regrettbly, many misconceptions continue to0 be taught and it is hoped that the following may help to dispel some of these.

Physiology

Man is a homeotherm, with a core temperature that is usually held within an approximately 0.5 degree range close to 37°C. Short term reversible variations of up to 2°C both up and down can however occur in healthy people. It is not uncommon for elite marathon runners to finish a race with a core temperature near 39°C and this level is probably approached by many when vigorously exercising in the heat or after a prolonged sauna or hot tub exposure. Likewise, significant cold stress can in some circumstances, such as when surfing or diving, reduce the core temperature significantly. A characteristic of such changes in core temperature is that the victim takes a prolonged time, up to several hours, before feeling fully recovered (and perhaps ready for a repeat exposure) even when there is no significant exhaustion involved.

Whilst immersion in water, which has a heat conduction of twenty-three times that in air and heat capacity per litre over 3,000 times that of air, is the main source of heat loss for divers, evaporative loss when out of the water should not be forgotten. Warming one litre of 10°C sea water to a comfortable 30°C requires only 80 kiloJoules. Evaporating the same water however requires some 2,500 kJoule, much of which may be extracted from the body under the soaked clothing or wet suit! Although neoprene provides good insulation, the outer surface of most wet suits is perfect for enhancing evaporative cooling and a windproof jacket should therefore be a requisite for divers from small boats in adverse weather when there will be no opportunity to dry off for some time after the dive, except by chilling evaporation.

Whatever the mechanism of heat loss, should its magnitude exceed heat production by the body cold stress results, with the core temperature beginning to drop once the body's defence mechanisms of vaso-constriction, peripheral cooling and increased muscle activity (including shivering) prove inadequate.

Hypothermia is usually defined as occurring when the core temperature has dropped below 35°C. It is cold exposure and cold stress not sufficient to reach this level however that causes the most common effects of cold on the diver.

Cold and the Diver

Cold exposure causes constriction of peripheral blood vessels and therefore reduction in skin and muscle blood flow. This causes expansion of the central blood volume which activates the familiar cold diuresis. Upon rewarming, the subject as a result is significantly dehydrated, a factor believed to both increase the risk of decompression sickness (DCS) and increase its severity should it occur.

When commercial divers first began using hot water suits, especially for surface decompression diving, a large increase in DCS incidence was seen. It was proposed that this was due to excess heating whilst on the bottom, resulting in vasodilatation and increased blood circulation and nitrogen uptake, with subsequent cold exposure during decompression, especially in a cold chamber, retarding off-gassing. Certainly reduced hot water temperature and heated chambers seem to have solved the problem. The same mechanism however may be present for wet suit divers who are initially warm enough for the first part of a dive, but chill progressively and are especially cold during the first half hour after the dive when wind exposed and still wet.

It is unfortunately not well recognised that perception of thermal comfort does not always correlate well with actual thermal status. Because skin temperature receptors are more sensitive to change in temperature than absolute temperature, and do not measure heat flux, it is possible to have a dropping core temperature without shivering, or an excessive sensation of cold. This can most readily occur when a slight increase in surface temperature gives a feeling of warmth and inhibits shivering whilst heat loss proceeds. The most familiar illustrations of this for many will be cold tap water feeling warm to the hands initially after they have been severely chilled, or cold seawater feeling warm after wet skin has been chilled by evaporation in the

wind. This mechanism has probably been one factor responsible for a number of deaths of rescued hypothermia victims who were wrapped in warm dry blankets that gave an initial sensation of comfort which stopped shivering without providing the thermal capacity or insulation needed to prevent continued rapid net heat loss in the face of reduced heat production by the body. Perhaps as a result of this problem divers can lose body heat significiantly at time without necessarily being aware of being cold

It is likely that many divers finish their dives, especially in winter and spring, with significantly reduced core temperatures, and certainly with a cold periphery and reduced body heat. In addition to increasing DCS risk, such cold stress has been shown to affect judgement, including the speech and appropriateness of emergency decision making and the actual judgement of time. Irrational behaviour is more likely. Limb cooling results in reduced muscle strength, speed and co-ordination, which is further severely affected if shivering occurs. Thus cold stress not only significantly increases the risk of accident or error in procedure, but makes the diver less effective in coping with incidents such as out of air, entanglement or a dislodged regulator etc. should they occur.

Cold Water Immersion

In addition to drwoning and hypothermia sudden exposure to very cold water without adequate wet or dry suit insulation carries an additional risk of rapid immersion shock related death.

The first, relatively rare mechanism for this is probably a combination of intense sympathetic and vagal stimulation that triggers a fatal cardiac arrhythmia. The victim, often young and apparently healthy, falls, or dives, into cold water and dies instantly.

The more common situation involves the victim drowning within minutes of hitting the water. Sudden cold water immersion causes an initial involuntary gasp reflex followed by a period of uncontrollable hyperventilation. There is a period of much rigidity and unco-ordination which resolves after a few minutes until actual muscle and nerve tissue cooling causes its return.* Unless the water is calm and a lifejacket is worn, it is thus easy for drowning to occur upon immersion and this mechanism probably accounts for most of the shipwreck and "man-overboard" deaths within seconds to minutes of hitting icy water that have often been wrongly ascribed to hypothermia.

* The Deputy Editor once saw theis happen to a plump RANR diver, wearing a wet suit, who went into 4° C water, on a 35° C day, below Eildon Weir wall. His comment on being hauled to the surface by his buddy line was "I'd have died if I hadn't had my regulator in. I couldn't stop breathing and I coul not move." The other five divers bobbed to the surface quite normally.

Although cooling rates vary widely, even a lean, unfit cold susceptible individual should survive at least 15 minutes in ice water before hypothermic cardiac arrest and better insulated idividuals should last an hour or more. In fact, the effects of dropping body temperature on muscle activity and conscious state usually results in drowning long before fatal cardiac cooling occurs.

Hypothermia

Classically, cooling has been shown as occurring progressively with specific signs and symptoms appearing at various temperatures as shown in Table 1.

TABLE 1

Core temperature (°C)	**Signs and symptoms**
37	"Normal" temperature (oral)
36	Shivering,
	Vasoconstriction
	Increased activity
35	Shivering maximal
	Slurred speech
	Slow
	Confused
	Dilated pupils
	Drowsy
	Low blood pressure
30	Shivering ceases
	Muscle rigidity
	Victim no longer able to self warm*
	Very slow pulse and respiration
	Unconscious
	Heart irregularities develop
25	**Death**

An example of the type of table often appearing in medical texts and first aid guides. The temperatures at which various signs and symptoms appear in fact varies widely with differing situations.

* This statement is not true (see page 35)

There have been a number of major sources of data concerning cooling, many of which have major flaws if used to try to describe accidental environmental cooling. Animal models, and studies on anaesthetised humans during cooling for neurosurgery or cardiac surgery, are affected by the altered physiology present. Most anaesthetic drugs affect some or all of the vascular, autonomic nervous system and central responses to cold as well as abolishing voluntary effort. Normal volunteer studies rarely cool the subjects below 35°C and thus study cold stress rather than significant hypothermia. Finally, the much quoted "experi-

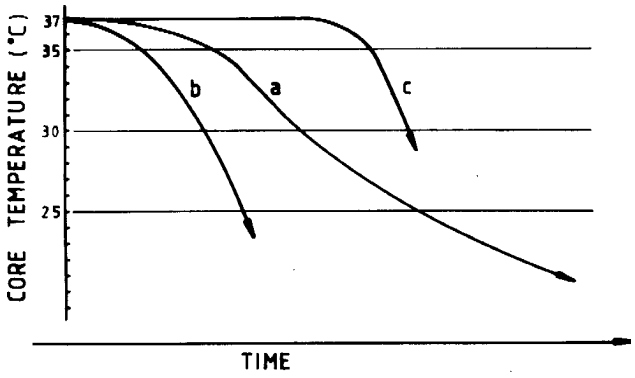

FIGURE 1.

Comparison of cooling patterns. The classical progressive cooling curve (a). Rapid cooling, e.g. a lean subject in ice water (b). The "exhaustion exposure" pattern where the temperature is initially maintained by exertion but rapid cooling ensues once the victim is exhausted and stops (c).

ments" carried out in the Second World War Nazi concentration camps, when victims were cooled to and sometimes re-warmed from extreme levels of hypothermia were carried out on people who were usually malnourished and emaciated.

Most immersion cooling experiments have been carried out with the subject at rest in a cold water bath with the head out. This is, of course, vastly different from swimming in turbulent water with a wet head exposed to spray and a cold wind. In a fully vasoconstricted adult, 40% or more of total body heat loss can occur through the scalp, face and neck. Exercise alters blood flow distribution, altering heat loss dynamics and increasing the rate of heat loss. In the case of exercise to exhaustion, in or out of water, increased heat loss may initially be balanced by increased heat production. When exercise slows or ceases, rapid net heat loss results as the glycogen depleted, exhausted body may not be capable of high involuntary heat production, and in some cases may not ever shiver. Death may follow rapidlyu and occur at a relatively higher temperature than expected from the cassical cooling curve. This situation is probably best illustrated by the mountain exposure cases who have struggled on to within sight of a refuge only to die shortly after stopping to rest.

In other cases, especially the rapidly cooled very fit subject, core temperatures as low as 24°C have been observed with the victim still conscious and shivering violently.

As survival chance seems more closely related to clinical status than actual core temperature, the Swiss Alpine Rescue approach for first aid situations has merit. Hypothermia is graded by signs and symptoms rather than by measured temperature, with higher conscious state and presence of shivering being favourable signs. This is not, however, to deny the usefulness of temperature measurement as a management aid for rescue or medical teams. For this purpose either an electronic temperature probe or at least a sub-normal thermometer is required, as normal clinical thermometers do not read below 35°C.

Predicted survival/time graphs are also very variable and unreliable because of the widely varying net cooling rates that result from body build, fitness, behaviour and all the other factors mentioned. Some individuals can be incapable of maintaining core temperature in tropical waters of 26°C or even 28°C whilst some cold resistant long distance swimmers and shipwreck survivors have coped with waters of 10°C for a prolonged period.

Being obese and passive seems beneficial, and whilst alcohol is certainly a risk factor for falling into the water in the first place, there is some evidence that inebriation may also be protective and a factor in survival in some circumstances.

Passive Rewarming

A fallacy often perpetuated about cooling is that "below a certain temperature, the victim is incapable of self-warming, and external heat must be provided". Whilst any animal is alive, heat is being generated by metabolic reactions. Human basal metabolic rate drops to half normal only at a core temperature of 28°C. The correct interpretation of observations made on rescued hypothermia victims should be that it may be extremely difficult to stop all heat loss, however, provided insulation is adequate (including reduction of evaporative skin and respiratory tract loss) anybody who is alive will slowly spontaneously rewarm. The thickness of "doona" or sleeping bag that is normally required for comfort in the cold should emphasize that the insulation necessary for field rewarming is considerable. More than one hiker's foam mat is probably necessary for insulation from the cold ground or boat deck underneath, with thick, windproof insulation around the victim.

If available insulation is inadequate, other bodies huddled together form a good substitute. The bare skin to skin contact commonly recommended however is often impractical and may not be as safe as huddling fully clothed or in separate sleeping bags. As previously mentioned, while skin warmth may feel good, any resultant reduction in shivering may in fact reduce net heat gain despite temporary improvement in comfort. Ideally, several companions should join the "huddle" as one additional person only provides a poor percentage of insulation cover for the victim.

As previously mentioned, insulation of the head and neck is most important, and any wetness of skin, clothing or blanket will cause considerable evaporative heat loss. If it is impractical to gently strip and dry off the victim without further exposure to cold or rough handling, the best way

to prevent evaporative loss is by completely enclosing the clothed victim in a plastic bag from the neck down. This also serves to keep the wet survivor from soaking dry insulation placed over this bag. If exposed to the weather, or a second plastic bag or sheet over the dry insulation will keep rain and wind off, thus "keeping the dry stuff dry" and maximising insulation, even if the victim remains wet and uncomfortable.

As vapour tight is the ideal for the inner layer, a bag is preferable to plastic sheeting, however even a set of waterproof clothing will be beneficial. The large garbage bags used for 240 litre wheeled bins are cheap and readily available, although a bit short for adults at about 150 cm long. Full size bags are available from many bushwalking suppliers. Aluminised plastic "space" blankets are probably over-rated. Although radiant heat loss may be significant in a warm skinned newborn infant, the skin of a hypothermic victim is usually close to environmental temperature, and net radiant heat loss is thus not a major factor. Although not usually in bag form, "space blankets" are nevertheless very compact large plastic sheets. The type of plastic used however presents problems in many rescue environments. Being an ultra high density film, it is very strong for its thickness when only blunt forces are applied. Any puncture or tear, however, rapidly propagates and, especially in the wind, these "splitting" sheets can be rapidly shredded to ribbons. As a final disappointment, Royal Air Force tests failed to show improved radar response if liferafts were draped with the reflective film.

Airway "Warming"

The other source of heat loss is from the respiratory tract, with a larger proportion of the heat being lost by evaporation than from breathing cool air in and warm air out. In cold dry air, 25 or 30 watts (approximately 100 kJoules/hour) will be lost this way (or more if exercising or shivering violently). Any reduction in this is useful. Merely wrapping clothing around the head will help somewhat,whilst the moisture exchange humidifiers sometimes used in anaesthesia (e.g. "Thermovents") are quite effective. Best of all is heated, humidified air or oxygen, which can provide 100% "airway heat loss prevention". There is little additional benefit from fully humidified gas heated much higher than 40°C, as evaporation is the main heat loss source prevented, and indeed attempts at further "core rewarming" by using higher temperatures may risk airway burns and cardiac instability. In many instances, use of airway heat and humidity will not increase the usual spontaneous rewarming rate of 0.5°C - 1.0°C per hour, but it tends to reduce shivering and thus metabolic requirements and cardiac workload without reducing core temperature rise. Being non invasive and without significant side effects, it is an excellent technique to use.

Warming

Minimisation of all further heat loss, thus allowing slow, passive warming of hypothermic survivors as described is the safest method of rewarming for all first aid situations and probably also for all but experienced, intensive care equipped medical facilities. Medical practitioners often feel uncomfortable with this as there is an interventionist urge to "do something" to correct the "abnormal test results", in this case core temperature reading. Hypothermia alone however, although a potentially dangerous condition, is in many ways protective rather than damaging, as is demonstrated by the deliberate cooling to as low as 10°C, of some patients requiring induced cardiac arrest for surgery with subsequent full recovery. In the case of elderly, slowly cooled urban hypothermia victims, rapid uncontrolled rewarming is definitely dangerous. Even 0.5°C per hour may be too fast to allow metabolic derangements to correct themselves. Young, healthy, rapidly cooled victims however tend to do well whatever methods are used, leading to case reports of success using many different methods. An examination of the likely causes of post rescue death, which is not uncommon, however, demonstrates some of the potential pitfalls.

Post-Rescue Death

Most severely hypothermic victims will be dehydrated, with slowed respiration and pulse and a low blood pressure. This reduced circulation is adequate for survival given reduced metabolic demands and a vascular system that is either lying horizontally or supported by immersion in water, thus allowing continued cardiac and cerebral perfusion at low pressures. The hypothermic heart has a lowered threshold for fibrillation and may fibrillate spontaneously if venous return drops, or if there are other stimuli for the heart rate to increase.

Conscious survivors who die during winching up to a helicopter by an underarm strap (as has occurred in Bass Strait, the Fastnet race disaster and other cases) probably suffer loss of hydrostatic support, resulting in their reduced blood volume pooling in the legs. This causes sudden reduction in venous return to the heart, which, combined with anxiety chest squeeze and skin chilling from evaporation in the helicopter rotor wash, all combine to induce ventricular fibrillation in the sensitized heart. As it is often impossible to defibrillate a cardiac arrest victim with a core temperature below 30°C, death has resulted in many of these cases.

It follows that any victim suspected of being hypothermic should be kept horizontal, or legs up during rescue and rewarming. This requires a stretcher, or if this is impractical, a double strop (one under the knees, one under the arms) for helicopter, crane or winch recovery.

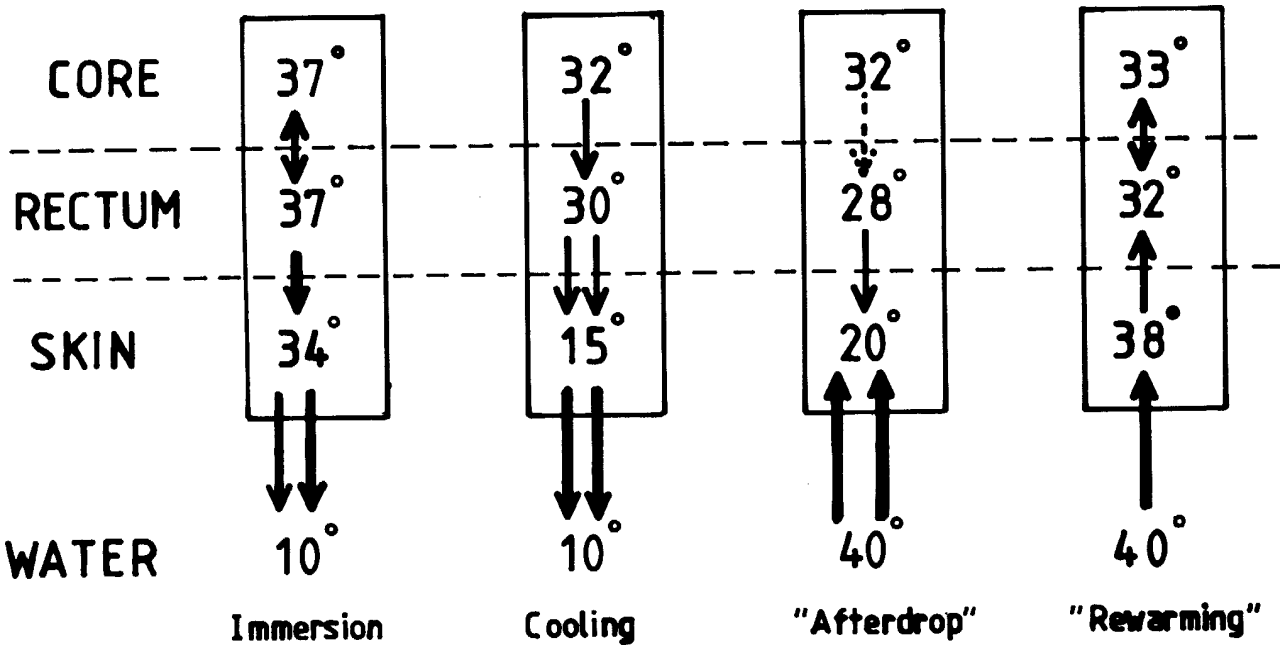

FIGURE 2.
Illustrative model of immersion hypothermia and rewarming demonstrating rectal temperature "afterdrop" without significant central core "afterdrop". Arrows show net heat flux.

Early rewarming death probably results from a related mechanism. As warming proceeds, surface and peripheral blood vessels dilate, reducing blood flow resistance and requiring increased cardiac output to maintain blood pressure, especially given a low blood volume. The cold heart again may not be capable of the necessary increased rate and may fibrillate. This is most likely to occur during rapid external warming, such as can result from using radiators, fire, hot water bottles or blankets or hot water immersion. Appropriate intravenous fluid infusion (which should be warmed if possible) combined with appropriate warming should prevent such deaths, and close monitoring of pulse, blood pressure and central venous pressure is recommended.

"Afterdrop"

Especially when "core" temperature is measured rectally, it is common for the measured temperature to continue to fall for the first 10 to 20 minutes after warming commences. As this time coincides with the pulse rise and blood pressure drop mentioned above, and the associated risk of early rewarming death, this temperature "afterdrop" has been blamed for rewarming complications. It has been assumed that as circulation increases, cold, acidotic blood starts to flow from the legs and arms, resulting in central blood temperature drop and cardiac instability. This is the reasoning behind leaving the arms and legs out during water bath rewarming. "Afterdrop" however is largely a rectal temperature phenomenon. When cardiac temperature is measured with a Swann-Ganz probe or an oesophageal probe, afterdrop is either much less than measured rectally or absent, and Golden has shown that "afterdrop" can be measured at a site 10-15 cm in from the surface in both water filled copper cylinders and pig cadavers which are cooled and then rewarmed. This phenomenon can be explained by passive thermodynamics and sensor location, and it seems likely that human afterdrop is similar.

