SPUMS JOURNAL
South Pacific Underwater Medicine Society
JULY TO SEPTEMBER 1982

CONTENTS

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EDITORIAL

Decompression sickness is one of those provoking conditions where a simple, satisfactory, rational scenario of cause and effect has been totally disrupted by the insensitive intrusion of facts. It was a condition having a special aura, an austere morality of retribution which fell inevitably onto the heads (or rather the joints and spinal cords) of all offenders against the Holy Writ of the Dive Tables. There were, it is true, a number of competing Dive Tables but each was to be regarded as infallible. Alas, Life, as ever, failed to obey such rigid limitations and there is now a certain permissiveness noticeable in the therapeutic management routines described by operators of recompression chambers. Naturally an informed permissiveness, with an awareness of the risks should results be poor and litigation occur. So there are no apologies for the seeming concentration of this issue on various aspects of the decompression sickness problem.

There is much to be learnt from an examination of the heroic saga of Tony Liddicoat's fight to save Dick Alba, not the least important being the reminder that one should NEVER take chances on decompression requirements and NEVER, NEVER dive without a back-up plan for disaster. This is particularly true if one is far from an adequately informed and equipped medical facility. The victim's actual dive times are unknown but it is quite obvious that the resort diving firm could not recognise, let alone treat, a severe case of decompression sickness. The medical back-up was a non-event also. In these circumstances Tony Liddicoat took the logical decision to brave a storm in an inflatable to attempt in-water treatment. Unlike SPUMS members, he was unaware of the value of in-water oxygen therapy and the poor track record of in-water air treatments: had he been aware of the risks of hypothermia to a pyjama clad person immersed for nearly two hours, and the risks of the victim drowning, he might have regarded the storm as the least of the adverse factors. The victim must be a remarkable man indeed, for few would remain calm if tied to a paddle, given a scuba regulator and lowered in a dark stormy sea. He was semi-conscious but aware of the gravity of his condition. The total lack of medical know-how is underlined by the lack of oxygen use in the management after this treatment, though time allowed contacting informed sources. It would have been better to have avoided the second in-water air treatment unless using oxygen. The case illustrates the need for instructors to be well informed about diving medicine because nobody else present may have the ghost of a clue about the victim's problem. A truly remarkable effort by those involved.

Diving accidents and misadventures seem to fall into one of two groups, the richly deserved and the highly unjust. The above "spinal hit" seems to have been in the latter group, but divers who take drugs are most certainly in the former. It is for readers to decide in which they would place the hyperthermia victim reported by Dr Ian Calder: death is a high price to pay for neglecting to drink fluids while assisting the treatment of someone else. Only one fatality involving chamber-related hyperthermia has been recognised in Australian waters, possibly a reflection of the fact that chambers are few and are controlled by the RAN or the big International diving organisations. One is enough.

What constitutes unfitness to dive will always include borderline problems, for Nature has few absolutes and many gradations. The project here advertised, the investigation of divers and would-be divers with any hint of "asthma" in their history, is welcomed. It is hoped that many will join this effort to define the permissible parameters for safe diving. It is not enough to rely on theory when facts are far more suitable backing for decisions. Case reports, about "asthmatics" who dive safely and those who certainly do not, are urgently requested.

Contributions are from the USA, the UK, New Zealand and Australia in this issue, indicative of the catholic nature of Diving Medicine. In the next issue it is hoped to have reports from Canada and Australia which indicate the avoidable factors in recent diving fatalities, the common lesson being that diving. Morbidity and mortality are reducible through the application of present standard advice. Dive correctly and you will be here to read all about it!

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PRESIDENT'S ADDRESS TO THE 1982 ANNUAL GENERAL MEETING

It gives me great pleasure to welcome Dr Fred Bove and his wife Sandra, to the 10th Scientific Conference of SPUMS. Our guest lecturer is a cardiologist, a diving instructor, a diving medical officer USN, a research worker both in cardiology and underwater medicine, an educator, being chairman of the Education Committee of the Undersea Medical Society and is now the President Elect of UMS.