See figure 2.

Severely vasoconstricted limbs contain little blood anyway, so where rapid immersion rewarming is appropriate then the limbs might as well be immersed so that they are both hydrostatically supported, and acting as additional "heat exchangers" to aid the rewarming.

Another proposed theory for cardiac irritability suggests that if the blood entering the heart is colder than the heart, the cardiac conducting system is selectively cooled and electrical impulses are more likely to travel via the muscle tissue, predisposing to arrhythmias and fibrillation. Significant temperature gradients are more likely to occur with rapid cooling and are presumably less likely if rewarming is slow and even, or "central" in nature (e.g. spontaneous, airway heating, peritoneal lavage or cardiac bypass techniques).

The other major groups of post rescue complications and deaths occur much later, and most frequently affect the elderly or the victim with concurrent disease that in some cases may have contributed to the exposure situation. These

may include drug overdose, diabetes, malnutrition, trauma, or infection. Especially when the hypothermia has been of slow onset or long duration, excess fluid becomes trapped in body tissues ("cold oedema") and thus on rewarming the blood volume that was initially reduced by dehydration may become fluid overloaded, resulting cardiac, pulmonary or cerebral complications. Post rewarming infection is also a major problem that frequently requires treatment.

Rewarming Recommendations

The optimum method of rewarming remains a subject of much debate. Appropriate decisions however become much easier if specific situations are considered rather than just the label "hypothermia". Experienced intensive care units capable of a high level of monitoring and physiological control have reported good results with many different methods and rates of rewarming.

Fairly rapidly cooled (e.g. by cold water immersion) victims, especially if young and otherwise healthy, and especially if still conscious and shivering when found, tend to tolerate rapid hot water bath (35-38°C) rewarming well. Certainly if an immersion victim is only cold stressed (T_{core}>35°C) or perhaps mildly hypothermic but fully conscious, the comfort and speed of warm water rewarming will be much appreciated.

In other cases, or in the field however, the aim is prevention of all heat loss, thus allowing "passive" rewarming as the method of choice. This method can be continued once medical care is obtained, with the addition of continuous blood pressure, pulse, CVP and temperature monitoring, IV fluids and biochemical monitoring. (The current consensus of opinion is that blood gas results should not be adjusted for temperature before interpretation and intervention).

If the patient is rewarming and complications arise, it may be necessary to reduce insulation and slow the rewarming, especially in the chronic hypothermia case. In some cases, 0.5°C or even 0.25°C per hour may be the maximum tolerable. It will thus take many hours before normothermia is attained.

If the victim fails to rewarm, additional insulation and/or active heating must be used. The main indication for active, rapid warming are when hypothermia complicates management of other illness (e.g. trauma requiring surgery, diabetic or other medical emergency) or when there has been submersion, and near drowning or when the victim presents in cardiac arrest or arrests during treatment.

If active, rapid warming is required, an increasing number of successful case reports support partial (femorofemoral) cardiac by-pass as the method of choice. This method provides assistance to the circulation, control of fluid balance, electrolytes and if necessary oxygenation as well as very high possible warming rates if the necessary equipment and skills are available (open heart surgery units). In other centres peritoneal dialysis is a useful addition to heated humid oxygen, insulation and heating mattresses, and also helps control electrolyte and fluid imbalance.

Other techniques reported have major drawbacks. Neither gastric nor rectal nor bladder irrigation exchange heat as well as peritoneal lavage and rather than help control fluid and electrolyte problems, gastric lavage in particular can cause water intoxication. Mediastinal lavage requires thoracotomy, which allows direct cardiac massage, but carries a high complication rate by comparison with other techniques. Short wave diathermy is very difficult to control and thus risks hypothermic tissue "cooking", and hot external packs must be likewise very carefully controlled to prevent excessive heat or pressure resulting in tissue necrosis. Water bottles, etc. at temperatures of 45°C or more have caused extensive tissue loss in some cases due to the very sensitive nature of cold skin, and other tissues, and their low blood flow. Finally the waterbath can be effective, but makes "tube management" and monitoring difficult, and in the cardiac arrest case, cardiopulmonary resuscitation (CPR) becomes athletic, difficult and somewhat chaotic and defibrillation is impossible in the bath and highly dangerous in the water soaked immediate environs.

Severe Hypothermia

Cases where a hypothermia victim is apparently dead when discovered are of significant concern, as inappropriate decisions and care can result in the death of an often young, healthy person who might have fully recovered. The following have been documented as not necessarily incompatible with survival in the severely hypothermic: apparently absent pulse and respiration, fixed dilated pupils, "rigor mortis" like rigidity and absence of response to pain, etc., minor skin decomposition, dependant lividity, glazed eye surface, sea foam in mouth or prolonged submersion. At the Mountain Medicine Symposium organised by the Institute of Ambulance Officers (TasmaniA) in 1987 Captain M.J.Nemiroff, a doctor in the U.S.Coastguard, presented a personal series of over fifty cases of survival often prolonged submersion in cold water. He now recommends resuscitation and rewarming of any victim of up to 60 minutes of submersion in water of 20°C or lower. Forty five of his fifty cases had suffered no significant clinical neurological impairment despite documented submersions ranging from six to sixty minutes and although most victims were very young, this was not always the case. Cases like these, and cases of prolonged survival after up to four hours of CPR and documented cardiac arrest after avalanche burial reinforce the tolerance to hypoxia that hypothermia may confer in some cases.

Hypothermia and CPR

The question of whether CPR should be performed in the apparently lifeless, cold individual is often asked. There is little doubt that CPR is counterproductive and dangerous if the victim has a spontaneous heartbeat. If a cold victim is discovered with no obvious, gross trauma or putrefaction, a good period of time should be spent looking for signs of life. Any breathing can be presumed to indicate heartbeat, even if nil is apparent. A full two minutes should be spent looking and feeling as rates may be very slow. Any movement is a sign of life, and the eyelash and corneal reflexes are reported not to be lost until the core temperature drops below 24°C or 25°C. The Antarctic Division recommends using an opthalmoscope or torch held close to one's line of sight to look for a "red-reflex" through the victim's pupils. Bright red at the back of the eye presumably indicates red, oxygenated blood and therefore life. Finally, if available, an electrocardiograph (ECG) can also be used to look for an organised cardiac rhythm, or a doppler stethoscope to listen for blood flow.

If life is present, field treatment should include very gentle handling and maximal insulation with either transport to a medical facility or waiting for arrival of on-site care whichever is most appropriate to the situation.

If there has been witnessed submersion with cardiac arrest when rescued, or if a rescued survivor suffers collapse and cardiac arrest during rescue or rewarming, there is little controversy about the recommendation to perform CPR.

Normal rates are recommended, as there is little sense in making a low efficiency techniques less efficient by using reduced rates of compression and ventilation. Recovery after prolonged CPR is perhaps explained not only by metabolic tolerance created by hypothermia, but also by rigidity of the cold heart making it a more efficient conduit for a circulation created by compressing the lung vasculature (now believed to be a major mechanism of CPR action). The cardiac arrest victim should be aggressively rewarmed, as defibrillation is often not successful until the core temperature exceeds 30°C, and resuscitation should not cease until the temperature exceeds 32°C. Although an initial defibrillation attempt may be made, repeated defibrillation or drug therapy is probably not indicated below T_{core} 30°C. Drug doses which have not been effective due to peripheral pooling may be mobilised and become active and toxic as the victim is rewarmed. Many drugs have reduced effects when the subject is cold, and most antiarrhythmics are ineffective. It is suggested that Bretylium however may retain its action, and that it should be tried early in these cases. Spontaneous cardioversion may occur and seem more common with "core rewarming" techniques.

Victims who are apparently dead upon discovery, but who might perhaps have a chance of resucitation, form the remaining group, and here a certain degree of pragmatism is appropriate. It is important not to risk the lives of rescuers or definite survivors. If feasible and safe, CPR can be commenced if it is believed that it can be continued until the victim is rewarmed, and that good medical facilities can be reached within, say, four hours. If there is any doubt, however, the victim's best chance probably lies in the existence of undetectable life rather than resuscitatable death of unknown duration and maximal insulation and observation at least gives some chance rather than immediate, on site declaration of death. Although death is classically said to occur from cardiac arrest (usually ventricular fibrillation) as the core temperature drops below 25°C, survivals have occurred following core temperatures as low as 15°C in exposure victims, and in controlled operating theatre induced hypothermia, humans have been cooled to below 10°C and subsequently rewarmed.

On a number of occasions, urban hypothermia victims have been erroneously delivered to the morgue, only to exhibit signs of life the following morning after a night under a cover in the comparative warmth of a non-refrigerated morgue holding room. These and other remarkable survivals from differing situations where hypothermia has been a factor reinforces the dictum that no case should be considered hopeless until the victim is "warm and dead".

Further reading

For those further interested, the following two recent and well referenced works are recommended:

The Nature and Treatment of Hypothermia
Eds. Pozos and Wittmers
University of Minnesota Press, 1983

Hypothermia and Cold Stress
E L Lloyd
Croom Helm, 1986

This paper has been adapted from a lecture given at the joint SPUMS and Royal Hobart Hospital meeting in Hobart in November 1988

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SPUMS ANNUAL SCIENTIFIC MEETING 1989

CEREBRAL ARTERIAL GAS EMBOLISM

Col (Dr) Jimmy How Yew Chen

Introduction

Pulmonary barotrauma arises from excessive changes in pressure affecting the lungs. This is widely known among the diving fraternity as "burst lungs". It is a clinical manifestation of Boyle's Law acting upon the effects of a burst lung. The clinical effects of pulmonary barotrauma such as pulmonary tissue damage, pneumothorax, surgical emphysema and cerebral arterial gas embolism (CAGE) are often serious and potentially life-threatening. Among recreational divers, CAGE is the commonest cause of death, which is often attributed to drowning. Leitch et al[1] reported an incidence of CAGE of 7 per 100,000 dives in a group of military divers. In the same study, CAGE was responsible for 31% of the diving accidents. Although with proper training and equipment the incidence is small, there is still a real risk in cases of panic surfacing, chronic smokers and those with a history of lung disease and previous pneumothorax. The various forms of pulmonary barotrauma are shown in Figure 1.

Pathophysiology

Cerebral arterial gas embolism is the most serious form of pulmonary barotrauma and early recognition and treatment is urgent. It results when air emboli are introduced directly into the blood circulation. CAGE can result in an acute anoxic episode with diffuse generalised ischaemia culminating inevitably in death if untreated.

The distribution of gas introduced into the arterial circulation is dictated by the posture of the diver and often exacerbated by the profuse liberation of air bubbles into the circulation. The air emboli distribution is dependant on the local perfusion pressure which is an interaction of the mean arterial blood pressure (MABP), cerebrovascular resistance and intracranial pressure. The interaction is made more complex by the variation of cerebrovascular resistance with mean arterial blood pressure, such that cerebral blood flow remains constant over a range of arterial pressures.[2]

Air embolus entrapment in a cerebral arteriole will occur when the forces opposing the embolus movement exceed the local perfusion pressure. These forces are related to the embolus size. The larger the embolus, the more likely is entrapment. For a given vessel this relates to embolus length, which is directly related to embolus volume and inversely related to the square of the diameter of the vessel it is occupying. Gorman[2] found that the cerebrovascular reaction to gas embolism differed from that to a solid embolus. In air emboli, localised vasodilation was seen while with solid emboli vasoconstriction was seen. In his animal experimental studies, cerebral arterial air embolism caused an increase of 42% + 28.13 SD in the cerebral arteriole diameters.

In gas embolisation a significant proportion of gas entering the cerebral circulation will pass through without causing vessel occlusion. In Gorman's study[2] spontaneous redistribution occurred in 5 out 30 animals (17%).

Entrapment of air emboli occurred in arterioles of 50-200 micrometers in diameter. The most frequently involved cerebral arterioles are those having diameters of about 150 micrometers. If an air embolus that had a length exceeding 500 micrometers entered an arteriole of this size entrapment with local circulatory arrest was inevitable. Conversely, if the length was less than 500 micrometers entrapment never occurred, with the embolus progressing without interruption.

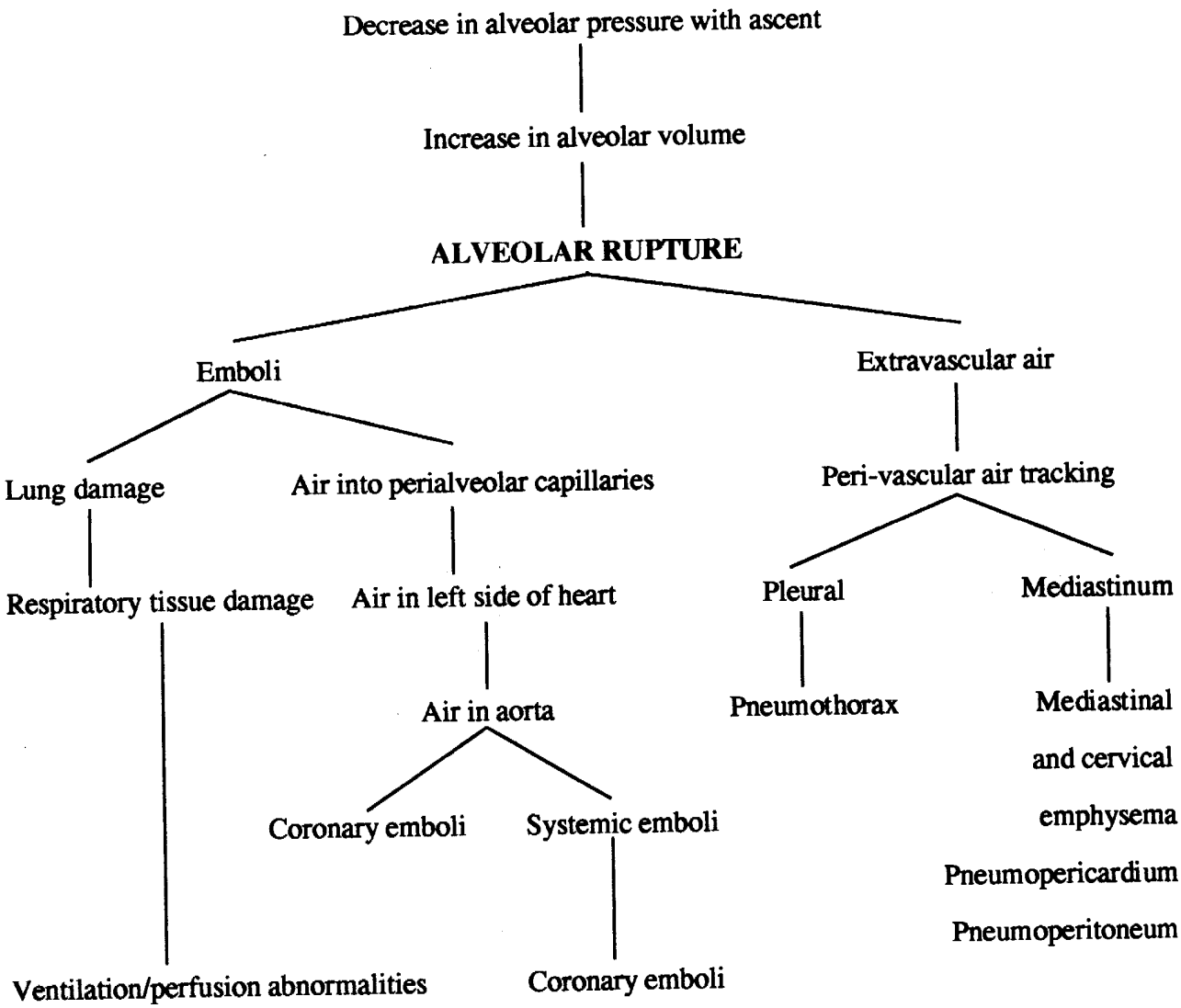
Pathological sequence of air embolism

Once CAGE causes vascular occlusion, a number of pathological processes are initiated. The following events take place:[2]

(a) Permeability of the blood-brain barrier increases.
(b) Ischaemia of tissues.
(c) Post-obstructive vessel coagulopathy.
(d) Microfoci of haemorrhage.
(e) Cerebrovascular autoregulation is lost.
(f) Changes in metabolism.

An increase in the blood brain barrier permeability results in increased brain water content, i.e. cerebral oedema. Tissue ischaemia results in infarction. The cortical gray-white junction is particularly vulnerable to ischaemia from air embolism due to its angio-architecture which causes preferential lodging of emboli in this border zone. The gray-white junction occupies a watershed between the penetrating arteries of the cortex and the deep penetrators nourishing the white matter.[3,4] Ischaemic hypoxia that is neither sufficiently prolonged nor profound enough to cause a classic watershed infarction can give rise to necrosis in the deep cortical areas alone. The passage of air emboli through vessels is associated with arterial thrombosis. This is thought to be mediated via platelets rich in serotonin and adenosine diphosphate (ADP) and aggravated by agents such as collagen, thrombin, adrenaline and noraderanaline. Gas bubbles in themselves function as platelet agonists. Scanning electron microscopy has shown interaction between air microbubbles and platelets. It shows adhesion of platelets

FIGURE 1



to the bubble surface with formation of aggregates.[5] Microbubbles act as a foreign surface and a resultant platelet clumping causes a fall in platelet concentration with formation of platelet micro-thrombi. Endothelial oedema and formation of platelet thrombi cause progressive arteriolar occlusion, small focal haemorrhages and cellular metabolic disruption. The process is further aggravated by haemoconcentration secondary to increased vascular permeability.

Case presentation

Numerous studies have shown the beneficial effects of hyperbaric oxygen in the management of ischaemic disorders of the brain and in cerebral oedema.[6] The following is a clinical case treated at the Naval Medicine and Research Centre, Singapore (NMRC). I would like to discuss this case to establish a clinical approach in the management of CAGE that results from pulmonary barotrauma.

Clinical History

HC was a 42 year old Chinese male, seen at NMRC in September 1984. Prior to the incident he was a sports diver for one and a half years with no previous professional training in diving. He used to dive about once a month for a few days at a time. He had a history of viral pneumonia in December 1983 which resolved with treatment but no other past history of note.

On 2 September 1984 at 1300 hours after diving for about half an hour to a depth of 30 metres off the coast at Mersing, he was seen to suddenly surface. He was apparently able to inflate his life jacket. However, when the safety boat (sited 60 metres away) reached him, he was found to be unconscious with blood stained froth discharging from his mouth. His scuba tank was found to be emptied of air. There was no spontaneous pulse and respiration. CPR was started. After about 12 minutes he regained spontaneous pulse and respiration but remained unconscious. He was

placed on his side and evacuated to a hospital in Mersing (6 hours away). At Mersing Hospital, he was noted to be in coma. Pulse was 102/min, BP 120/70, respiration 28/min. His pupils were sluggish reacting to light. He responded to pain. He was intubated and given I/V dextrose, ampicillin, decadron and frusemide. I/M anti tetanus toxoid was also administered.

He was evacuated to Singapore by helicopter. During the flight he had 2 fits. He arrived at the Department of Neurosurgery, Tan Tock Seng Hospital, on 3 September 1984 at 1200 hours. He was assessed to be in Coma III. His pupils were noted to be unequal. The left pupil was 5 mm and the right pupil 4.5 mm. He showed a decerebrate response to pain. He had a febrile episode which was investigated. A provisional diagnosis of diffuse anoxic brain damage secondary to a diving accident was made by the neurosurgical team.

Investigation

Table 1 shows the investigations done during his stay in hospital.