This is a very special SPUMS meeting as we have present all three people who have been president of the society and three of the people who have been secretary. It is a great pleasure to see Carl Edmonds and Ian Unsworth here and to thank the foundation President and foundation Secretary for their midwifery. Their infant is growing steadily. A year ago I posted 415 journals, this June, if I had not delivered yours, I would have posted 496.

We have tried to produce an interesting and enjoyable programme. As usual it relies heavily on our visiting speaker - Dr Fred Bove. We are grateful to Dr Bruce Bassett for offering three papers. This year we have a larger than usual Australian contribution which is an excellent augury for the future.

You will have noticed that with the programme and the latest Journal you were given two Diving Medical history examination forms. We hope that you will study them, compare and contrast, and consider amendments to them. And then be vocal on the topic at the last session when we will be discussing "What should we ask for in a Sports Diver Medical?"

SPUMS is now associated with the Undersea Medical Society. Chris Lourey has been the architect of this advance and will tell you more about it.

The College of Occupational Medicine has been incorporated with an interim council. Negotiations are proceeding with the education steering committee about a qualification in underwater and occupational medicine. The correspondence is on pages 4 to 7 of the April-June Journal. It is likely that to become an Associate of the College of Occupational Medicine (Underwater Medicine) one will have to do the ten week Occupational Medicine course as well as the four week course at the School of Underwater Medicine. The position of holders of the SPUMS diploma is uncertain. However, the College council is more in tune with reality than is the education committee which is insisting that only fulltimers may become Fellows of the College. Apparently the College is going to start with a few fellows, many members and will acquire associates by examination.

Victor Brand has carried through the production of the First Aid poster for hospitals and ambulance services. He has some here to show us. We will also send them to dive shops and instructor organizations.

Once again the willing horses have been unopposed for election. While this may be the easy way to run the Society, at least until we run out of willing horses, the Society would be better served if more members offered their services. The key job is Secretary which needs about three to four hours a week. The President and Treasurer have bursts of activity which average out at less than an hour a week. The Editor and layout man (myself) spend a lot of time on the Journal. The committee members spend two or three hours attending a meeting two or three times a year.

Anyone can organize a meeting. All you have to do is get hold of speakers and a hall and advertise. So let us see if we can make SPUMS a more lively society in the year before we hold the next scientific conference at the Regent of Fiji in June next year, with Professor Brian Hills as our guest speaker.

SPUMS MELBOURNE MEETING

20th November, 1982

FOR DETAILS PLEASE TURN THE PAGE

NOTICE

Diving and Scientific Meeting organised by NUMBS, sub-branch of SPUMS.

DATE: Saturday 30th April, 1983 and Sunday 1st May, 1983.

PLACE: Hamilton, New South Wales, Australia.

DIVING: Tanks and belts available. Slack tide 10.28 am 30th April 1983. Halifax Park. Slack tide 11.11 am 1st May, 1983 Fly Point. Easy entries and exits. Maximum depth 70 feet. Good visibility. Big tame groupers to feed, corals, sponges.

MEETING:

THEME: Marine Venomous Critters, possible speakers: Neville Coleman, Dr Straun Sutherland, SPUMS members.

FILM: By Roche Institute of Marine Research.

SOCIAL ACTIVITIES: Saturday - RSL Club Style Dinner
Sunday - BBQ and Wine Tasting.

ACCOMMODATION: Some available.

CONTACT: Dr Brian McLaughlin,
PO Box 157,
Hamilton NSW 2303
Australia
Telephone: 61-5004

WANTED: INTERESTED SPUMS MEMBERS TO ATTEND/DIVE/SPEAK/PARTICIPATE

YOU ARE WANTED

SATURDAY NOVEMBER 20TH AT 2PM
SPUMS MELBOURNE MEETING

LECTURE THEATRE, ROYAL VICTORIAN EYE AND EAR HOSPITAL, 426 ALBERT STREET, EAST MELBOURNE.