On admission his urea was mildly elevated at 79 m eq/l. This was probably due to dehydration as he had a raised haematocrit. Septic workout in the form of lumbar puncture, urinanalysis, chest X-ray and sputum analysis were done for his fever and raised total white count. Klebsiella was isolated from sputum culture and his endotracheal tube and he was treated with the appropriate antibiotics. His fever subsequently resolved and total white count returned to normal. Chest X-ray was reported to have evidence of infective changes.

The blood gas picture showed a compensated metabolic acidosis with a low PCO_2, low HCO_3 and a negative base excess. The anion-gap on 3 September 1984 was 10 m eg/l and in the presence of normal K^+ levels was consistent with early renal failure. This was probably of pre-renal origin and hydration helped reverse the picture. His blood coagulation profile was normal and platelets were within normal range without evidence of consumptive coagulopathy. CT scans of the head done on admission and 9 days later were reported as normal. A repeat scan one month later showed features consistent with resolving cerebral oedema or cerebral atrophy.

Hyperbaric Treatment

He was referred to the Naval Medicine and Research Centre on 4 September 1984 at 2300 hours about 58 hours after the diving accident. A clinical diagnosis of pulmonary barotrauma with air embolism was made based on the history.

Hyperbaric treatment was instituted with oxygen using an extended Table 6A for a duration of 7 hours. No definite changes were noted in his coma state immediately after treatment. On the third day following treatment, he was noted to be in a “lighter state” by ward staff and the neurologist. Four days later he was able to withdraw from pain. Seven days after the treatment he was able to open to his eyes. In the following days to weeks, his condition gradually improved. Auditory evoked potentials done on 11 September 1984 showed signs of brainstem demyelination. This test is useful in the diagnosis of small vessel lesion, pontine myelinoses and hypoxic brain damage.

TABLE 1

INVESTIGATIONS

Full Blood Count

Date	**3/9**	**5/9**	**7/9**	**8/9**	**13/9**	**18/9**	**28/9**
WBC	25.7	15.8	19.7	16.9	18.4	15.1	10.1
RBC	5.41	5.41	4.52	4.65	3.90	3.63	3.52
Hb	16.2	16.3	13.7	13.7	11.8	11.0	10.9
Hct	51.6	52	43.3	43	37.9	34.2	34.0
MCV	93	94	93	90	94	92	94
P	98	86	96	83	98	91	86
L	2	12	4	10	2	8	12
M	-	2	-	7	-	1	2
E	-	-	-	-	-	-	-
Platelet	-	-	-	250	-	310	-

TABLE 1 (CONTINUED)

Urea and Electrolytes

Date	**3/9**	**5/9**
Urea	79	48
Nat	149	145
K+	4.7	4.3
Cl-	119	110
NCO_3	-	28.1
G	2.8	1.3
G/U	170	120

Blood Gases

Date	**4/9**	**5/9**	**6/9**
O_2%	Air	40%	40%
pH	7.39	7.375	7.457
PCO_2	33.6	29.7	33.0
PO_2	77.9	135.6	77.0
HCO_3	20.0	17.0	23.1
TCO_2	21.0	17.9	24.1
Base excess	-3.8	-6.5	-2
SBE	-4.1	7.1	-0.4
O_2 saturation %	94.1	98.2	94.9
SBC	21.1	19.1	24.5

PT:	14 seconds	Control	13 seconds
PTT:	30 seconds	Control	38 seconds or less

Urine Microscopy (3/9/84)

Packed with RBC AP +++ Granular casts +

Lumbar Puncture (4/9/84)

CSF appearance	clear
Cell count	11
Sugar	129
Total protein	less than 10
Globulin	negative

No torula or AFB organism

Liver Function Tests (16/2/85)

TP	6.5 g/ds
Alb	3.9 g/ds
Bil	0.3 mg/dl
Alk PO4 alt	69 u/l
ALT	28 u/l

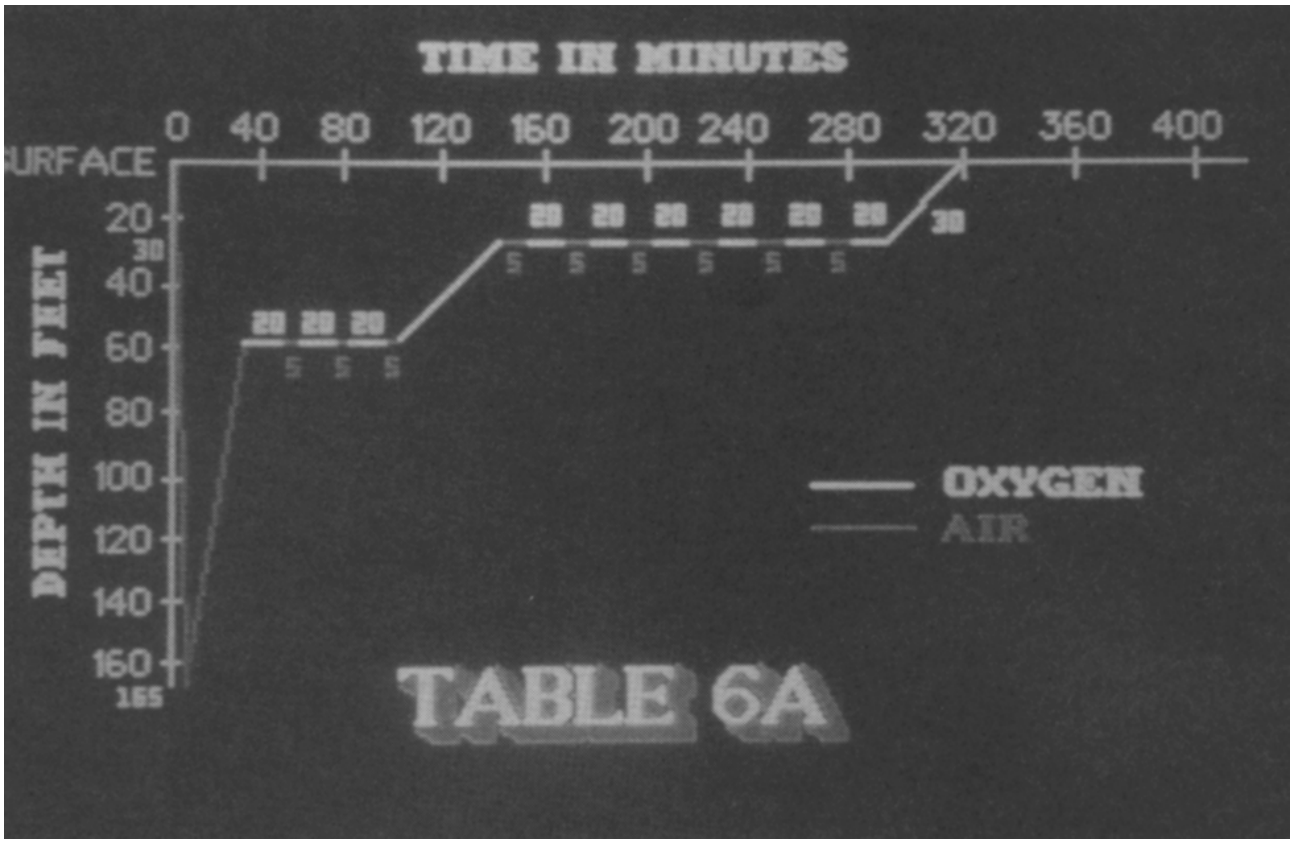
Auditory evoked potential (11/9/84)
Showed brainstem demyelination.

Culture and sensitivity tests

Blood	5/9/84	Negative
CSF	4/9/85	Negative
Sputum	5/9/85	grew Klebsiella
ETT Swab	5/9/85	grew Klebsiella
Blood	7/9/84	Negative
Urine	8/9/84	grew Klebsiella
Blood	22/9/84	Negative

Radiology

Chest X-ray	3/9/84	Ward film taken. Bilateral infective changes.
CT Scan	3/9/84	NAD
	12/9/84	Normal
	8/10/84	Compared to that of 3/9/84 the scan shows that the lateral ventricles and cerebral sulci are slightly larger, consistent with resolving cerebral edema or cerebral atrophy.



Progress

By early October he was able to open his eyes spontaneously and blink at a visual threat. Pupils were equal and reactive, corneal and gag reflexes were present and Doll's eye manoeuvre positive. By early December he had regained control of bladder and bowel function and was able to respond to verbal commands. There was generalised hypertonus and reflexes were brisk but power had improved. Speech therapy was started soon after in late December and over the next few months with a regular programme at the rehabilitation centre he improved considerably.

Today, though confined to a wheelchair, he is able to carry out most of his daily activities such as toilet, dressing, eating and movement. His speech is mildly impaired and he has some loss of recent memory but apart from this, he is able to function well. His last tested IQ was 98 which falls within average intelligence category.

Disscussion

The commonest presenting symptoms and signs of air embolism arising from pulmonary barotrauma are the loss of consciousness, sensation and the loss of motor power depending on the area of brain involvement. A study of 117 cases of CAGE in divers done by Leitch[1] is shown in Table 2.

TABLE 2

PRESENTING SIGNS AND SYMPTOMS OF CAGE

(prepared from information in Leitch and green.[1])

Unconsciousness		38.5%
Power Loss		
(a)	Generalised	6.8%
(b)	Legs	16.2%
(c)	Unilateral	26.5%
Sensory changes		
(a)	Unilateral	19.7%
(b)	Bilateral	22.2%
Loss of co-ordination		11.0%
Visual changes		21.4%
Confusion/disorientation		36.7%
Speech problems		11.1%
Convulsions		
(a)	Generalised	8.5%
(b)	Focal	2.6%

Other symptoms included somatosensory changes, personality changes, retrograde amnesia, vertigo and deafness.

In the case of HC, unconsciousnss and convulsions were noted as presenting symptoms.

The clinical presentation was that of anoxic encephalopathy secondary to cerebral air embolsim. Anoxic encephalopathy is commonaly seen in other conditions such as:

(a) Suffocation
(b) Carbon monoxide poisoning
(c) Neuromuscular disease
(d) Myocardial Infarction, with secondary circulatory collapse.

The severity of the clinical presentation depends on the degree of anoxia.[7] In mild anoxia consciousness returns after a short period of coma and full recovery is usual.

In moderately severe anoxia the patients are profoundly comatose with decerebrate postures. In the first 24-48 hours, death may terminate this state in a setting of rising temperatures, deepening coma and circulatory collapse. Such individuals usually survive, if at all, in a state of irreversible coma or persistent vegetative state. If coma lasts for more than 5 days the patients almost never recover.

In severe anoxia complete unawareness and unresponsiveness is noted with the abolition of brainstem reflexes. Natural respiration cannot be sustained. Only cardiac action and blood pressure are maintained. EEG shows no electrical activity (i.e. isoelectric). This is the brain death syndrome.

HC fell into the second group of moderately severe anoxia based on his clinical presentation and the natural course of events should have been progression to death or at best a persistent vegetative state.

Hyperbaric oxygen was used to combat his ischaemic cerebrovascular condition and his traumatic brain oedema. The use of hyperbaric (HBO) produces more dissolved oxygen in plasma resulting in improvement of hypoxia in the ischaemic tissue. It is also known that progression of brain oedema can be suppressed by cerebral vasoconstriction due to high PO_2 in combination with the increased availability of oxygen.

In animal experiemental studies the use of HBO therapy up to 3 hours after artificially induced ischaemia, via bilateral carotid artery ligation, significantly prolonged survival time compared with that in non-treated ischaemic animals.[3] The mechanism of this has been attributed to oxygenation of ischaemic tissue, suppression of brain oedema and reduction in red cell agglutination. In addition, ATP in the treated group tended to be higher than in the non-treated group. The metabolic data suggest that prolonged survival in these treated animals is due to suppression of further increase in lactate.

In the management of pulmonary barotrauma with air embolism, it has been recognised that the time from onset of symptoms to adequate recompression therapy is the main determinant of success in treatment. Any delay in therapy allows progress of the blood-bubble interaction which leads to intravascular coagulation, capillary leakiness, oedema, haemoconcentration and infarction.

In a review of 117 cases of CAGE Leitch[1] noted 21% of individuals recovered completely without treatment. In those treated, the relationship between delay and response to treatment is shown in Table 3.

Cure was defined as apparently complete recovery without signs or symptoms at any time following treatment. In our case HC was started on treatment 58 hours after the diving accident. From the study above it was noted that likelihood of cure was reduced as the delay to treatment increased. The cut-off time for favourable outcome was about 4 hours. The likelihood of achieving a cure was reduced to less than 50% when the delay exceeded 4 hours.

While commercial or naval diving operations usually have access to on-the-scene recompression chambers, the growth

TABLE 3

RESPONSE TO TREATMENT IN CAGE CASES

Delay to treatment	Cases cured	Cases not cured	Total cases treated
< 1 hour	16	8	24
1 - 2 hours	13	2	15
2 - 3 hours	12	4	16
3 - 4 hours	7	4	11
4 - 5 hours	3	4	7
> 5 hours	7	9	16
Totals	**58**	**31**	**89**

of sports diving in remote areas presents a major area of concern.

In such settings, the initial transportation of the casualty in a 30° head-low tilt and administration of 100% oxygen continuously until arrival at definitive chamber management will be beneficial. Adequate fluid administration begun early and maintained during transportation to the chamber would help towards eventual successful recompression therapy. Steroids may also be useful in the treatment of cerebral oedema.

At the time of arrival at the chamber, a pre-treatment examination is done to gauge the seriousness of the patient's condition. Clinical examination, including a neurological assessment, is mandatory. In the absence of chest X-ray the physician should at least clinically exclude a pneumothorax. If present, a chest tube with Heimlich valve or underwater seal is required prior to any recompression. Conscious patients can clear their own ears but in the comatose or unconscious patients bilateral myringomtomies may be performed.

Royal Navy Table 63 or US Navy Table 6A may be used for recompression therapy. This is the generally accepted treatment for CAGE. The suspected cases are taken directly to 50 m (6 ATA) and treated according to the requirements of the tables.

Conclusion

The clinical case of HC presents itself as a unique example of how much work there still needs to be done in the field of CAGE. Although the neurological team felt HC was a hopeless case destined either for death or a persistent vegetative state, hyperbaric treatment was nevertheless instituted.

The hyperbaric treatment challenged conventionally accepted criteria for delayed management in that it was started 58 hours after the initial hypoxic event. Regardless of this, HC did over a period of weeks to months show considerable improvement in his clinical state. Whether this would have been the natural outcome had HBO not been used is open to question.

Far from being an area of consensus and understanding, CAGE is an area for further study and active debate. Our task of understanding relates not only to the underlying pathological process but also to the ideal treatment of CAGE.

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MASSIVE HAEMOPTYSIS AND AGE A CASE REPORT

A Santos

Introduction

We have the opportunity to review the theory of Caisson disease (decompression sickness or DCS) and diving related maladies. These are the particular risks for us as we attempt to transcend what is described as the teleological barrier between land and sea. I would like to present a case for demonstration, particularly the clinical aspects of diving medicine and its varied presentation. In America we try to differentiate the various entities in diving medicine into two major groups, DCS and pulmonary barotrauma. In differentiating these groups we usually label joint pain and spinal cord symptoms, with loss of motor and sensory

function below the umbilicus, as DCS. We divide pulmonary barotrauma into two groups, arterial gas embolism (AGE) and air in spaces in the body where it is not supposed to be, skin, lungs, hemithorax, mediastrinum, and even the peritoneum. Going a bit further we can say that AGE is primarily a disease of ascent and DCS is a disease of exposure over a period of time. In addition there is otic barotrauma, a disease of descent, which is commoner than anything else.

We have a difficult time in diving medicine as it is hard to differentiate between those types of diving maladies. Any one patient may present with both kinds of problem at any one time. Haemoptysis is, for us, an unusual event in AGE, massive haemoptysis is even more rare. So I am presenting a case report of a patient who had massive haemoptysis.

Case history

A 40 year old male made his first ever scuba dive, in Mexico, to 4.5 m (15 feet) for approximately 5 minutes. Then he panicked. Later he said that he had had difficulty with the regulator and could not breathe as well as he thought he should. He removed his weight belt and made a breath-hold ascent to the surface. On the surface it was noticed that he was breathing freely but had a depressed level of consciousness. He began to expectorate large quantities of blood. He was taken to a nearby hospital and given intravenous fluids and his vital signs were monitored. He was noted to have a respiratory rate of approximately 30-40 breaths/minute and his pulse rate was 120-130/minute. He was transferred to a second hospital where he promptly went into cardiopulmonary arrest. He was resuscitated , given 4 ampoules of bicarbonate, was intubated and successfully recovered after 3 minutes. His deep tendon reflexes at that time were intact and he was felt to be stable. By this time he had brought up about 800 ml of blood, which was actually measured and sent to the University of California from where transport was arranged for him by the Divers Alert Network (DAN).

He was transported by jet, at 1 ATA. I think that sea level transfer is essential. We have a similar problem in that we dive in Imperial Valley which is below sea level and we have to get back over 5,000 foot mountains to San Diego. When he arrived at the University of California, San Diego (UCSD) he had an adequate urine output and his vital signs were much the same, respiration. 35-40, pulse 105 and seemed to be not too bad. He complained of double vision, blurred vision and back pain which we attributed to being on a trauma board for 10-11 hours. We examined him and found marked cerebellar dysfunction which was the main neurological problem. Interestingly, he had very little in the way of motor findings, or sensory findings. In his right arm there was some sensory deficit, but marked past pointing when trying to do finger to nose and having him follow the examiner's finger. He was placed in the chamber after arterial blood gases were obtained. During transport he had been placed on 6 l/min oxygen and his PaO_2 as he came through the door was measured at 43 mm Hg. On 10 l/min of oxygen the PaO_2 jumped to 79 mm Hg.

We have been in the habit of taking chest X-rays before putting people in the chamber at UCSD. This patient had bilateral diffuse alveolar infiltrates, in addition his ECG showed that he had elevated P waves and, in retrospect, probably pulmonary hypertension. Back pressure pushing through the right ventricle into the right atrium creates the effect seen on the ECG. Precordial ST segment and T wave changes were also present but he had no evidence of myocardial infarction. His chest X-ray showed no evidence of preumothorax. He had a diffuse ground glass appearance to both hemithoraces and he was intubated. He had no subcutaneous emphysema anywhere. In retrospect again, there was a line which represented the reflection of the pleura on the pulmonary artery separated from the artery by an airspace and there was another airspace that pushed the pleura away from the aorta. There was also a suggestion that there might be a preumomediastinum as well as air along the left cardiac silhouette. The chest X-ray finding is interesting and it is unusual to have a diffuse generalised process. It was something we did not expect. The alternatives for that picture include aspiration, diffuse alveolar haemorrhage and even shock lung.

We took him into the chamber after this 45 minute work up and treated with a USN table 6A. In our unit we usually treat patients on a table 6A for what we suspect is AGE. This is a controversial topic. Table 6A is initially to 50 msw (165 fsw) on air then back to 18 msw (60 fsw) on oxygen with airbreaks. In the chamber he bled a couple of hundred ml then promptly closed up and subsequently did not bleed at all. This is what was interesting. We had no trouble with him after that. He rapidly improved and he was extubated the following day. He was on 50% oxygen and there was improvement in his neurological deficit especially the right arm sensory deficit as well as the past pointing. However his dysdiadochokinesis and his cerebellar dysfunction remained. The chest X-ray gradually cleared. There is no evidence of subcutaneous emphysema and there was no crepitus. He was treated again 30 hours post injury, this time with table 6. Improvement in cerebellar dysfunction was noted at this point and he had a CT scan of his head which was read as completely normal. By the 3rd day his chest X-ray had cleared further. He began to walk alone and was recompressed 50 hours post injury to 14 msw (45 fsw). This is the pressure where most of our clinical hyperbaric patients are treated. We treated him a further 3 times and as there was no further neurological improvement we stopped.

After the last treatment the neurological deficit was a wide based gait, his heel toe walk was abnormal and there was a residual abnormality of rapid alternating movement

of his left arm. A PaO_2 breathing air of 86 showed the resolution of his pulmonary problem. The haematocrit had dropped significantly suggesting a loss of about 25% of his red cell volume. The CPK peaked at about 5000 on the 3rd post injury day in the skeletal muscle band and this may have been ischaemic injury to the skeletal muscle. His ECG prior to discharge was normal and the CPK in the heart muscle band was not elevated so there was no evidence of myocardial infarction.

CPK is an interesting enzyme and we are studying if it can be used as an adjunct to diagnosing AGE. There may be evidence that ischaemic muscle injury has occurred. Such injury releases the enzyme and it can be detected biochemically. A significant muscle injury may represent massive or significant AGE.

Just prior to discharge he became shaky, had visual problems and felt uncomfortable. Most of this, I think, was apprehension about going home and the fact that he was an anxious individual. Other interesting results were a normal EEG while the nuclear magnetic resonance (NMR) scan showed cerebellar and cerebral cortical volume losses without any evidence of focal deficit. This goes back to what Dr How was saying earlier. Our patient had a normal CT scan and EEG but an abnormal NMR scan. It may be that the Though the NMR is more expensive it may be more sensitive to the specific neurological injury that we can not find clinically. He was discharged and went home to Alaska on his 6th post injury day.

Discussion

The mechanism of pulmonary barotrauma is something we have covered. It is an ascent injury that occurs from the lung actually rupturing. It can create a situation in which the gas runs either down the outside of the vessels or inside the vessels.

The pulmonary injury resolved fairly rapidly both on the chest X-ray and the arterial blood gases. In a series of our patients with AGE problems we found that the chest X-rays have not been very helpful. However looking at them retrospectively we can see small areas of pneumomediastnum but they are very subtle findings. They certainly were not of the magnitude that would require clinical treatment. From the submarine escape papers published, they did not do chest X-rays prior to recompression before about 1964. In those days the patient was picked up and put directly in the chamber. Today I think our cardiac resuscitative measures before and while we are in the chamber are a bit better. Their findings of evidence of pulmonary barotrauma on chest X-ray, whenever they were taken, was not extensive. That was surprising as one would expect from submarine escape training that there would have been more radiographic evidence.

Haemoptysis is a real problem for thoracic surgeons and physicians. In most non-diving patients it is is related to pulmonary arteriole rupture and some sort of bronchial pathology which leads to relatively rapid bleeding into the bronchus. This becomes a problem in airway maintenance. Another mechanism we can suggestis pulmonary hypertension, especially in this patient where there was evidence of pulmonary hypertension based on his ECG, leading to rupture of a pulmonary arteriole in a localised fashion. A third theory is that a diffuse form of bleeding occurs. The reason that I mention pulmonary hypertension is because this patient bled for about 12 hours following the incident and to get bleeding of that magnitude for that length of time there must be a higher than normal head of pressure in the pulmonary tree. We define haemoptysis usually in terms of both amount and time. Serious haemoptysis is 800 ml/24 hours. This is significant only if the patient drowns in his own blood. Submarine escape training is the primary model for arterial gas embolism. The trainees ascend from up to 100 feet at a rate 6-8 times that we normally use on scuba. So it is a pure ascent related thing. Even with these, haemoptysis is relatively rare.

Summary

I have presented a case of AGE complicated by massive haemoptysis. His cardiopulmonary arrest may have been due to the fact that his airway was not well controlled. Intubation is the key form of treatment at this time. Such patients do not need bronchoscopy, pulmonary angiography, bronchial artery angiography or CT scan when they prtesent but do need immediate recompression. This patient with his neurological insults and delayed recompression did quite well. He is presently active.

Questions and answers

Dr C Lourey

Do you have any idea what percentage of patients who have cardiopulmonary bypass get AGE? It is my impression that the incidence of AGE varies from nil in cardiothoracic units where there is no hyperbaric facility to a significant number in units where there is a hyperbaric facility.

Dr A. Santos

That goes back to the history of cardiothoracic surgery. The worst thing a cardiothoracic surgeon can do is to pump air into the patient. One takes blood from the venous system, oxygenates it, then pumps the oxygenated blood back into the aortic arch. If one does not watch the level in the oxygenator one can pump air into the patient. Unfortunately most thoracic surgeons know very little about hyperbaric medicine. There have been a number of patients in this situation and the surgeons did not know what to do about it. In thoracic surgery the patient completely

antiocoagulated, sometimes they get steroids and they are fully oxygenated. What a perfect set up to be taken to a chamber to be fixed and one would think the survival should be excellent.

Unfortunately one of my first experiences was a 39 year old woman operated on for an atrial septal defect. She got some air because of the technique and they did not do anything about it, they just basically let her go. It is only recently that a chamber has been put in that city. That represents the sort of problem that cardiac surgery can present. There is one surgeon in the US who operates in the chamber. He has been jumping up and down for years and no one has ever listened to him. Cardiothoracic surgeons are not the only ones in medicine who create that sort of a problem. People on dialysis have the problem with their arteriovenous fistulae when the dialysis machine infuses air into the patient. Neurosurgery also does the same thing to people sitting up when, especially in the posterior fossa approach, air can be sucked into a vein because of the low venous pressure. These require the same sort of recompression treatment.

Dr A Slark

I have a suspicion that this sort of case presents great concern to the people managing it because the physician is alarmed at the sight of blood and the surgeon always wants to do something, actively to put his finger on the leak. As soon as you put a patient in a chamber you have taken him away from the person who can actively stop the bleeding. Did you have a great deal of discussion about this particular pattern of management in this most unusual case?

Dr A.Santos

No. The uniqueness of the situation dictated that he just go to the chamber and no one thought any more about it. It is only one case so one can either postulate nothing or conversely one can postulate everything. It is certainly a bad time. One needs to have somebody in there with him, one needs to maintain the airway in the chamber and if something does go awry or persists too long one needs to do something about it. With diffuse pulmonary haemorrhage that would be very tough problem. Conversely if it was bronchial artery rupture we now have the capability of putting a catheter into the site of the rupture and embolising gel foam or clots into the artery and stopping the bleeding that way.

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HYPERBARIC MEDICINE IN CHRISTCHURCH, NEW ZEALAND, 1979-88

Michael Davis

Introduction

In 1980 at the SPUMS meeting in Singapore, I reported on the establishment of a new hyperbaric chamber at The Princess Margaret Hospital (TPMH), Christchurch, and our first year of activities[1]. The chamber was then, and still is, the only hospital-based multi-place chamber in New Zealand. Monoplace chambers exist in hospitals Dunedin (currently non-operational), Auckland and Wellington (operational). Until a decade ago, the only chamber actively treating divers was that at the Naval Base in Auckland, and even today about 80% of all decompression sickness (DCS) and cerebral arterial gas embolism (CAGE) cases in New Zealand are treated there.

The Christchurch facility's history is an interesting one. In brief, the chamber and a small compressor were donated to the Hospital Board by the local diving community in 1979 following a vigorous fund-raising effort. The whole thing was installed on the cheap, the Board undertaking to house and maintain the unit, but no funds were earmarked to establish a clinical service, staffing was to be on a voluntary basis.

The chamber was generally regarded to be a White Elephant, and Harry Guy (a respiratory physiologist now working for NASA) and I to be a pair of eccentric quacks. However, the referral of 33 patients in the first 15 months brought about something of a crisis, forcing the Hospital Board to address rather more constructively the provision of a "service", however rudimentary. Board policy was and still is that we may provide an acute emergency service only, and that there would be no additional staffing establishment to do it! Nevertheless, within these limits the Board has been supportive in many small ways over the years to maintaining our rudimentary service. At a time of severe financial constraints in health care in New Zealand, the establishment of formal hyperbaric medical services is likely to remain a low priority compared to many other perceived needs.

Despite the voluntary nature of the service, we are surprisingly well staffed. Medical cover is currently provided by 7 doctors, 3 consultants (2 anaesthetists and a geriatrician), 3 registrars and a family physician, all on a ring-round basis, there being no obligation to make oneself available. Only the family physician is paid on a fee-for-service basis by the Hospital Board, the rest of us do it for free, over and above our usual commitments. Only I had previous hyperbaric or diving medical experience.

We have 10 chamber operators. Four are Board employees, whilst the other six are local divers on whom

we rely heavily. Patient attendants are drawn from a voluntary pool of TPMH nurses who have undergone an introductory 18 hour hyperbaric nursing course run by me. Nurses are generally called when on duty. They are free to decline the call if they wish to. We try to keep our pool above eight to ensure nursing availability on this emergency basis. As you will appreciate, these arrangements do not make for an efficient service, yet the whole thing works surprisingly well because of the enthusiasm of all those involved. In fact, we have a waiting list of people wishing to work in the chamber!

Monoplace Chamber, 1972-78

Christchurch was no stranger to hyperbaric medicine even before 1979 as a Vickers monoplace chamber was used from 1972 for a trial of hyperbaric oxygen (HBO) in head and neck cancer. Between 1972 and 1978, 108 patients, 87 in the cancer trial, 18 with presumed clostridial infections and 3 others received a total of 693 HBO sessions. Apart from minor aural barotrauma in some patients, the only complications of therapy were three oxygen convulsions, none of which resulted in any morbidity. The Vickers chamber was sold to the Royal Adelaide Hospital in 1980.

Clinical Experience

Our clinical experience in the last nine years is summarised in Table 1. What lessons have we learned? Whilst this is a small case load by world-wide standards it has certainly been a learning experience for all those involved.

CLOSTRIDIAL INFECTIONS

Our Christchurch experience in the management of Clostridial infections since 1972 was reviewed two years ago[2]. There is little to add since then, except to note that over the last five years there has been a definite fall in the number of cases referred. This seems to reflect a real reduction in the incidence of this devastating infection, perhaps related to changing practice in the use of prophylactic antibiotics for surgery and trauma.

CEREBRAL ARTERIAL GAS EMBOLISM (CAGE)

Our overall experience with CAGE is too limited to draw useful conclusions from, except to say that we have not been vigorous enough in the use of HBO for this condition. Other disease entities such as osteoradionecrosis represent only a smattering of cases. I therefore propose to review only our experiences with decompression sickness (DCS) and carbon monoxide (CO) poisoning.

DECOMPRESSION SICKNESS

Of the 49 cases with a presenting diagnosis of DCS, six were considered to have musculo-skeletal problems unrelated to diving, whilst seven patients were managed, some in the monoplace at Wellington Hospital, by the Critical Care team in phone consultation with ourselves, leaving 36 patients who were managed in Christchurch.

Six of these patients were not given hyperbaric therapy: One refused treatment, three were observed in hospital and two late (over 10 days) referrals were declined treatment as no longer acute emergency cases. Reviewing the medical records of the latter five patients, the decision not to provide hyperbaric care was incorrect in all, and follow-up was incomplete, so whether they made a full recovery is unknown. Clearly, in suspected DCS, observation is not sufficient, hyperbaric oxygen therapy is required. A patient whom we initially managed incorrectly exemplifies this basic rule:

TABLE 1
CHRISTCHURCH PATIENT REFERRALS, OCTOBER 1979 - DECEMBER 1988

Presenting Diagnosis	Referred	Treated
Decompression sickness	49	30
Cerebral arterial gas embolism	10	3
Carbon monoxide poisoning	77	61
Clostridial infection	26	21
Radionecrosis (soft tissue and bone)	9	5
Miscellaneous	11	4
Total	**182**	**124**

Case 1

This 40 year old man undertook six dives (three on each day) to varying depths between 24 and 45 m (80 to 150 ft). His dive pattern grossly exceeded the no-stop limits but he undertook no decompression stops. He presented two days later to Wellington Hospital with mild persistent shoulder pain. It was decided by phone to manage him conservatively with anti-platelet therapy, high flow oxygen and observation rather than undertake the considerable expense of an air evacuation at that stage for mild symptoms.

By the following morning, he had developed neurological signs of a cervical cord lesion! He had hyporeflexia on the left side and areflexia on the right with bilateral up-going plantar responses. Power was diminished in the right arm particularly proximally. However, tone, gait and posture were all normal, with a negative Rhomberg's. There was diminished pin prick sensation over the right arm particularly the C5-7 dermatomes. He denied previous trouble with

his right arm. CXR showed a small area of atelectasis at the left base and a small amount of pleural fluid on the right.

Most of these features resolved with HBO but he remained slow mentally and hyporeflexic. With a history of over a decade of similar diving practice he presented very much the picture of the "punch-drunk" diver!

As our experience has grown we have become less impressed by any distinction between Type I and Type II DCS and their epidemiology. In our series only six patients had "classical" Type I symptoms alone, whereas 24 had neurological signs and/or symptoms. In nine, these features were combined and three patients also had respiratory symptoms at some stage. Thus in our experience, DCS in sports diving is a predominantly neurological disease, an impression seemingly shared by others. Whether the pain-only patients all console themselves in the pub is a moot point! Case 1 again illustrates the point that ALL DCS must be taken seriously. The costs of air evacuation of a diver with mild symptoms may seem awe-inspiring at the time but they fade into insignificance in comparison to the costs of neurological rehabilitation!

Another interesting aspect has been the chronology of symptom onset. Francis et al[3] have recently reviewed the onset of DCS in a large series of patients drawn from centres all over the world. Their data clearly demonstrates the acute nature of DCS with about 80% of cases having an onset time of *less than one hour*, 40% within 5 minutes of completion of the dive! In our small series the onset times were very similar to these reference data with 38% at 5 min, and 60% within one hour. By contrast, it took over 24 hours for two-thirds of the patients to reach our unit. Much of this delay was the fault of the patients themselves in failing to present for treatment for some considerable time. Two typical examples are:

Case 2

A 49 year old qualified diver with 9 years' experience, carried out a dive to 31.5 m (115 ft) for 20 minutes. He made too rapid an ascent because of bouyancy problems. About three hours later he developed chest tightness and felt uncoordinated in the right leg. Suspecting he had a bend, he went back down to 3 m (10 ft) for about 5 to 10 minutes, then breathed oxygen on the surface. This had no effect, but he went home anyway! Overnight he noticed continuing incoordination, weakness and paraesthesia in his right lower leg and difficulty passing urine. He finally presented to hospital the following day and was helicopter-evacuated to Christchurch. He gradually improved over several days of HBO, but has remained impotent since the accident.

Case 3

This 45 year old qualified diver with over 10 years' experience was wreck diving using an Orca "Skinny Dipper" dive computer. He did three dives. The first dive was 31.5 m (115 ft) for about 15 minutes. He then had a 2 hour 20 minute surface interval. The second dive was to 31.5 m (115 ft) for about 15 minutes. About 15 minutes after this second dive, during which he had a physically demanding swim with a heavy load of contraband, he noticed that he was unsteady standing and had pins and needles in his legs. He rested for a while and then elected to do a third dive to 21 m (70 ft) for 5 minutes with stops at 10 m (20 feet) for 5 minutes and 3 m (10 ft) for 10 minutes. That evening he developed progressive weakness and numbness from the waist down and had difficulty passing urine. However, he hid his symptoms from his diving buddy as they sheltered out overnight under a growing NW gale. The following morning evacuation was extremely difficult and he did not reach the chamber until mid-afternoon. During his evacuation a medical officer discontinued oxygen "because he was not cyanosed"!

On arrival, he was paraparetic to the T9/10 dermatome. Following 12 HBO treatments he had an incomplete recovery, but was able to return to work.

One message here is that the sports diving teaching agencies must teach new divers the importance of seeking out qualified medical advice early after a dive if they feel unwell. The modern trend with some agencies seems to emphasise the safety of the sport but to exclude any mention of such disastrous consequences as this to divers. Likewise, the medical profession needs to be better informed on diving medicine and its acute management, so that decisions such as that above concerning oxygen are not made!

Hyperbaric Management of DCS

It has been instructive to look critically at our management of DCS. In 14 patients, the initial treatment was a short oxygen table (US Navy Table 5), modified in five cases for the Wellington monoplace prior to transfer to Christchurch. One patient underwent a 10-hour unsupervised air treatment in a non-hospital chamber prior to referral. In the remaining 15 cases, a US Navy Table 6 was used, only three being with extensions.

The distribution of the number of treatments per patient is shown in Table 2.

In recent years we have learnt to treat more vigorously initially and provide repeat therapies more readily. Now Table 5 would be regarded by most as inadequate initial treatment and we now rarely employ it as the first treat-

Table 2

HYPERBARIC OXYGEN TREATMENTS FOR PATIENTS WITH DECOMPRESSION SICKNESS

Number of HBO	1	2	3	4	4+
Number of patients	15	7	1	2	3*

* these were given 5, 11 and 12 HBO treatments respectively.

ment except for 'missed decompression' in asymptomatic patients. In several cases the initial treatment was almost certainly too conservative and the patient was left with residual neurological signs at the end of his course of treatment. To be fair to ourselves, in the vast majority of patients a good outcome was eventually achieved, but the impression is certainly that we might have enhanced recovery rates by being a bit more vigorous initially. Because of our limited facilities and relative inexperience we have been reticent about embarking on very prolonged treatments. The following case illustrates this point:

Case 4

This 29 year old man undertook three dives in a morning, the first 30 m (100 ft) for 30 minutes, the second 42 m (140 ft) for 25 minutes and then 42 m (140 ft) for approximately 25 minutes. No decompression stops were undertaken. About 30 minutes after the third dive, he developed severe chest pain and painful joints. He then convulsed, remaining unconscious for about three quarters of an hour. During this time he was transferred to hospital and recompressed in a monoplace oxygen chamber to 3 ATA and held at this depth for 60 minutes before being decompressed and air evacuated by helicopter to Christchurch.

On arrival he was mentally alert and orientated to time and place but shocked, with a systolic BP of 70 mm Hg. He was completely paraplegic below T5/6. Some weakness of the left arm was noted, but this was not examined properly because a drip was in that arm.

After 40 minutes HBO at 60 ft when he had been given 2,000 ml lactated Ringer's solution, he was warm and well-perfused peripherally. He was now able to straight leg raise against moderate resistance, pin prick sensation was present, but still diminished from T7 on the right and T10 on the left. Light touch and proprioception were normal. Because of the considerable neurological improvement at this stage it was decided to treat him on an extended USN Table 6 with 2 additional 20 minute oxygen periods at 60 ft and 60 minutes at 30 ft.

However, when first re-examined after the ascent to 30 ft, there had been significant deterioration in power and sensation, and he was tachypnoeic. Despite this clinical deterioration, it was decided to complete the extended Table 6 as planned, finishing about 0400 hours. Despite 4,000 ml of intravenous fluids during HBO his haematocrit was over 0.65 at the end.

By 0800 hours he again had a complete T5 paraplegia, as well as diminished power in both arms, especially for fine hand movements, this being worse on the left than the right, and pins and needles in both hands. No objective improvement occurred with another Table 6, but by the end of two weeks of daily HBO, his arms had fully recovered, muscle power in the legs was grade 2-3, and he had deep pressure sensation in his buttocks and thighs. Three years later he is walking with the aid of 2 sticks but still unemployed.

The message is a well known one. Treat vigorously and to the maximum capability of your facilities. Treat repeatedly, until no further improvement is seen with each treatment, and do at least one more beyond that point.

Evoked Potentials Measurements

An area of assessment of DCS patients in which we have become interested recently is the measurement of evoked potentials (EP). The general view at the UHMS Hawaii meeting in 1989 seemed to be that somato-sensory evoked potentials (S-SEP) do not greatly contribute to management or research, since those with abnormalities invariably have clinical signs anyway. However, in addition to S-SEP, we have been evaluating brain stem auditory EP and visual EP. In several divers these have been abnormal without concomitant clinical neurological signs, and have shown recovery with time. We are beginning to collect some long-term follow-up data as well, but so far there is too little for further comment. It would be interesting to know whether other chambers have been using all three EP measurements on divers.