Dr Geoff Macfarlane Dr Peter Laverick	Two cases of Spinal Decompression Sickness
Dr D Hackman	A Case of Air Embolism in a Diver
Dr John Knight	1st Aid for Diving Accidents
Dr Harry Oxer	Western Australian Diving Accident Network

8TH SYMPOSIUM ON UNDERWATER PHYSIOLOGY

TO BE HELD AT
GRAY ROCKS INN
ST JOVITE
PROVINCE OF QUEBEC
CANADA
from JUNE 15th to JUNE 19th 1983

Registration fees:

UMS and SPUMS members US$130.00 before 1.4.83, US$140.00 at the Symposium.

Accommodation including three meals a day will be less than US$70.00 per person per day.

Topics for the Symposium:

1. Inert gas exchange, counterdiffusion and bubble formation.
2. Interactions of ventilatory control, hyperoxia and increased gas density at high ambient pressure.
3. Oxygen toxicity.
4. Circulatory and haematological effects of hyperbaric pressure.
5. Molecular and cellular effects of pressure.
6. Thermal effects of the hyperbaric environment.
7. Behaviour and performance under hyperbaric conditions.
8. Decompression sickness, barotrauma and osteonecrosis.
9. Applied hyperbaric physiology and clinical science.

For further information write to:

8th Symposium on Underwater Physiology
Undersea Medical Society Inc.
9650 Rockville Pike
Bethesda, Maryland 20814
USA

SPUMS 1983 ANNUAL SCIENTIFIC MEETING

PLACE: THE REGENT OF FIJI HOTEL
DATES: June 20th to 27th 1983

GUEST SPEAKER: PROFESSOR BRIAN HILLS

DIVING BEFORE, DURING AND AFTER

A BROCHURE WILL BE POSTED IN OCTOBER

CONFERENCE ORGANISER:

DR JOHN KNIGHT,
80 WELLINGTON PARADE
EAST MELBOURNE VIC 3002
AUSTRALIA.

PRELIMINARY NOTICE

It is intended to hold a SPUMS conference in Rockhampton in September/October 1983.

The theme will be those aspects of diving and Hyperbaric Medicine that have an intensive care aspect. These include:

Drowning
Hyper therapy
Hypothermia
Marine animal envenomation and treatment
Evacuation and resuscitation of diving accidents
Hyperbaric oxygen and spinal artery thrombosis

The organiser is:

Dr CJ Acott,
Rockhampton Base Hospital,
Canning Street,
ROCKHAMPTON QLD 4700

Anyone interested in contributing a paper or in attending (there will be diving) is requested to contact Dr Acott.

A YEN FOR INTELLIGENT RESPONSE

Life is said to imitate fiction, and the predicament of the Sorcerer's Apprentice caught up with a citizen of Tokyo recently. He simply desired to obtain a bowl of hot noodles and inserted the necessary coin into a vending machine. The machine obliged, and continued to supply bowl after bowl. He was unsuccessful in his attempts to stop this largesse of steaming noodles, and a policeman he summoned was no more successful. The manufacturer's agent, summoned from his warm bed, brought the matter to an abrupt conclusion by simply pulling out the power plug.

Divers, who are certainly no noodles, will immediately note the moral of this tale. Trouble is most readily managed and minimised by those trained to understand what problems they may meet.

TONY LIDDICOAT DIVER OF THE YEAR MANAGEMENT OF A CASE OF SEVERE SPINAL DECOMPRESSION SICKNESS UNDER SEVERELY ADVERSE CIRCUMSTANCES

Kendall McDonald

Tony Liddicoat was deactivating an army scuba-diving Adventure School when informed that a helicopter was on its way with a seriously ill diver with symptoms of spinal decompression sickness. A storm was about to break and the nearest recompression chamber was about 1,400 km distant "as the crow flies". He had one experienced companion, a Gemini inflatable, a number of scuba tanks and a British Joint Services Diving Manual. The action he took, in the absence of desired facilities, are described.

The date: Wednesday, August 5, 1981. The place: Chapel Cay, off the coast of Belize. The weather: the still that comes before a tropical storm ...

Dick Alba, the 53-year old proprietor of Dyna-Sea Inc., a commercial diving company of Harvey, Louisiana, USA, is finning slowly upward along the wall of the reef. He is taking part in his favourite holiday diving occupation - he is filming fish.

This is his second dive of the day with a resort diving firm. His first dive was to 20m. On his second he touched bottom at 24m. He is experienced enough to note both depths carefully. He is, however, about to suffer a massive attack of the bends.

Within a short time of surfacing he is paralysed down his left side and completely immobile below the waist. He can only burble words and to his horror he hears what he knows to be the bends diagnosed as a heart attack.

Plans are made to airlift him to the nearest hospital, the British Army Hospital in Belize. He is given the last rites by a Catholic priest ...

The same day 15 miles away in the shallow water near the British Army Adventure School on St George's Cay, another island off the coast of Belize, Staff Sergeant Tony Liddicoat does two 10m dives instructing beginner divers. When that diving is over, many of the empty cylinders have their pillar valves removed. The Adventure School is closing down for a week's maintenance.

At six pm Tony Liddicoat feels he has earned a beer and starts walking towards the only little bar there is on the island. The palm leaves are rustling ominously as the dark clouds out at sea, outlined against the sunset, begin to deliver their wind. A storm is coming.

Dick Alba, in the British Army Hospital, is worse. He is now 75% paralysed. The British doctors have discarded the heart attack theory. They diagnosed a spinal bend but there are no recompression facilities at the hospital.

There is only one chance for Dick Alba and that lies in a 15-minute helicopter ride away at the Adventure Training School. His chance is Staff Sergeant Tony Liddicoat. The radio crackles and a breathless squaddie stops the Staff Sergeant on the way to his beer.

Tony Liddicoat now had an equipment problem. While they waited for the helicopter, he assembled what full cylinders they had, found that only one Gemini inflatable and engine were serviceable, and grabbed the most experienced diver he could find to help him.

The most experienced man on the island, next to Tony, was Vic Butcher, a 36-year old Army Physical Training Corps Warrant Officer. He had just passed his BS-AC Third Class.

Dick Alba, as he never stops saying today, had at that moment found a piece of luck. Because 33-year old Tony Liddicoat was not going to hesitate about doing what he knew had to be done.

Tony Liddicoat is one of the Army's most experienced divers with over 2,000 logged dives. A senior diving instructor with the Royal Engineers, he has been diving professionally for 17 years and instructing on all aspects of commercial diving for 12.

He made his first dive in 1965 as a boy soldier in Dover Harbour. He'd been a diving instructor at the Kiel Diving School in North Germany. In 1974, he'd been a guest of Jacques Cousteau on an expedition to the "Blue Hole" off Belize, and had dived the Blue Holes many times.

He'd played a big part in running Army sub-aqua clubs. Underwater photography is his hobby. He likes diving enough to do it for pleasure even though it is his work. He'd taken any diving experience where he could find it. He'd served in the Outer Hebrides, blasting deepwater channels for trawlers and constructing pipelines.

In 1977 he was off to Mombasa as part of the Joint Services Expedition to excavate a wrecked galleon. In 1979 he went back to the wreck again with the Royal Engineers Diving Team.

And now, in 1981, he had been seconded to the Adventure Training centre on St George's Cay as diving supervisor and instructor. He'd been trained as a chamber operator and had picked up even more therapeutic decompression knowledge in the North Sea.

His experience was vital because out here, with the nearest chamber hundreds of miles away, there was only one place you could carry out decompression - the sea. But even with all his experience Tony Liddicoat knew he faced a daunting task. He had to find deep water despite the storm.