CARBON MONOXIDE (CO) POISONING

There remains disagreement in medical texts regarding the value of HBO in the management of CO poisoning. Despite this, in the author's opinion there is convincing laboratory and clinical data to support the view that HBO is the treatment of choice in CO poisoning. There may even be a case for urgent transfer of such patients from a distance, as recent clinical reports suggest that delayed HBO may also

be of benefit in preventing long-term sequelae. Readers are referred to a recent review in "Surveys of Anesthesiology" for an up-to-date statement on the evidence4.

Of the 77 patients referred for acute CO poisoning, 45 were from suicides and 16 accidental. Fifty seven were male, with ages ranging from 16 to 78 years. In the early 1980s it became apparent that a significant proportion of CO poisonings in Christchurch were not being referred for HBO. This was readily corrected by a medical grand rounds presentation, and the establishment of clear guidelines for referral. Since that time we have received 10 to 15 cases annually.

Our guidelines for referral are any of the following:

1 Asymptomatic with carboxyhaemoglobin (COHb) level of over 35% (now reduced to 30%);

2 A history of impaired consciousness at any time;

3 Neurological or behavioural signs and symptoms of any sort, excluding headache alone, on admission, irrespective of the COHb level.

Until recently the majority of patients had received only a single HBO treatment (either 60 minutes at 2.8 ATA or a US Navy Table 5). Increasingly we are now repeating HBO as our follow-up and liaison with the medical teams has improved. Recent evidence in the literature demonstrates that repeated exposures result in improved outcome.

The average admission COHb level was 33% (range: 2-57%). We place no reliance on the initial COHb in determining severity or prognosis. It is the clinical picture that is important. There was only a poor correlation between the admission COHb and the time interval between the end of exposure and sampling. Of the 77 patients, three died, and four were left with residual neurological problems. The % COHb levels were 11, 57, not known, 32, 39, 47 and 57 respectively in these patients. Thus, we have an overall treatment "failure" rate of about 9%.

It is widely believed amongst the general medical community that a CO poisoning victim suffering cardiac or respiratory arrest is unlikely to survive. In our series there were 8 patients who required Rescue Breathing or full CPR by ambulance teams at the scene of the exposure. Of these, four made a full neurological recovery, whilst two suffered residual neurological damage and two died. Clearly the outcome is worse in this group, but nevertheless the possibility of a successful outcome remains in all cases.

We were particularly interested to look at the delays in retrieval of these patients. Unfortunately accurate timing of discovery, ambulance retrieval, admission to the Emergency Room (ER) and commencement of HBO could only be ascertained in 33 patients from the metropolitan Christchurch area. In these 33 patients, the interval from discovery to HBO was two hours, ranging from one to five hours. We felt this was not particularly good for a city which you can drive across in under 20 minutes !

Of even greater concern was that most of this delay occurred in the Emergency Room, the mean time interval from arrival in the ER to commencing HBO being 96 minutes, with a range of 20 to 280 minutes. In a few cases this was the result of delays in finding one of us, but this was never more than about 30 minutes. Our task is clearly to engender a greater sense of urgency about HBO referral in junior ER doctors. In the last two years the St John Ambulance have been circumventing this whenever possible in serious cases by bringing victims directly to the chamber, rather than taking them through the ER. Looking at this problem chronologically, there has been a definite reduction in time delays in recent years compared to the early 1980s, which is encouraging.

The Future

So far the recompression chamber in Christchurch has survived on the enthusiasm of a small band of dedicated volunteers. However, this is not enough to ensure the lasting development of hyperbaric medicine in New Zealand, particularly when this field represents only a small portion of one's professional activities. Given the present crisis and dramatic changes in the delivery of hospital services in this country there is little chance of further developments in the foreseeable future being achieved from within our region.

However, several external pressures may change this. Firstly, there is an increasing awareness within the sports diving community of the need for properly funded hyperbaric facilities in New Zealand. In the last few years this has resulted in an increasing commitment from the New Zealand Underwater Association in this area, with the funding of the DES phone at HMNZS PHILOMEL, sponsoring of diving medicine courses and involvement in planning strategies for better hyperbaric facilities in Auckland. This is very much to be applauded, since without the public commitment of the diving community further developments are very unlikely.

Coupled with this, changes in the pattern of diving around our coasts continue to occur and may well force matters along. For instance, in South Island, in a little over two years the Mikhail Lermontov wreck in the Marlborough Sounds has claimed three lives and produced at least four DCS cases. This and the opening-up of Fiordland for sports diving, magnificent deep drop-offs in a geographically extremely remote region will surely present us with major problems in the years to come. In the last decade our diving accident referral rate in the South and Central regions has not risen, in sharp contrast to many other parts of the world.

For some years I have suspected this to be the calm before the storm!

What happens in the Auckland region will directly influence us in Christchurch. With nearly half the country's population centred there, there is little question that a major facility, similar in size to that in Adelaide, could be sustained and would be cost-effective. How the Minister of Health's Commissioner, Harold Titter, might be convinced of this, whilst looking to prune some NZ$ 60 million from the Auckland Area Health Board budget is an entirely different matter!

Finally, from the hyperbaric medicine viewpoint, developments in Australia are particularly important to us. It is essential that the new facilities being opened fulfil more than a service role, publishing high quality clinical and applied sciences research to back up the claims for hyperbaric medicine in various fields. Equally important will be the outcome of the UHMS submissions for the current Blue Cross/Blue Shield funding review of HBO. A successful defence of hyperbaric medicine in the United States over the coming year will be vital to the future development or otherwise of hyperbaric medicine throughout the world, let alone in Christchurch, New Zealand.

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THE BITE OF THE WAHOO

J McKee

The Wahoo is a fish of the tuna/mackerel family and resembles the Spanish Mackerel. It grows to about 45 kg (100 lb) in weight and six feet in length. It is very streamlined and is said to be the fastest fish in the ocean, at times reaching speeds of 80 km/hr (50 mph). It has a very large mouth and very sharp teeth with serrated edges and incredible cutting power with its jaws. The distribution of the Wahoo is mainly around Australia from the North West of Western Australia, across the northern coast, along Queensland and New South Wales as far as Lord Howe Island. The fish is a renowned game fish and Hemingway reports it is the best eating of all the game fish. The fish feeds by catching its prey and biting off the tail then devours the rest at its leisure. It is a fast game fish which is also good eating.

My interest arose by accident when asked to see a patient who had been bitten by a Wahoo. This was the first time I had seen this injury and I present it to reveal how incredibly sharp the teeth are. The teeth are one of the wonders of the sea. As far as I know they do not attack humans in the water. Usually the bite is an accidental attack on a fisherman or by some one brushing the skin against the Wahoo's teeth.

The patient was a Melbourne builder on holiday, who went out Marlin fishing and accidently caught a Wahoo. Just as it was brought into the boat, it decided to have its recognised last bite at anything close and it managed to seize the patient's lower leg. One single bite. When this solidly built builder presented to the Casualty Department he had no extensor power in his ankle, it was just a floppy foot. Under anaesthesia the reason for this became apparent. There were 2 wounds, a horizontal wound at the level of the medial malleolus about 5 cm long, absolutely clean, no contamination, incised as well as any surgeon could do with a scalpel. On the lateral side there was a similar horizontal incision a little higher again cleanly incised.

On exploration of the outer wound the bite had gone right through the bone and in doing so, divided 3 extensor tendons. Over the next 2 hours both wounds were debrided for safety's sake, the tendons repaired and the medial wound was then explored. I was amazed to find the medial ligament totally divided. This gives the principle medial support to the ankle and a further extensor tendon had been damaged. In all, over two and a half hours were spent cleaning the wounds and repairing the damage.

It was explained to the patient that as far as we know, that particular wahoo had been eating fairly clean fish of late. With the passage of time and antibiotics there was no subsequent contamination and he went back to Melbourne.

LETTERS TO THE EDITOR

LETTERS TO THE EDITOR

3/9 Muriel Avenue
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25th January 1990

Sir

I have for a long time now shared the following views with just a few of my close associates for fear of being publicly branded a radical (not that this is necessarily disputed !), but recent events compel me to speak out on behalf of, I believe, a large number of EXPERIENCED divers.

During my nearly two decades involvement in scuba activities, I have on numerous occasions endured the sarcastic, self-righteous attitudes of know-it-all theorists, neophytes, diving instructors, doctors and a variety of "professional" divers who felt that it was their God-given duty to teach me the evils of "breaking the Sacred Diving Laws". You know the "Commandments", never dive alone, never do decompression dives, never do two deep dives the same day, etc.

Such annoying (but understandable) noises have recently become more than mere jibes. I believe that we are now beginning to see the growth of an "Ultra-Conservative Wowsers" cancer and fear that, like a religion, such intolerant and disrespectful attitudes will dramatically affect us all if we do not DO something about it soon!

Let me hasten to add that I have the utmost respect for the work being undertaken by such diving medicos as Doctors Des Gorman and Carl Edmonds (both of whom I have known for some years). my interest in diving safety in fact prompted me to become an accident investigator for Dr Douglas Walker's "Project Stickybeak" in 1979, and since I recently published a book to do with South Australian accidents[1], I believe that I am far from ignorant of such matters. Sadly, this is not the case with 95% of even the most experienced people, because they rarely learn about more recent medical discoveries and still believe that publications like the "Divers Medical Companion" are fairly accurate and up-to-date (a view which is NOT shared by some diving specialists). I am also involved in a considerable amount of voluntary research diving, and the fact that I have safely performed more than 200 dives to depths in excess of 30 metres (fairly typical of the diving done by most Mount Gambier cave divers) should put me in some sort of position for commenting on these activities, unlike many of the critics!

The main problem is the "I know best" attitude which is being forced down our throats. I would not be at all surprised in the next 10 to 20 years to see this wonderfully conservative "Big Brother" country of ours introduce Draconian laws which would ban recreational diving if on-site recompression chambers, surface-to-diver communication equipment and surface-support teams were not used in all dives beyond 17.9 metres.

Professional divers such as those in the police force are already required to follow such restrictive and expensive Australian Standards (no doubt for occupational health and compensation purposes), and I fear that desk-bound, autocratic "experts" and rule-makers will fail to see that recreational divers should NOT be slotted into the same category as professionals, since they wish to dive for FUN and are NOT being employed.

Too much ado about nothing? Signs of such disturbing trends became obvious to me recently when I took the often-promoted "responsible" action of seeking hyperbaric therapy for some suspected DCS symptoms (which appeared after I was forced to skip a few minutes of planned decompression to search for a missing diver). A senior medical supervisor with extensive training in hyperbaric medicine (but no personal diving experience or knowledge about my own) accused me of being irresponsible for doing a dive to 36 metres without all of the professionally-required support gear (even though our dives occurred within 15 minutes of a portable RCC), and I was further flabbergasted when a chamber operator came out with statements such as "We would ban all sports dives beyond 18 metres if we had our way" while condemning me for "wasting tax-payers' money"!

I cannot condone such ignorant and inflexible attitudes in hyperbaric or dive training circles, and I am fed up with lectures by inept, goody-goody novices who seem to have a propensity for quoting the "Sacred Diving Laws" ad nauseam. A responsible awareness of safe practices is one thing (and by all means, dive shops should ensure that novices are taught only the SAFEST practices in their BASIC courses), but people need to realise that there are different levels of diving skill and different ways to assess risk factors. I sometimes wonder just how far this conservative outlook is spreading when I see dive shop staff under-filling scuba cylinders for fear of accidental explosion occurring (even though they are in test and hold less air than aluminium cylinders when they are actually filled properly)!

The basic fact of the matter is that, contrary to many "experts" opinions, some of us DO happen to know what we are doing. We believe that we have the proper experience and knowledge to tackle the known risks responsibly, and we DO NOT like being told how we WILL or will NOT dive. We believe in following the RECOMMENDATIONS where possible, but I feel that some of you medical and professional diving people need to realise that one can be TOO obsessed with safety in recreational activities. Who would

enjoy skydiving if jumpers were forced to wear six parachutes, or rock-climbing if a standby helicopter had to be on site at all times?

By all means, PLEASE continue with your important studies and publish your findings as widely as possible, but ALSO try to treat us as being responsible and partially-intelligent people who deserve the same respect which you expect from us!

Yours sincerely
Peter Horne.

Reference

1 Horne P. South Australian diving fatalities 1950 - 1985. 2nd edition. Adelaide: Australian Underwater Federation, 1987

GROUPIE DIVING

Department of Anaesthesia
The Christchurch School of Medicine
Christchurch Hospital
Christchurch, New Zealand
3 January 1990

Sir

The review of Australian and New Zealand diving fatalities by Edmonds and Walker is a valuable contribution to the epidemiology of diving medicine. Not only is the presentation of data first class, but the authors have appreciated that epidemiological work is not an end in itself, but that the data gleaned must be applied back to the situation being studied.

The difficulties experienced in interpreting such data are well illustrated by the buddy diving concept. Our problem is that we have no denominator. Whilst the majority of fatalities were associated with poor or absent buddy diving techniques, we do not know whether the breakdown of good buddy diving technique per se results in greater numbers of fatalities, or whether the proportion in this report merely reflects the overall quality of buddy diving techniques in sport diving. I suspect the answer to be the latter. However, it really does not matter, if Edmonds and Walker's view is correct that good buddy diving is likely to result in a non-fatal rather than a fatal outcome for an incident. There is, of course, no evidence for this, but it makes sense.

Unfortunately, what data we have does not help us to resolve this one either! What is clear is that training techniques for buddy diving have failed abysmally since it seems likely so many sport divers pay lip service to the principle. This implies that the training schools need to reassess the way this aspect of diving is taught. A further dilemma is the question whether solo diving is inherently more unsafe. Some divers (including myself) would argue that there are circumstances in which solo diving is an acceptable, safe technique. I think this whole issue needs very careful re-thinking by the educators in the sport diving industry.

Related to this, I want to pick on a particular bete noire of mine "Groupie Diving" (more than 3 divers together with a common leader or moniteur). The illogicality of this system, common to many SPUMS trips, is beautifully described by Edmonds and Walker in their section on buddy diving. There are several problems to Groupie Diving as it is run by many diving operators:

1 There is the implicit abdication of responsibility by the individual divers. This aspect is hotly denied by dive operators, but the "for we like sheep" mentality is assumed very rapidly. For instance.two dangerous incidents occurred during the diving at Uepi, before the SPUMS meeting at Honiara in 1987, arising out of this attitude.
2 There is an unacceptable level of risk acceptance for the dive leader. It is impossible to be truly responsible for 3, 4, or more divers at any one time underwater.
3 There is frequently no clear definition of individual responsibilities during the dive. Often only the leader knows the full dive plan and this may not take into account individual capabilities or wishes, etc.
4 There is considerable inertia in establishing a response to a diving incident. This involves bringing the problem to the dive leader's attention (distance, effort, intelligibility, etc.); checking all other divers, reaching a decision and finally, acting on that decision.
5 Responses to in-dive problems are often inappropriate. For instance, the designation of "low on air" divers to new buddy pairs (both low on air) for surfacing or the dive leader surfacing with the diver concerned and leaving the group leaderless or leaving him on the surface alone and then rejoining the group or ruining the dive for the entire group by surfacing everyone.

I believe that Groupie Diving is primarily commercially driven and arguments regarding its safety are merely a rationalisation of this process.

Whilst decompression sickness once again did not contribute to mortality, this is not to say it should be discounted. It is important to remember that sport diving decompression accidents carry a significant morbidity, as well as a major cost in their treatment and rehabilitation.

Finally, the discussion highlights the complex interplay of factors that contribute to diving accidents. It is very rare for one adverse factor alone to result in a tragedy. This being so, a rational approach to teaching dive accident prevention and management is feasible. This could follow the same broad principles as those underlying Bill Runciman's recent "COVER, A SWIFT CHECK" for an-

aesthesia practice presented at a meeting of the Faculty of Anaesthetists. Perhaps SPUMS and the dive training organisations should look at a similar concept for sport diving?

F. Michael Davis
Senior Lecturer in Anaesthesia

UNDER AGE DIVING

228 River Street
Ballina, NSW 2478
20 October 1989

Dear Sir

I was recently put on the spot when a 12 year old boy (accompanied by his father) presented for a diving medical, stating that scuba diving was an accepted sport at his school.

My immediate reaction was "no way", and a couple of quick telephone calls to underwater medicine trained colleagues confirmed my decision. I explained to the lad and his father my decision that the boy was too young to use scuba and my reasons for making this decision.

My reasons why a 12 year old boy (and other people under 16 years of age) should be considered unfit to dive are:

a. This age group does not posses the maturity or confidence to avoid a sudden panic and rapid surfacing, thus undergoing the risk of cerebral arterial gas embolism (which can occur at depths greater than 1.5 m (4.5 feet).
b. This group does not possess the maturity to fully understand and implement the "buddy" system whereby a diver in trouble may be completely reliant on his "buddy".
c. Although there is little evidence to support the possibility of rapidly growing bones (such as in this age group) being more sensitive to dysbaric processes, there is a real possibility that diving at this age, even well within USN or BS-AC no-stop bottom times, may lead to dysbaric osteonecrosis.
d. Persons under 16 are often of small stature with greatly varied physical appearance, which will inevitably lead to problems with ill-fitting equipment and discomfort, which will probably be accepted as just apart of training. Discomfort often leads to disability and subsequent trouble.
e. At the completion of a diving course, irrespective of "limited" qualifications, persons of this age groups are liable to disregard their limitations and be tempted into diving situations outside safe diving practices. This may add their names to the long list of diving casualties or fatalities.

After contacting the school and finding out that scuba diving had been a Department of Education accepted Class C sport for Year 7 and above for 12 months, I was taken aback. However, I pursued my original line of action and brought the matter to the attention of the school principal and area State School Sport Administrator.

Having had some time to reconsider the matter, I believe the appropriate response would be:

1. have any diving candidate, but specifically one under 16, examined by a doctor with recognised expertise in Underwater Medicine;

2. require that the candidate is sufficiently physically robust for the rigorous aspects of diving;

3. ascertain that the candidate is mentally mature enough, i.e. has the common sense required for safe diving practice and not be tempted to use his gained skills unwisely in the future;

4. be restricted to buddy line diving with an experienced older diver until requalifying at age 16; and

5. keep well within the BS-AC tables as the rapidly growing bone of the under 16 age group may be unduly sensitive to dysbaric effect.

In retrospect, I would still fail a year 7 student for school scuba diving even if he fulfilled the listed criteria as I think passing such an individual would be discriminatory and create undue peer pressure which may affect safe diving practice.

Colin Macdonald
MBBS LCDR RANEM

STATEMENT ON SPORT DIVING

The Diving Medical Advisory Committee
28/30 Little Russell Street
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Tel: 01 405 7045 Telex: 267568 IMCOSM G
31 October 1989

Sport diving has become big business. There are major commercial interests that service the sport diving field, including the provision of gear, instruction of new divers through schools and the organisation of diving related holidays. Sport divers have begun to diver deeper, longer and more often, with the use of increasingly sophisticated gear. The dividing line between commercial and personal diving has become progressively less clear as the capabilities of sport diving equipment have increased. Some sport divers,

tempted by the rewards of salvage, dive on deeper wrecks in the hope of turning their sport into a lucrative pastime.

Sport diving casualties account for the vast majority of diving injuries treated throughout the world. Recent trends in the numbers and types of diving casualties have created increased concern among members of the medical community and of this Committee. New scientific evidence heightens our concern that permanent central nervous system damage occurs following some incidents of neurological decompression sickness. Also of concern are the potential effects on other systems, notably the skeletal system, in the form of dysbaric osteonecrosis. These kinds of damage, while subclinical in most cases, may lead to serious long term disability and are, for the most part, avoidable with a reasonable degree of caution.

Whilst the majority of sport divers are considered to be well trained and responsible, there appears to be a prevalent philosophy among some that they can dive deeper, longer and more often without penalty., The following points are stressed:

1. The depth limit for North Sea commercial diving on compressed air is 50 metres. This depth is based on safety considerations and a recognition of the increasing risks to divers at greater depths. In a commercial situation, dives conducted at depths in excess of 30 metres are carefully controlled and normally require a recompression chamber on site as well as full supervisory backup. It is stressed that sport divers should never exceed 50 metres and that, in isolated areas or in the absence of proper supervisory personnel, a shallower depth is recommended. Thirty metres is considered a reasonable depth limit for most sport diving activity.