Gradually the pile of full cylinders mounted in the boat. Then suddenly it was dark and the wind started going mad.

Eleven minutes later the Puma "chopper" landed on the volleyball court. "It dropped out of the dark," recalls Tony, "like a sack of potatoes!"

It needed six men to lift the paralysed 19-stone Dick Alba to the waiting boat. He had now been three-and-a-half hours out of the water since surfacing to the bends.

Now Tony had the boat, some full cylinders, a willing assistant, Vic Butcher, but he needed to know depths and times so that he could apply his therapeutic decompression knowledge. It was difficult to understand Dick Alba's burbled barely audible words, but finally Tony was convinced that he had got the truth as Dick Alba knew it. But was he under the influence of any drugs administered by the hospital?

He checked and checked again with Alba's wife Marcie, also a diver, and was finally convinced that he had got the right figures (20m for the first dive, then 24m for the second).

He calculated combined dive stops for a dive to two increments below his maximum depth and for a combined dive time for both dives plus two time increments for safety.

Then he went to the BR 2806 Diving Manual, as used by the British Joint Services and from Table II in Chapter 5 he worked out the following decompression schedule:

10 mins at 31m
5 mins at 12m
20 mins at 9m
30 mins at 6m
45 mins at 3m

Dick Alba was unable to help himself in any way and because of his paralysis there was no question of getting him into a wet-suit. So Tony and Vic made a seat out of a paddle and lashed him, still in his pyjamas, on to it.

Now they had to find deep enough water for the recompression. The only suitable place was two miles offshore.

"It was all we could do with the equipment we had", says Tony. "We set off within 15 minutes of him landing and got out into a very rough sea. Twelve-foot seas would be a conservative estimate, with very strong winds. The storm had arrived. We lowered the anchor. We were in 2,000 feet of water and the anchor was just going straight down.

We dressed him in an aqualung and rolled him over the side. I got into the water and caribineered him to the anchor line, so that when he descended he was strapped on the paddle-seat, dressed in pyjamas, fully kitted up. And down he went.

He went straight down to 31m and after about 12 minutes he started to get movement back in his legs. I had written in underwater chalk on the side of the boat for Vic the stops and where I wanted the cylinders suspended. I also had the same written on my fins. Thank God for Vic, otherwise I'd have had to keep bobbing up ...

After about 12 minutes Dick started to get some feeling back in his legs and in his spine. And the use of his hands, and he came to more openly, if you know what I mean, so he could do hand signals ..."

It says a great deal for Dick Alba that when he found himself surrounded by pitch black water, with only the light of Tony's torch for company, he did not panic. Tony Liddicoat was full of admiration for him, particularly when into that beam from that torch swam an eight-foot long blue shark.

"Fortunately", says Tony, "the shark was only curious and after giving us the once-over with that great big cold eye it swam away!"

Now they moved upward through the stops. Six times Vic Butcher sent down fresh bottles as the boat was blown in pouring rain towards the shore. Six times Tony changed Dick's air supply.

"There was only one moment when I had to come up and that was to ask "where are the f-ing bottles". What had happened was that the boat had drifted in so that the anchor caught on the reef and sending the bottles down meant that the anchor rope was at an angle compared to the shot line and they ended up some distance away from me."

As the decompression routine reached the shallower stops at nine, six and three metres, the movement of the boat in the big seas was making it inaccurate and as Dick Alba was suffering great discomfort from all the manhandling and jerking, Tony and Vic got him back into the boat. "I knew now that his life was not threatened, so we took him ashore and kept him under observation."

Now Dick Alba had some relief from symptoms - but he still had numbness, spasms, prickles, rash and blurred vision, and he was sore but Tony did not know how much of the soreness was due to the manhandling that they had been forced to give him during the boat ride and the decompression.

During the 30 hours that he was kept under observation he partially recovered, his left arm was completely restored, there was feeling back in his legs, but he was not strong enough to support his own weight.