2. The single most identifiable cause of decompression sickness and other diving related problems is the time depth profile of a dive. Multi-day repetitive diving increases the risk of an incident. While a decompression incident can occur following a dive within the established limits of any table, dives involving decompression stops in the water are at an increased risk compared to dives conducted within established no stop times. Careful planning and execution of a dive remains the best way to avoid a diving related problem.

3. Dive only on well tested and accepted tables and stay well within the guidelines of these tables. Great care must be exercised in the use of decompression computers. Where used, they should be as a backup to a properly planned dive on accepted tables. Consideration must be given to known risk factors such as age, fatigue and degree of fitness. Never push a dive to the limits of your table and avoid incurring a decompression stop requirement if possible. No stop diving is recommended for most sport diving activity.

4. Diving is an exciting but potentially dangerous sport. Each year a number of divers die in diving related accidents. Others are left with a permanent disability. Decompression sickness is not an innocuous disease. Although the majority of divers appear to recover normal function following treatment, the end result in some cases is likely to be underlying central nervous system damage of a permanent nature.

5. Carefully planned and executed, diving can be a safe and enjoyable sport. Experience alone will not protect you and may lead to a false sense of security. The potential risks of diving must never be forgotten.

The Diving Medical Advisory Committee

1251 East Dyer Road #100,
Santa Ana,
California 92705-5605,
USA.
November 28, 1989

Sir

We have followed with interest your reprint1 of the Robert Monaghan *Undercurrent* article and the one published in the *SPUMS Journal* [2]regarding diver population and accident rates.

As those articles have shown, PADI and others have repeatedly refuted his claims and his misuse and misrepresentation of certain data. This final chapter deserves comment as well.

To put his “analysis” in perspective, the following are but a few of the misrepresentations Monaghan has made in his reports:

1. Monaghan claimed a PADI survey indicated an 80% annual diver drop-out rate. Actually, PADI’s survey reported the opposite, that the drop-out rate could **not** be 80% (Monaghan was informed of this misrepresentation but has continued to make it).

2. Monaghan claimed that published Australian diver fatality rates should be proof that published US rates are too low. What he did not share with readers was that the Australian rates he quoted were not total fatalities compared to total diver population (as the US rates are computed), but instead the total fatalities compared to the number of divers certified in a year by PADI Australia. Such a ratio would obviously be higher than the figures reported in the US.

3. Monaghan claims that "PADI Australia reports 20 diving deaths per 100,000 active Japanese divers". The term active is **not** used by PADI Australia's report and PADI Australia actually reported there were 19.4 fatalities per 100,000 Japanese divers in 1986 (according to the *Jinko Dotai Tokei* by the Japanese Ministry of Welfare). Incidentally, this figure fell to 0.6 fatalities per 100,000 in 1987. Monaghan then used this to again "prove" that the US rates must be too low. What Mr Monaghan withheld from his article, however, is that *Diving Accident Management in Australia* also showed that, despite the high overall rate, **PADI** divers in Japan had a fatality rate of only 3 per 100,000 (a figure remarkably similar to the rates independently reported for the US by NUADC).

4. Monaghan claims that Diagnostic Research Inc. (DRI) (the company that conducted a recent diver drop-out and diver population study for DEMA) reported "only 3 million" divers in the US and goes on to say that his model (with certain additions) produces similar results. In reality, the DRI study reported that there were between 5.27 and 7.07 million people in the US who had become certified divers at one time or another. Further, they reported that "2.65 million adults are current or active scuba divers". The DRI report went on to detail a statistical accuracy of +0.3, concluding "The estimated number of active divers ranges from a low of 2.09 million to a high of 3.22 million".

The DRI study, rather than supporting Monaghan's model as he implies, disputes it.

5. Further, Monaghan claims that "PADI's own US diver estimates are substantially less than the NUADC figures". Actually, PADI's estimate was 2.5 million **certified** divers (this number does not include, however, all uncertified divers, military trained divers, commercial divers, etc., a sizable group by any estimation). NUADC's estimate of diver population was 2.6 to 2.9 million. Since the Diagnostic Research Inc. study was released, however, NUADC has adopted the DRI figures and current fatality rates are based on DRI's population estimates.

Monaghan goes on to make several other statements that have no basis in fact. He implies that PADI claims there is a low death rate. PADI makes no such claims. PADI does, however, publish the data provided by bonafide organisations, such as the NUADC.

He also impugns the NUADC by implying that it is not "independent". This is spurious and uncalled for. The NUADC has been in operation since the late 1960s and its operating procedures meet the guidelines and audits of both the University of Rhode Island and the US Federal Government's National Oceanographic and Atmospheric Administration (NOAA).

Finally, Monaghan implies that those who report the NUADC findings "prefer to remain complacent about diving safety based on the low claimed death rates". The NUADC analyses have shown a clear trend of improving diver safety over the last 12 years. This has occurred because the diving community in the US has put forth significant effort and the results are showing. The fact that safety statistics show improvement needs to be known by the diving community, this knowledge serves as a powerful reinforcement that efforts toward diver safety are working.

Perhaps the most unfortunate aspect of this matter is that it is not the result of poor statistical science. It is not well-known that Mr Monaghan was employed by the law firm that filed the Dibble vs. PADI lawsuit. The lawsuit attempted to claim that PADI's methods were harming diver safety, NUADC's statistics contradicted those allegations and an attack on NUADC's statistics and its principle researcher, Mr John MacAniff, was begun. Monaghan's recent articles are only a part of that effort.

There is no question that the trend in the US is increased diver safety. In 1976, there were 147 recreational diver fatalities in the US and in 1987 there were 87. Even if one does not consider rates (which requires the defining of the total participant population), safety has clearly improved. However, we doubt seriously that anyone would claim that the number of divers has not increased since 1976! The conclusion that diving is becoming safer is shared by Divers Alert Network (DAN). DAN's Fall 1988 issue of the publication "Alert Diver", stated "while diving is not without risk, it has a much better safety record than many have previously believed, no matter what numbers you use or how you look at it".

When the statistics are presented correctly, it is clear that one important, undeniable trend is taking place. In the US, in Japan, and in Australia, as PADI's market share has increased, (currently exceeding 60% of divers trained), there has been a corresponding decrease in diver fatality rates. PADI does not feel this is, nor could be, mere coincidence.

Al Hornsby
Executive Vice President

References

1 Monaghan R. The risks of sport diving: just how many divers are there ? *SPUMS J* 1988; 18 (2): 53-60.
2 Monaghan R. Australian diving death rates: comparisons with USA and Japan. *SPUMS J* 1989; 19 (1):24-25.

Abbreviations used in this letter.
PADI = Professional Association of Diving Instructors
DEMA = Diving Equipment Manufacturers Association
NUADC = National Underwater Accident Data Center

This letter has been shortened for publication. Interested readers can obtain the original text from PADI Australia.

This correspondence is now closed. Editor.

CARDIAC SHUNTS AND POSTURE

Department of Cardiology
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23 January 1990

Sir

In the October-December 1989 issue there was discussion of the effect of posture on right to left shunting of blood in divers who have intracardiac shunts and what effect this might have on decompression sickenss.[1,2] Both papers mention that with head down and legs raised, the right atrial pressure is increased and the authors speculate that this could increase paradoxical gas embolism.

The flow across interatrial shunts is determined mainly, but not entirely, by the pressure gradient across the septum. In most instances, raising the legs, alters right and left atrial pressures similarly, but coughing does not. Therefore an individual in the head-down position will not necessarily increase the size of an interatrial shunt if one is present, but he may do if the weight of viscera on his diaphragm makes respiration laboured. Our published observations on altering posture of divers with demonstrable shunts, show that the head-down position has no consistent effect on shunt size.[3]

Peter Wilmshurst, MRCP
Consultant Cardiolgoist
Medical Adviser, British Sub-Aqua Club

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2 Davies D. Patent foramen ovale. *SPUMS J* 1989; 19 (4): 151-3.
3 Wilmshurst PT, Byrne JC, Webb-Peploe MM. Relation between interatrial shunts and decompression sickness. *Lancet* 1989; ii: 1302-1306.

DIVING AND CENTRAL NERVOUS SYSTEM DISEASE

Employment Medical Advisory Service
Health and Safety Executive
Greyfriars House, Gallowgate
Aberdeen AB9 2ZU
UK
January 1990

It is clear that in the carrying out of diving activities, both of a recreational and commercial nature, a small number of people each year contract acute, and in some cases long lasting, damage to the central nervous system.

This is a matter of concern because of the consequences for young lives and particularly for future employment prospects.

So far as the commercial sector is concerned the number of cases is small, but the individual consequences for future employment in diving are great.

Just as the Department of Energy is concerned to regulate diving practice to reduce the incidence of cases, we in HSE are concerned to discover whether there are predictive factors which can enable individuals to be advised against undertaking commercial diving, or particular forms of commercial diving activity, or in the event of "early warning" to be removed from continued diving work.

To these ends discussions are in progress with the Diving Medical Advisory Committee and other interested parties to agree on a research programme in which divers attending for medical assessment at centres in the United Kingdom may be invited to participate.

It is intended that the work done in the United Kingdom will be correlated with work of a similar kind being undertaken in other countries.

HYPERBARIC FACILITY FOR WESTERN AUSTRALIA

Hyperbaric Medicine Unit
Fremantle Hospital
Fremantle, 6160
Western Australia
11 December 1989

Sir

The new Hyperbaric Medicine Unit for Fremantle Hospital has opened, and within its first week was involved in the management of three cases of decompression sickness. It forms another link in the chain of hospital based Clinical Hyperbaric Medicine Units that is being established around Australia. This two chamber, three compartment unit is proving very flexible, and is already treating routine clinical cases.

The Unit has developed a close working relationship with the consultants in the Emergency and Intensive Care Departments at Fremantle Hospital, all of whom have undergone training courses in the recognition and early management of dysbaric illness, and other acute problems requiring emergency hyperbaric treatment.

The Chief Hyperbaric Technician is Tom Nalpon, lately of the Alfred Hospital Hyperbaric Unit, Melbourne.

A data entry program has been established both for diving and non-diving Hyperbaric Medicine cases.

Harry F Oxer
Director

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

PHILOSOPHY SECTION
(Skip if sober)

Bob Halstead

I love listening to Mike Emmerman talk about diving - he is full of wonderful ideas, very few of them conventional. Some of his recent research has highlighted the problems caused by flying BEFORE diving (dehydration, so drink lots of water when flying). He loves to dive and puts his ideas into practice, sometimes by making dives that are regarded by some authorities as "dangerous". For example, he dived the Andrea Doria, using air, and wrote about the experience in an article called "Crazy but not Stupid". We were discussing safety and Mike made the important comment that diving is not a sport. "Sport" is just the WRONG word. So what is the right word? .The closest we came up with was "discipline".

So I got to thinking about "safe" and "dangerous" and realised that most discussions are based on the conventional rules and limits taught by diving instructors, and not on an analysis of what is really going on. This is why instructors are generally poor educators, they are just "givers of directions or orders". If they knew anything about education they would be called "teachers" instead. So a typical instructor might make the statement "Dives deeper than 130 feet (39 m) are dangerous and should never be attempted by sports divers". Is this true of false? There is no answer as, in my opinion, the statement is meaningless (does it imply, for example, that dives to 127 feet (38 m) are safe?). Let us try to sort it out.

Diving involves RISK. As soon as you step near a full scuba cylinder you are at risk. What happens now is that every step that you take getting to, on and into the water increases that risk. What we do have is an escalating scale of risk as dives become more complex. The risk of a certain dive is a function of the technical requirements of the dive, the conditions, etc. IT HAS NOTHING TO DO WITH THE DIVER. In theory we should be able to grade every dive for its risk factor, but this is difficult in practice, although cave dives have been graded. Ocean conditions, being more variable, may make a dive low risk one day and high risk the next, so any system would have to be one that would produce a score on the spot. Nevertheless, an accurate assessment of the risk factor for any dive HAS TO BE MADE before the dive is attempted. This is why experience is so valuable.

The actual DANGER on any particular dive depends on two factors, possibly three. First, the dive itself - the risk factor; second, the diver attempting the dive - the skills available to overcome the risks; and third, the buddy the diver is diving with - the wild card. I've probably written too much about buddies lately, so I'll shut up about that now. So what is the art of safe diving? SAFE DIVING OCCURS WHEN THE SKILLS, EXPERIENCE AND KNOWLEDGE OF THE DIVER MATCHES OR EXCEEDS THE SKILL, EXPERIENCE AND KNOWLEDGE REQUIREMENTS OF THE DIVE.

Diving shallower than 30 feet (9 m) in calm, clear, warm water, devoid of any marine life is a low risk dive, but is dangerous if the diver does not understand the consequences of breath holding on ascent. Similarly a dive to 200 feet (60 m) in dark, cold water with a current is a high risk dive but one that can be safe IF the diver has the appropriate abilities. Professional divers make these kinds of dives all the time. It might be a crazy dive but is not stupid. Stupid divers are those that believe they have abilities that in fact they do not possess, or divers who do not understand, or care about, the risks of a particular dive. Pioneers are people who try to find out what the risks are of a particular activity because nobody really knows, obviously pioneer diving can be dangerous. Of course, if our pioneer diver is in fact repeating the errors of a previous pioneer but did not know this because his research was inadequate, then he is not a pioneer, merely stupid.

Nothing is absolute - a dangerous dive is one where it is LIKELY that an injury will occur; a safe dive one where it is UNLIKELY (but not impossible) than an injury will occur. A high risk dive is one which is deeper, longer, colder, rougher, involves penetration of a wreck or cave, has a current, involves dangerous marine animals, is difficult to enter or exit from, etc. My point is that a high risk dive need not be dangerous if the diver can identify the risk factors and overcome them with disciplined diver education and training. We must also realise that there is no such thing as a COMPLETELY safe dive. The reason for this is that, first, NOBODY knows all the physiological risks associated with diving; and second, that some marine phenomenon (and buddies) are unpredictable. A SAFE DIVER IS ONE WHO IS ABLE TO ASSESS THE RISK FACTOR OF A DIVE ACCURATELY AND HAS ACCURATE SELF KNOWLEDGE THAT HIS/HER ABILITY IS SUFFICIENT TO OVERCOME THESE RISKS. Isn't this fun, aren't you glad you're not sober?

Now the question needs to be asked: Why is it that we have so many stupid divers? Remember these are divers who imagine that their diving ability is far greater than it actually is. These divers are ALWAYS making dangerous dives, even though they dive within the "safe limits" promulgated by the instructor agencies (which demonstrates why most of the rules and limits are so lacking in intelligence. They do not make diving safer!). Well, I'm not sure about this, but my feelings are that the responsibility lies with instructors. First I am going to blame one particular instruction agency. I'm not going to name it because the purpose

of this exercise is not to start instructor agency warfare. This organisation issues an Advanced Openwater Certification to divers who have made as few as nine dives. These people believe they are advanced divers! One business which operates with the same agency advertises "Basic Diver to Instructor in Three Weeks". These new instructors would presumably think that they know enough about diving to pass on their ignorance to others. This agency is DANGEROUS. Two divers in Australia just certified as "Advanced" attempted a 140 foot dive to a wreck together. Only one returned and he wasn't feeling too good.

There should be more emphasis in diving courses on assessing risk factors for dives and personal critical assessment of one's dive skills, knowledge and experience. High standards need to be set for students to aspire to. Positive reinforcement is used by good instructors to increase the rate of learning but at the same time the successful basic student must be made to realise that vast improvements can still be made. They need a true assessment of their skills and knowledge. All too often newly qualified divers, proudly clutching their shining "C" cards believe that they really do know how to dive. I would like to see a probationary period of 100 dives established if for no other reason than to remind the diver that he or she has to CAUTIOUSLY gain experience before he or she is completely qualified. The development of speciality courses has been a very positive step in the right direction, but only a small proportion of divers take these courses. Perhaps the attendance at these courses could accelerate completion of the probationary period - this would encourage attendance. Most just go diving and believe that they are as skilled as anyone else. I remember the astonishment of instructor candidates, at the Instructor training courses (ITCs) that I directed, on being awarded fail scores on the performance of basic scuba skills when they assumed that getting through the exercise was sufficient.

Here is a very simple example of an exercise that could be tried during a basic course, that also involves practice of a VITAL skill that is never practiced enough during these courses (if you did not practice at least fifty times, it was not enough). Remove and replace weightbelt. The students should be on the bottom, in shallow water, towards the end of a dive and be told to remove the weight belt, hold it as if they were going to ditch it, and then replace it (you can try this too). Then score: did you look at the belt - you fail; did you change hands - fail; did you undo your BC belt - fail; did you thread the belt through the wrong slot on the buckle - fail; did the belt get caught or hung up - fail; did you drop the belt or a weight - fail; did you loose balance or float up - fail; did you stir up the bottom - fail; did you hold the belt by the buckle end - fail; did the belt end up twisted - fail; did you hold the belt completely clear of your body - if no, you fail. I know that different styles can be used, but the important thing here is to describe the skill the way you want it done and demand perfection in execution.

Stupid divers lack discipline, they go diving casually in the same way that they go to have a game of tennis or go bowling. It is a pity that diving has been promoted as a "sport for the family that everyone can enjoy". This is not true. To be a safe diver requires dedication to the study and practice, and mature, thoughtful preparation and execution. I also do NOT think that "diving is FUN". This is the wrong word too. These phrases were used to attract new recruits to the dive schools, but they are the wrong recruits too! Diving is about ADVENTURE, and NATURE, about EXPLORATION, and WILDERNESS, about EXCITEMENT and BEAUTY, and WATER and WEIGHTLESSNESS. It is not "fun". If I want fun I'll go to a party. I'd like to find whoever coined that phrase and jump on his mask!

DIVING IS ADVENTURE!

The consequences of these blunders are far reaching. Let us take the typical family out to have some fun, but let's not tell them that they are going to have to be disciplined and work to be proficient, that will put them off. Don't worry about the fact that half of them can barely swim, by the end of the course they will probably be able to struggle through the swim test. Stick regulators in their mouths and show them how to breathe, and hey you can do it, you are divers! (People who have not dived before imagine that it is hard to breathe underwater, and once they have taken their first few breaths they experience a massive boost in confidence and self esteem and will believe anything that the instructor tells them. Good instructors use this to lead their students through the more demanding skills). Pretty soon they have their cards and are in the Caribbean with a resort operator anxious not to get sued out of his business. His answer is to gear the dives to the lowest common denominator and appoint officious dive masters who regulate the divers' every move in the water. Any real divers who happen to be mixed up with this family are going to get a pathetic experience. If they are new to diving they will probably think that if this is what diving is all about they would rather do something else. This strategy neither attracts nor keeps the very people we need most in diving.

I am also alarmed by the appearance of mini cruise ships, "love boats", masquerading as dive boats. These vessels promote gambling, booze, sex, rock and roll (did you know that sex can lead to rock and roll?) and diving. This is madness, everyone knows that drinking and diving do not mix.

TELITA was built for disciplined hard core divers (we rent whips and leather wet suits). We know that there are many very competent sports divers who are becoming more and more frustrated by the restrictions placed on them at dive resorts, often by dive masters who know less than they do. We deliberately aim our standard at divers who want adventure, who want to see exotic marine life, who want to match their skills to the most exciting dives that we can provide, and who also know when to take a break and

let a dive go by. We want people who love the ocean as we do, with its infinite variety of moods and surprises. We are not dive masters or dive guides, though we are very happy to introduce new techniques to divers who have not had the appropriate experience. Attitude is often more important than experience. We do not avoid risk, but we do try very hard to prevent dangerous dives from being made by our clients. We do not order our clients around. Going diving is a great way to get away from ignorant bureaucratic authority, and you will not find it aboard Telita. We believe that with true adventures, divers discover FOR THEMSELVES. We provide opportunities for those with the spirit and ability to do just that.

This article first appeared in Telita Cruises Newsletter, November 1989.

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HIGH ALTITUDE FLIGHT HAZARDS AND DESIRABLE ACTION

R C Adkins

Case History

The owner of a Mooney Turbo 252 travels regularly between Perth and Sydney on business. The Mooney Turbo 252 is a single , turbo charged, piston engined aircraft. (Mooney is the maker's name) Normal cruising speed is 195 knots and the range 1,820 nautical miles at 27,000 feet (all figures approximate). He usually tries to improve on his inaugural point to point speed record (homologated (confirmed and approved as a record attempt) by the Federation Aeronautique Interationale (FAI) which is the oldest aviation body in the world, having been founded in 1905, which has its headquarters in Paris), by cruising at around 27,000 feet to take advantage of the strong Westerlies. At these altitudes he has adequate supplies of, and breathes, oxygen for the entire flight Because of the single pilot aspect, his wife insists that he take someone with him, so that there is a back up in the event of an oxygen problem, i.e.. kinked hose, bottle runout, etc., with the attendant hypoxia problems. Both breathe oxygen for all the time that they are above 10,000 feet.

On the flight in question, his co-pilot was Mooney rated with considerable private pilot experience over many years. She had been for a "run" in the decompression chamber at RAAF Pearce, so was familiar with the symptoms of hypoxia.

The flight departed Jandakot at 0720 WST, with a flight planned time of 7.19 hours for the 1792 nautical miles to Bankstown, cruising at 27,000 feet, and that altitude was reached in approximately thirty minutes.

After about one hour and fifty minutes on the cruise, the co-pilot began to feel an uncomfortably gaseous or bloated feeling in the abdominal area followed by nausea (having never been airsick in her life) and needed to use THE bag. After another one hour and thirty minutes she began to experience aches and pains in the arms, so took a couple of Panadols, and after a while felt the same ache and pain in her legs. She tried alleviating the problem by flexing the arms and legs as much as possible, given the restricted space available.

The pilot became concerned, and proposed going down, but the co-pilot did not want to negate his record attempt and insisted that he keep going, although she was not feeling very bright.

At around Griffith, and about six hours and forty minutes into the flight (the winds having gradually dropped off), the co-pilot began to come good, and then helped to "run" the descent, timing, working out speeds, and over Bankstown Air Traffic Control Tower, logging the time and calculating the overall record speed which was increased by 7 knots to 207 knots.

On landing, the crew deplaned in the parking area and the co-pilot promptly collapsed on the tarmac as her legs gave way under her. Time now was 1800 EST. The total time in the air had been eight hours and thirty minutes

Another woman pilot was there to meet them, and after propping up the co-pilot against a hangar door, went to the Flight Service Unit (FSU) for assistance, as they were nonplussed by her condition. The FS officer took action by ringing the Fire Station, knowing the staff were well versed in first aid, and they responded straight away. The co-pilot's condition had them baffled, but they made her as comfortable as possible where she was, covered her up in their big warm jackets and then contacted the paramedics at Bankstown Hospital. (It is interesting to note that by this time the co-pilot was professing to be alright and just wanted to go and lie down.)

The paramedics responded with their ambulance, examined her, and on seeing marked bruising on her arms, declared that she probably had the "bends", and took her off to the hospital.

By this time it was about 2000 hrs, and she was admitted to casualty. Over the next couple of hours she was examined, her low blood pressure and weak pulse noted and an intravenous drip started. Various calls were made to various people and departments, and eventually the decision was made to transfer her to Prince Henry Hospital, at Little Bay on the other side of Sydney, the diagnosis being suspected as the Bends.

Prince Henry Hospital has a Hyperbaric Unit for this and many other treatments, and after an hour's ride in the

ambulance with the same medics, she was admitted there around midnight.

More examinations followed admission to the ward. At about 0200 hrs she was trundled out on the stretcher again (it was now pelting with rain so that they had to pull the blankets over her head to stop her getting drenched) and driven to the Hyperbaric Unit whose staff had by now come on duty to handle the case.

There she was placed in the recompression chamber, breathing 100% oxygen and with an attendant sent "down" to 18 m (60 ft) of sea water, where they stayed for two hours.

After this treatment she was returned to the ward and rested until examined by two specialists later in the morning. They had by this time definitely diagnosed her as suffering from altitude decompression sickness (DCS) after testing her reflexes thoroughly, and then sent her for another session in the chamber similar to the last, at 1400 hrs. Co-incidentally, one of the above (a professor), had experience of aviation bends going back to the Meteor/Vampire days.

All this time she was on the drip, and about 2000 hrs, it was determined that her kidneys were not functioning due to dehydration. A specialist arrived, as he put it most succinctly, "to kick start your kidneys". This took about an hour and a half, by increasing the flow rate of the intravenous drip under pressure, and soon her system was back to normal.

Two more days in bed ensued, with many tests of reflexes and blood pressure and she was eventually released on the Thursday afternoon.

Discussion

Although the story may seem a bit long winded, it is an attempt to show things as they happened and the puzzlement of the various participants when confronted with an unknown situation in the air.

The writer has been engaged in civil aviation since 1948, including 33 years as an airline pilot, and had never heard of the bends in the air. Certainly he has flown pressurised aeroplanes for 26 years but apart from a "run" in the RAAF decompression chamber at Pearce Base (WA) in the early sixties, has never seen or read anything on the bends in civil flying. That "run" was to demonstrate the effects of hypoxia by removing the oxygen mask and noting the drop off in mental and physical capabilities. Even back then, flying DC3s for high altitude photography, at 25,000 feet, no mention was made of the possibilities of the bends.

And yet it appears from discussion since this occurrence, that there is quite a bit of high altitude flying taking place, such as survey/photography, etc., and a lot of people on the ground know of the problem.

Recently a parachutist set out to establish a new jump altitude record, but got the bends before leaving the aircraft. He was treated in the chamber, and later made another attempt with the same result, so abandoned the idea.

Further enquiries to Prince Henry have thrown up SPUMS, the South Pacific Underwater Medical Society, devoted to underwater medicine, whose April/June 1989 magazine issue has a six page article on "The Flying Bends", which traces the history of it back to 1934, including the fact that prior to 1959 over 17,000 cases of altitude DCS were reported, of which 743 were serious with seventeen fatalities.

Admittedly, a lot before and since were very high altitude military flights but also a lot were quite lower. It is a great pity this information is readily available, but not to the ones actively involved.

The problem occurs because nitrogen is dissolved in the blood. As the ambient pressure falls bubble formation occurs in the gas saturated body tissues causing joint aches and itching, tingling, and sometimes, choking and spinal cord damage.

So, how to prevent it; how to recognise it; and what to do:

1 Anybody can get it, but a lot do not. Increasing age is a factor, susceptibility increasing nine-fold between the ages of 18 and 28 years. And females are three times more susceptible than males.

2 High rate of climb is a factor. However, pre-breathing with adequate 100% oxygen before flight goes a long way to combat it.

3 Be alert to the onset of a gaseous feeling in the stomach, and aching joints, and itching, and do not move limbs in an attempt to relieve the discomfort. That only helps to increase the severity of the problem. Apparent "bruising" could be a pointer.

4 Descend at once. The longer the exposure, the worse it gets. Threshold altitude for onset is around 18,000 ft but may be as low as 10,000 ft.

5 Do not be in a hurry to land. Head for where you think the best help is available. Immediate treatment is necessary.

6 Recompression chambers are situated at Sydney, Melbourne's Alfred Hospital, HMAS STIRLING near Perth (and soon at Fremantle Hospital, WA), the Royal Adelaide and Royal Hobart Hospitals, Townsville and Darwin. So it looks like if you are

in trouble, head for a capital city.

7 Going flying after scuba diving is a no no. A dive to 15 m (50 ft) then a climb to 1,000 m (3,000 ft) can cause onset.

8 Do not be put off if the sufferer appears to recover and wants to be left alone - GET HELP.

In the case reported above, the pilot, without knowing it, did all the right things (as I see it, the victim being my wife). He offered to go down, but the co-pilot said no. Landing at, say Ceduna, would only have compounded the problem with further delays, before being flown out to one of the above locations. Although he did not descend, (which medically is best), this was probably a good thing, as he kept his tailwind, thereby increasing his range, and so not having to land short of the best help.

What a help to have somebody to meet you! The woman friend said she would meet her friend (the co-pilot), and even though they were an hour late from losing the winds, she was still there. They were tired after all their problems and the long flight, and somewhat confused by it all. She took over and set the help chain in motion, ensuring swift and adequate action was taken.

Overall, a frightening experience for all concerned, but hopefully now a lot more people will have some idea of what it is all about, and what to do.

After all, a lot of people on the ground appear to know about the "Flying Bends", why not the poor old pilots?

Last but not least, the Prince Henry Hospital Hyperbaric Unit treats, not only DCS, but gas embolism, carbon monoxide poisoning, cyanide poisoning, hydrogen sulphide poisoning, cerebral oedema, crush injuries, compromised skin grafts, non-healing wounds (skin ulcers are said to virtually heal before your eyes), radiation necrosis, soft tissue infections, osteomyelitis and gas gangrene. Does your doctor know that!

The article has appeared AOPA Magazine for October 1989 (Aircraft Owners and Pilots Association) on page 108. Co-incidentally, another article on the Bends, entitled "What a Fizzer" appeared on page 122. The address of AOPA is PO Box 1065, FISHWYCK A.C.T. 2609.
It is reprinted here with minor changes to make it more easily undedrstood by divers. We are grateful for Mr Adkins permission to reprint.

Mr Reg Adkin's address is 267 The Esplanade, Mount Pleasant, Western Australia 6153.

BUBBLE GROWTH IN BIOLOGICAL SYSTEMS

Brian Hills

Introduction

No diver likes to think that bubbles have formed in his body during any decompression, especially one which has proved asymptomatic. The same philosophy tends to apply to those who formulate the diving tables which are the ultimate product resulting from experience and research into the mechanisms of decompression sickness. Although the wording accompanying the presentation of methods for calculating decompression tables may sound plausible, it is *equations* and not words which are used in the computation and these may not necessarily say the same thing. Before discussing the fundamentals of bubble growth it is therefore desirable to identify the vital aspects which could affect the final product - the diving tables.

Spectrum of approaches to decompression formulation

Most calculation methods used today are still variations of the original Haldane theme whereby the mechanism leading to "the bends" is only triggered when some critical condition is violated. In most approaches this is a ratio[1] or 'M' value which may be a constant[2] or vary with depth[3]. In keeping within these "trigger points" for clinical manifestations, there is *mathematical symmetry.* In other words the same equations are used to estimate gas elimination as were selected to estimate gas uptake. Hence mathematical symmetry implies that no gas phase has formed during decompression because this would change the physics of the system and that must, in turn, be reflected in the mathematics. Hence, whether stated or not, most popular approaches to the formulation of decompression tables still follow the popular philosophy of assuming that "the bends-free diver must be bubble-free". This assumption tends to be obscured when it is emphasized how the ratio or "M" value can reflect the volume of gas which could be released from solution in returning to equilibrium from a state of supersaturation.[1,4] However this is a *potential* volume and not an *actual* volume, so the use of such "trigger points" still assumes that the bends-free diver is bubble-free.

The validity of mathematical symmetry was first questioned following some classical experiments by Hempleman[5,6] reviewed in detail later[7]; while this investigator went further and used the *actual* volume of gas 'dumped' from solution as the indication of the imminence of "bends"[8]. At least, it is based upon gas volume in so far as this parameter determines the pressure differential in tissue bending or otherwise distorting a nerve ending to induce the pain characteristic of Type I decompression sickness,[7,8] human experiments having shown a well defined threshold of injection pressure for pain induced by extravascular air[9]. Thus there is a wide spectrum of approaches to

the vital issue of the gas phase in the asymptomatic diver, ranging from *all gas remaining in solution* by supersaturation theories to the other extreme of phase equilibration, but why is this issue so important in formulating decompression schedules?

Why do we need to know if bubbles are present?

Conventional calculation methods based upon "trigger points" and mathematical symmetry are very popular because the computation is much easier if one can simply usef the solubility (S) to equate the mean inert-gas tension (P_t) to the mean inert-gas content of a tissue (SP_t). Taking the tension difference between arterial gas (P_a) and the mean tissue tension (P_t) as the driving force for transfer, one then has a simple linear equation:

$$P_a - P_t = kS(dP_t/dt)$$

most conducive to programming, even on home computers.

Whereas this equation, or something similar undoubtedly holds for gas uptake, it would break down badly *during decompression* if gas were separating from solution. Total tissue gas would now be the sum of two terms - one representing "dumped" gas and the other gas remaining in solution. However only the gas remaining in solution contributes to the driving force for elimination ($P_t - P_a$) and, hence, this vital parameter could be greatly over-estimated of loss of gas from solution were ignored in calculating P_t. This vital point is illustrated by the anology with water flowing out of tanks shown in Figure 1.

Effect of 'dumped' gas on decompression format

Many Doppler studies have shown circulating bubbles in asymptomatic decompressions since the introduction of this technique.[10] On the other hand, it can be argued that Doppler signals represent intravascular bubbles which may not be relevant to limb "bends" where the offending gas would appear to be extravascular because these symptoms are repeatedly pressure reversible. However many direct and indirect methods have demonstrated the presence of gas in tissue following sub-symptomatic decompression.[7,8,11,12]

The counter-argument to the major error implied by assuming mathematical symmetry is that, although some gas may be "dumped" as bubbles, most remains in a state of supersaturated solution. Even my own whole-body studies would support this view.[7,13] However it can also be argued that initiation of gas phase separation is random and that one should consider the "worst possible" zones where seeding is so profuse that all gas in excess of true saturation is "dumped" out of solution as bubbles or as less regular shapes which coalesce to form bubbles.

The net result of allowing for gas phase separation is to introduce much deeper initial stops than indicated by Haldanian calculation methods[7,8] and generally spend more time deeper. This general philosophy has been incorporated into commercial decompression tables and is strongly supported by the vast wealth of experience of human air diving (100-200 million air dives) distilled into methods used by the last of the pearl divers on the North coast of Australia.[14] These individuals would routinely decompress in one half to two thirds of the corresponding time needed by a US Navy diver, even before he takes the common practice of moving over one or two columns in the USN. tables.

Fundamental questions

While phase equilibration represents the "worst possible" driving force, the other aspect of gas transfer concerns the kinetics. Two decades ago, there were heated debates concerning whether the rate-limiting process for gas exchange was diffusion[8,15] or the blood perfusion rate.[2] However the direct observation of one bubble growing in a living tendon while another was simultaneously shrinking *in the same tissue* of a decompressed animal[16] led to the discovery that blood flow in a tendon bundle is intermittent[17]. The mathematical complexity of such a finding when combined with a non-linear driving force led this investigator to abandon the computation of decompression tables from fundamentals. Another factor in this decision was that, whereas one may derive a good model for limb "bends" as the predominant symptom, the serious residual injuries in divers were neurologic in origin.

Intravascular bubbles

Even if the gas causing limb "bends" is located in extravascular sites, there is no doubt that intravascular bubbles are undesirable. Should the quantities of gas found in the venous system upon asymptomatic decompression reach the brain, the diver would receive serious neurologic injury, if he survived at all. Hence the pulmonary circulation must play a major role as a bubble trap. However bubbles have been shown to escape entrapment, there being a very well defined volume rate of embolisation (0.35 ml/Kg.min. in dog) as the threshold for spill-over[18].

Since the volume rate of embolisation of the lung is a major parameter determining escape of emboli into the arterial system, the rate of bubble formation is therefore an important factor in avoiding cerebral decompression sickness. This, in turn, requires a close look at the growth of bubbles whether they are formed directly within vessels or enter the venous system by rupturing vessel walls[19].

Bubble formation

It is an interesting academic question as to whether there are micronuclei or macronuclei and what activates them into growth[20]. However, one feature of bubble formation is beyond dispute and this is the tendency for bubbles

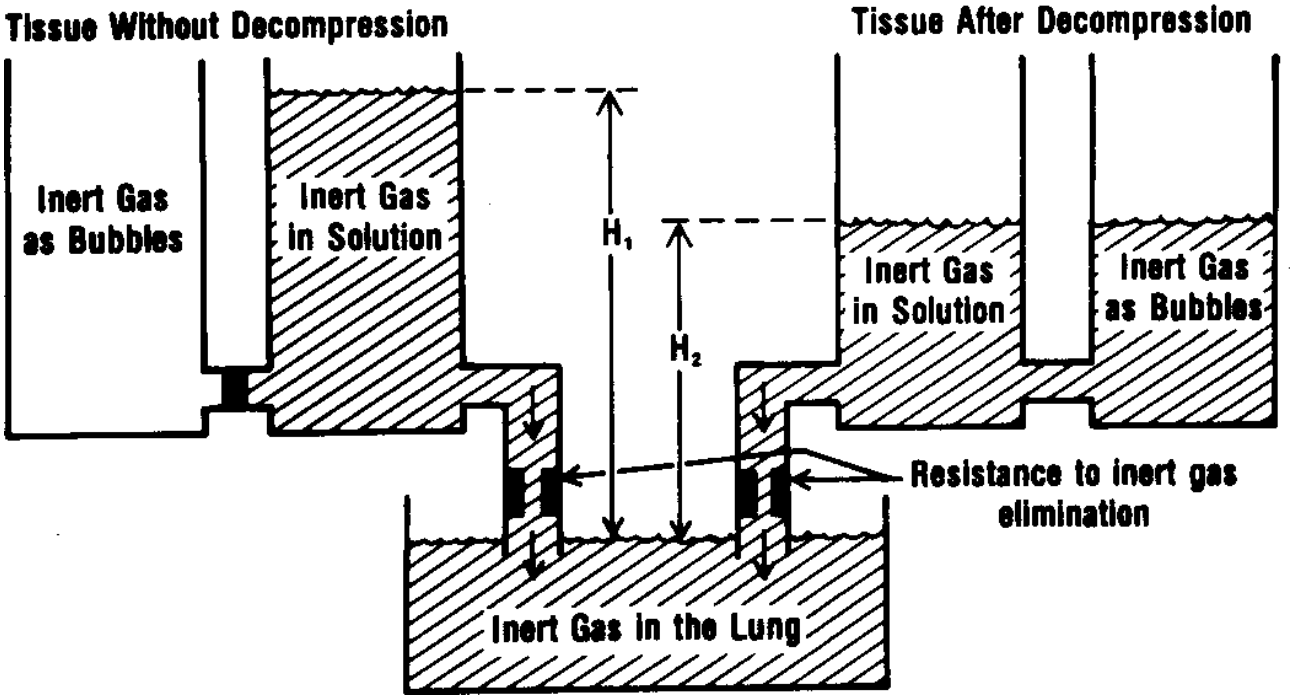

FIGURE 1

A hydraulic analogue emphasizing how the driving force for gas elimination from tissue can be appreciably reduced by formation of the gas phase. Tissue gas is simulated by water in the tanks. If gas remains in solution the head of water for its outflow is H_1 but, if the same gas is now distributed as both bubbles and as that remaining in solution, this is now equivalent to placing the same water into two interconnected tanks. The head for outflow (H_2) can now be much lower than H_1 and the elimination rate will be accordingly slower.

to form at an interface, usually where there is an imperfection. Moreover bubble formation is highly dependent upon the interfacial energy as exemplified by Harvey's simple experiment of immersing a candle in soda water when the very hydrophobic surface causes profuse bubbling[21]. This can be appreciated from considering basic thermodynamics where a hydrophobic surface has low surface energy in air but this becomes very high when the air is replaced by an aqueous liquid. Hence the overall energy is reduced when the gas phase forms to separate it from water, so the energetics are very conducive to bubble formation at hydrophobic surfaces. Interesting as this may be, Harvey largely dismisses such factors from decompression considerations on the basis that blood vessel walls are very hydrophilic. This attitude is also consistent with the conventional model for the structure of membranes which are present at almost all interfaces *in vivo* and present a vast matrix of surfaces in all tissues.

Membrane surfaces

The classical model of Singer and Nicholson[22] depicts a membrane as a lipid bilayer in which phospholipid molecules are packed together with their non-polar (fatty-acid) tails forming two rows closely apposed to each other in their desperate attempt to escape from water[23] and form a central hydrophobic domain. This orientates the polar moieties outwards to form hydrophilic surfaces(Figure 2b). Even the intercalated protein is believed to be folded with the hydrophobic zones inwards and the hydrophilic outwards, thus enhancing the wettable nature of membrane surfaces.

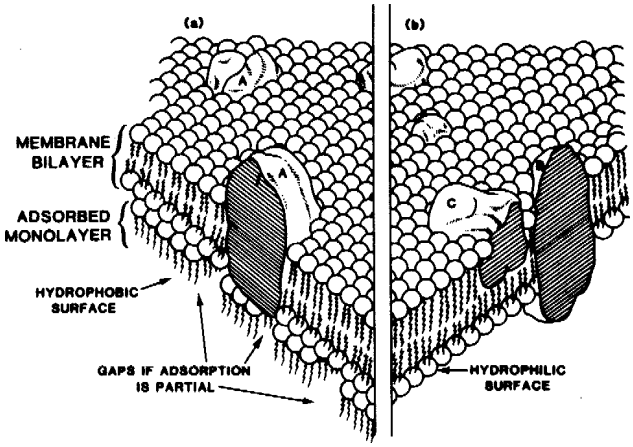

FIGURE 2

Depicting (b) the popular model of a membrane as a lipid bilayer with intercalated protein (after Singer and Nicholson[22]) the polar (wettable) ends orientated outwards. It is proposed that surface-active phospholipid is adsorbed as shown in (a) to render the surface hydrophobic (from Hills[25].)

Hydrophobic surfaces

Most tissue surfaces do indeed prove wettable when tested but, in many cases, this can be attributed to a mucoid layer which may be adhering or incorporated as a glycocalyx[24]. However, when certain surfaces are well rinsed to remove adhering mucus, many membranes prove to be hydrophobic[25].

Hydrophobicity can be most easily demonstrated by the tendency for water (or saline) to "bead up" when placed on the surface. This leads to a contact angle between the solid surface and the tangent to the air-liquid interface at the triple point where all three meet - solid, liquid and air (Figure 3). The contact angle (q) can range from 0 for a perfectly wettable surface up to 180° with such very hydrophobic surfaces as candle wax, polyethylene and Teflon having contact angles of 106°, 94° and 108° respectively[26].

In view of traditional theory, it was therefore surprising to record values of up to 105° on gastric mucosa[27], 103° on articular cartilage[25], and up to 153° on the amnionic surface[28]. The latter is shown in Figure 4. These surfaces

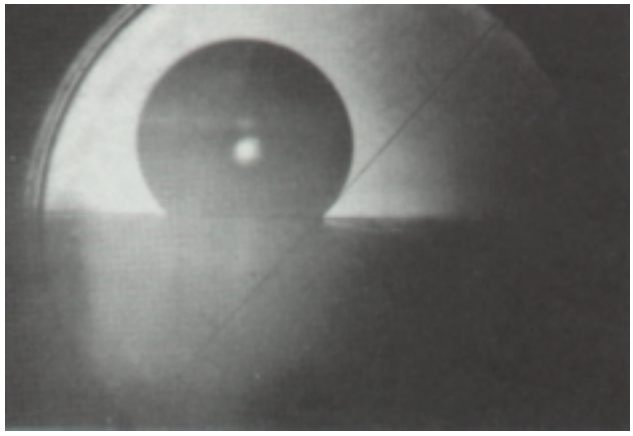

FIGURE 4

A contact angle measured on the rinsed human amnionic epithelium after placing a small drop of saline on the surface. This demonstrates just how hydrophobic some membrane surfaces can be[22] despite the hydrophilic nature assumed in the popular model depicted in Figure 2b.

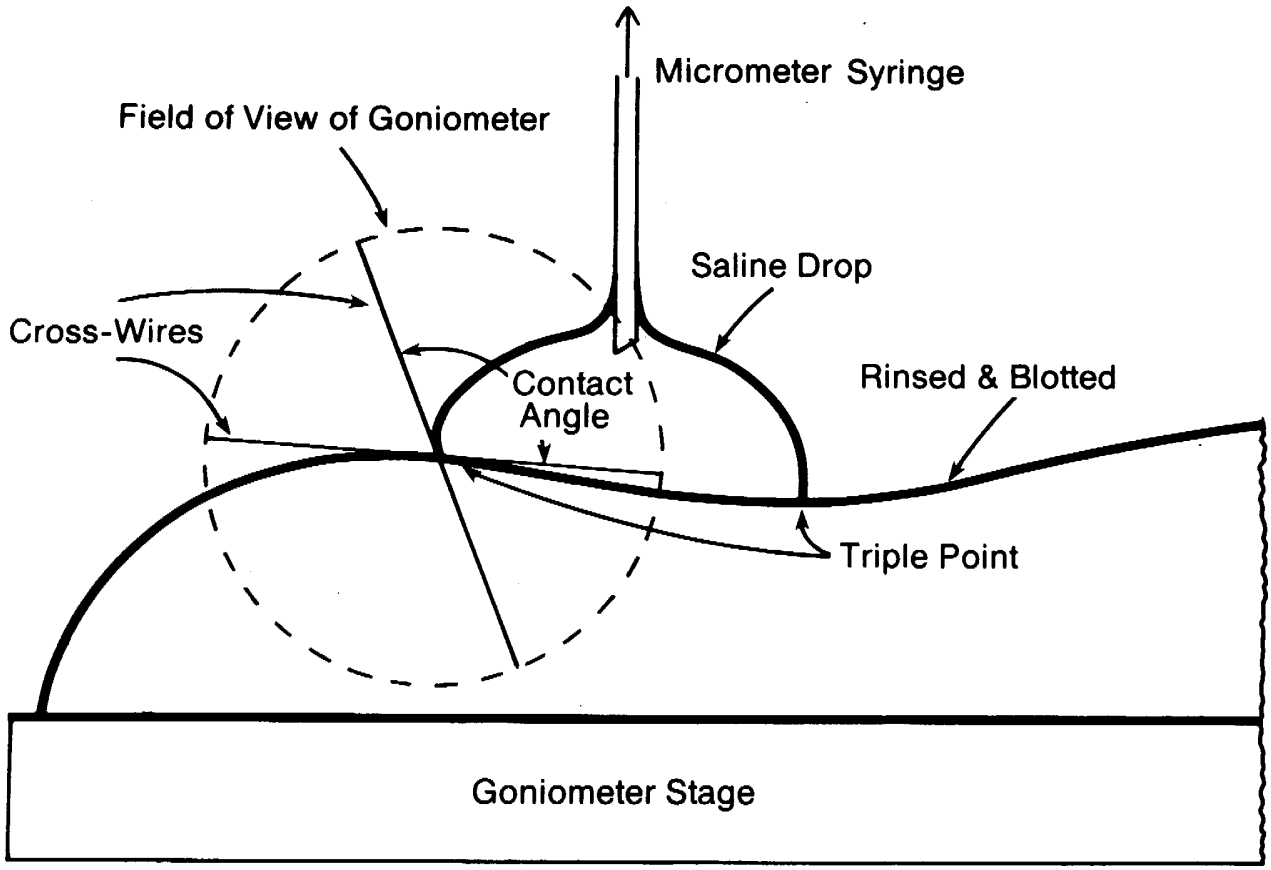

FIGURE 3

Defining the contact angle and showing its method of measurement by a goniometer. The contact angle is a very simple and convenient index for quantifying the hydrophobicity of a surface.

and others were studied for the highly desirable properties which can be imparted by an adsorbed layer of surface-active phospholipid, including gastric mucosal protection, joint lubrication and water repllency in preventing alveolar flooding[25]. These are all well established applications of surfactants in the physical sciences[26] where one of the tests of adsorbed surfactant is the hydrophobic nature imparted to the surface. The standard theory for the total change in "personality" of a surface by adsorption of a surfactant is, perhaps, best explained by referring to Figure 4 where it can be seen that the outwardly orientated fatty-acid chains can pack together with those from molecules adsorbed to neighbouring sites to form a thin hydrocarbon coating not unlike candle wax or polyethylene. This is particularly interesting in view of the ease of formation of bubbles on those surfaces in their relevance to extravascular sites, but what about intravascular surfaces?

The vascular lumen

Lawns of human endothelial cells can be cultured from umbilical cords by standard methods but it was particularly surprising to find that they were appreciably hydrophobic with contact angles ranging from 50°-70°[25]. A similar range of values was obtained after well rinsing the original cords before removing endothelial cells.

Morphological studies

It might well be asked why anything as obvious as a third layer (Figure 2) has not been sen in electron micrographs. A review of the subject[25], has indicated that third lines were reported until Sabbatini et. al.[29] introduced glutaraldehyde as the universal fixative for electron microscopy, aldehydes having the effect of "destroying hydrophobic surfaces"[30]. When tannic acid is substituted for much of the aldehyde in lung fixation, three or more lines are routinely seen for the alveolar membrane and there is some indication of similar adsorption to pulmonary endothelium[31]. Hence there is a reasonably plausible explanation for a moderately hydrophobic luminal lining to blood vessels.

Implications

There are three major implications to a hydrophobic luminal lining. Firstly, such surfaces are highly conducive to bubble formation as discussed already, thus reversing Harvey's reasons for dismissing vascular walls in this context. At least, any of these surfaces are only hydrophobic if rinsed free of any mucoid substances found to be particularly effective wetting agents in attaching to hydrophobic surfaces, reducing their otherwise high interfacial energy with aqueous fluids and thus stablising adsorption[32]. In this regard it is tempting to speculate an interesting role for heparin and other potential wetting agents in controlling the net hydrophobicity and, hence, the bubble-forming potential of vessel walls exposed to supersaturated venous blood.

The second implication of a hydrophobic lining is that it will impart a collapsing pressure, especially to smaller blood vessels and might well contribute to the difficulty in re-perfusing vascular beds after circulatory collapse. In this context it could provide the mechanical tension characteristic of the cerebral circulation where autoregulation is lost at a particular level of hypertension[33]. It could be particularly significant that this point coincides with breakdown of the blood-brain barrier[33] since another effect of adsorbed surfactant is to reduce permeability.

The notion that an adsorbed layer of phospholipid might provide the blood-brain barrier offers an interesting explanation for its breakdown by bubbles[34] or other circulating emboli whose interfaces with blood would compete for the surfactant adsorbed to the cerebral vascular wall[25]. It could also offer an explanation for the similar symptomatology between chronic decompression sickness and multiple sclerosis and the fat embolism theory as revised by James[35]. Moreover, the concept of emboli acting as solvents or interfaces competing for surfactant rather than acting as an infarcting agent now makes this theory much more compatible with the pathological findings[25].

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This paper was presented at the International Symposium on Supersaturation and Bubble Formation in Fluids and Organisms, held at Kongsvoll by the Royal Norwegian Society of Sciences and Letters, The Foundation, from 6th to 10th June 1988.

The proceedings of the symposium, from which the above has been taken, with necessary alterations for the different reference style of this journal, have been published by Tapir Publishers of Trondheim, Norway.

We are grateful to he Royal Norwegian Society of Sciences and Letters, The Foundation, and to Professor Brian Hills for permission to reprint.

GLEANINGS FROM MEDICAL JOURNALS

The following articles have come to the notice of the editorial staff and these notres are printed to bring them to the attention of members of SPUMS. They are listed under various headings of interest to divers. Any reader who comes across an interesting article is requested to forward the reference to the Journal for inclusion in this column.

HYPERBARIC OXYGEN FOR CARBON MONOXIDE POISONING

Trial of Normobaric and Hyperbaric Oxygen for Acute Carbon Monoxide Intoxication

Raphael Jean-Claude[1], Elkarrat David[1], Jars-Guincestre Marie-Claude[1], Chastang Claude[2], Chasles Valerie[1] and Gajdos Philippe[1]

Lancet 1989; ii: 414-419.

Summary

The value of hyperbaric oxygen in the treatment of acute carbon monoxide intoxication was assessed in 629 adults who had been poisoned at home in the 12h before admission to hospital. In patients without initial impairment of consciousness (group A) the effect of 6h of normobaric oxygen (NBO) (group AO, n = 170) was compared with that of 2h of hyperbaric oxgyen (HBO) at 2 atmospheres absolute (ATA) plus 4 h NBO (group A1, n = 173). At the 1 month follow-up 66% of A0 and 68% of A1 patients had recovered. In patients with initial impairment of consciousness the effect of one session of HBO (group B1, n = 145) was compared with that of two sessions (group B2, n = 141); all group B patients also received 4h of NBO. At 1 month of follow-up 54% group B1 and 52% group B2 patients had recovered. The 7 patients left with neuropsychiatric sequelae (3 B1, 4 B2) and the 4 who died (2 B1, 2 B2) had all presented with coma. HBO was not useful in patients who did not lose consciousness during carbon monoxide intoxication, irrespective of their carboxyhaemoglobin level, nor were two sessions of HBO in patients who sustained only a brief loss of consciousness. The prognosis is poorest for those presenting with coma; the trial needs to be pursued in this group of patients until the power of the study is sufficient to demonstrate the value or otherwise of HBO.

From

1 Service de Reanimation Medicale, Hopital Raymond Poincare, 92380 Garches, France

2 Department de Biostatistique et Informatique Medicale, Hopital Saint Louis, Paris

Hyperbaric and normobaric oxygen in acute carbon monoxide poisoning

James PB.

Lancet 1989; ii: 779-800

Letter

Hyperbaric oxygen for carbon monoxide poisoning
Brown SD and Piantadosi CA.
Lancet. 1989; ii: 1032.
Letter in favour of HBO treatment
From
F.G. Hall Hypo-Hyperbaric Center and Department of Medicine, Duke University Medical Center, Durham, North Carolina 27710, USA.

Hyperbaric oxygen for carbon monoxide poisoning
Gorman Des F., Gilligan JE Fred and Clayton David G.
Lancet. 1989; ii: 1032.
Letter. in favour of HBO treatment
From
Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, Adelaide, South Australia 5000.

Hyperbaric oxygen for carbon monoxide poisoning
Neubauer Richard A[1] and Gottlieb Sheldon F[2].
Lancet. 1989; ii: 1032 - 1033
Letter. in favour of HBO treatment
From
1 Ocean Hyperbaric Center, Lauderdale-by-the Sea, Florida, USA
2 Department of Biological Sciences, University of South Alabama, USA.

Hyperbaric oxygen for carbon monoxide poisoning
Raphael Jean-Claude[1], Elkharrat David[1], Jars-Guincestre Marie-Claude[1], Chastang Claude[2], Chasles Valerie[1], Vercken Jean-Baptiste, and Gajdos Philippe[1].
Lancet. 1989; ii: 1033.
Letter of reply.
From
1 Intensive Care Service, Hopital Raymond Poincare, 92380 Garches, France.
2 Department of Biostatistics and Medical Informatics, Hopital Saint Louis, Paris, France.

Hyperbaric oxygen for carbon monoxide poisoning
Broome JR1, Sykes JJW[1], Francis TJR[1], Tighe SQM[2], Edmondstone WM[2] and Clark RJ.
Lancet 1989; ii: 1529.
Letter. in favour of HBO treatment
From
1 Undersea Medicine Division, Institute of Naval Medicine, Alverstoke, Hampshire, UK.
2 Departments of Anaesthesia and Medicine, Royal Naval Hospital Haslar, Gosport, Hampshire, UK.

RETINAL VESSEL CONSTRICTION UNDER HYPERBARIC CONDITIONS

Retinal Vessel Constriction under Hyperbaric Conditions
Polkinghorne P J, Bird A C and Cross M R.
Lancet 1989; 2: 1099.
This letter is reprinted in full by kind permission of the Editor of THE LANCET.

Certain forms of decompression sickness in deep-sea divers are thought to be due to vascular occlusion caused by bubbles in the systemic circulation. The potential for bubbles to obstruct flow may be compounded by changes in other rheological indices. Constriction of the retinal vessels and consequent reduction of blood flow have been recorded in response to increased blood oxygen tension induced by breathing 100% oxygen,[1] and a similar response under hyperbaric conditions has been suggested.[2]

To verify this, fluorescein angiography of the ocular fundus was undertaken at five atmospheres in a hyperbaric chamber (simulating a dive to 40 m) on two healthy volunteers. These were compared with angiograms on the same individuals at one atmosphere. The width of the fluorescein column within the arterioles and venules was measured, with callipers on high contrast prints, at six identical locations during the arteriovenous phase of the studies. Under hyperbaric conditions there was constriction of both arterioles and veins of 15% in one subject and 10% in the other. These calibre changes are comparable to those recorded during ventilation with 100% oxygen;[3] the partial pressure of oxygen would be similar in both circumstances.

Narrowing of the retinal vessels under hyperbaric conditions may render the retinal microcirculation especially vulnerable to occlusion during deep-sea diving. This response may be relevant to the pathogenesis of retinal vessel changes recently recorded in a diving population. Similar alterations may occur in the cerebral vasculature since the two circulations react in a comparable way to change sin partial pressures of oxygen in blood. Alteration of flow and vessel calibre may have a role in the genesis of the central nervous system lesions.[4]

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From

Department of Clinical Ophthalmology, Institute of Ophthalmology, University of London, London EC1V 2PD and Diving Diseases Research Centre, Fort Bovisand, Plymouth, United Kingdom.

TREATMENT OF *CHIRONEX* STINGS

First aid

Retrieve the victim from the water if necessary (children) or restrain if necessary (adults).

Send others for an ambulance/medical help and antivenom.

Assess the conscious state and treat airway, breathing and circulation if necessary.

Liberally pour vinegar over the stung area for a minimum of 30 seconds to inactivate remaining stinging cells on any adherent tentacles.

Apply compression bandages to major stings (one covering an area greater than 50% of one limb or one causing impairment of conscious state).

Remain with the victim, treating with cardiopulmonary resuscitation if necessary.

Hospital treatment

Continue treating airway, breathing and circulation if necessary. Add oxygen if available.

Secure an intravenous line with a crystalloid solution running, and administer a minimum of one ampoule of antivenom (20 000 units slowly by the intravenous route) if none has been given. Up to three ampoules may be used if the sting is severe, or if the response to a lesser amount of antivenom is not sufficient clinically.

Monitor breathing and circulation, and give verapamil (5 mg) by the intravenous route for any cardiac abnormality (arrhythmia and/or hypotension) which persists in spite of antivenom therapy.

Intravenously administered analgesia (1-2 mg/kg of pethidine [50 mg for an adult patient] whenever necessary in conscious patients.

This advice appeared in The Medical Journal of Australia, 1989; 151 (Dec 4-18): 709.

For further details refer to

Williamson JAH. The marine stinger book. 3rd edn. Brisbane: Queensland State Centre, Surf Lifesaving Association of Australia, 1985: 27.

COURSES AND CONFERENCES

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Members wishing to present papers should contact the organiser of the Scientific Meeting.

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Royal Adelaide Hospital,
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South Australia 5000.
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JOINT MEETING ON DIVING AND HYPERBARIC MEDICINE ASSOCIATED COURSES

Dr David H. Elliott is preparing a modular post-course, meeting the training recommendations of DMAC and NATA on the Medical Aspects of Diving Accidents and Illnesses. The plans are as follows:

Compressed Air Diving. A practical course in diving medicine for doctors, at Den Helder with the Royal Netherlands Navy, 6-9 August 1990. Cost N fl 1,705 before May 1990.

The Medical Management of decompression illnesses. An advanced seminar and review, at the Hotel Okura Amsterdam, 10-11 August 1990. Cost N fl 465 before May 1990.

Mixed Gas Diving. An advanced course for doctors at the Cattedra di Medicina Subaquea ed Iperbarica and with the co-operation of the Italian Navy, 20-24 August 1990. Cost N fl 1,550 before May 1990.

For additional information, please contact
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