It was then that Tony Liddicoat decided to recompress him again and give him this time the complete Table 81 therapy from the Joint Services Diving Manual. And as they started, another storm began to build up. But at least it was daylight and so down they went with Alba still in his pyjamas.

It took seven changes of air this time. The schedule was:

5 mins at 30m
45 mins at 30-20m
80 mins at 20-10m
60 mins at 10-0m

To the disappointment of the men, Dick Alba showed little sign of further recovery or relief of symptoms. On Sunday August 9, 1981, his company sent a Lear jet to fly him at low level to a New Orleans hospital. After two-and-a-half months he was discharged and though he still walks with a stick he jokes about taking up jogging very soon. He can drive his car.

In January this year, Tony Liddicoat received a letter from Dick Alba, which says: "Due to your successful treatments of me at St George's Cay and my subsequent flight to New Orleans and admittance to JoEllen Smith Memorial Hospital, I am walking forever in your debt ..." The letter went on to invite him to be "Guest of Honor at the official opening of the new Hyperbaric Treatment Facility at the hospital on Wednesday, February 17".

The hospital had no doubt about the effectiveness of Tony's treatment. At best, without it, Dick Alba would have been a paraplegic. The hospital said that the decompression had completely removed the nitrogen from Alba's system and thus given him mobility despite the residual damage to the spinal cord.

Tony went to New Orleans and was feted "overwhelmingly ... almost embarrassing ..." But he was not embarrassed by Dick Alba's personal gift to him - an engraved Rolex helium diving watch.

In fact he looked long and hard at that watch when it was put to him that he took a risk in doing what he did. Finally he said simply: "There was no alternative, regardless of the weather and the lack of equipment - you had to do something. If I hadn't, he'd have died."

There was little doubt in anyone's mind about that. Least of all in the Army's reaction. Staff Sergeant Tony Liddicoat of the Junior Leaders Regiment, Royal Engineers, at Dover, was awarded the Queen's Commendation for Brave Conduct for his lifesaving action.

The announcement in the "London Gazette" says that his award was for "bravery, prompt action and skill when undertaking a complicated decompression programme in adverse conditions, in order to save the life of a man suffering from decompression sickness."

Which sums up why Tony Liddicoat is "Diver" magazine's Diver of the Year!

Reprinted by kind permission of the Editor from DIVER, June 1982.

COMMENT

SPUMS' members and other readers of this Journal will have recognised that this was a case for the Edmonds' Underwater Oxygen Therapy Apparatus. When the issue of DIVER containing this article was delivered to its readers, the SPUMS Annual Scientific Conference was discussing the treatment of decompression sickness without a chamber.

NOW READ ON...

SPUMS ANNUAL SCIENTIFIC MEETING 1982

THE RATIONALE FOR DRUG THERAPY IN DECOMPRESSION SICKNESS

AA Bove

Although decompression sickness (DCS) is a disease unique to changes in the pressure environment, data obtained over the past 20 - 25 years has led to the concept that DCS can be treated like many other serious diseases, using standard therapy developed for other purposes. Various clinical investigators, basic researchers, and a few non-medical diving engineers and supervisors conceived of recompression therapy for "diver's rheumatism" in the latter part of the 19th century. Recompression was probably first used for the treatment of caisson workers. Organized decompression and treatment tables for air appeared in the late 19th and early 20th centuries. Oxygen for decompression and for the treatment of DES appeared in the 1920-1935 period, and until recently, recompression and oxygen were the only means for treating DES. From the 1930's to the present time, a body of knowledge slowly developed which culminated in the understanding of DES as a process which triggers body wide inflammation, which can be treated like any other disease process that activates the inflammatory process.

Based on these newly developed approaches to the treatment of DES, one can outline a treatment strategy which, of necessity, begins with recompression and oxygen, but which also includes fluid therapy, the use of corticosteroids, consideration of anti-coagulants, the use of anti-platelet agents and of non-steroidal anti-inflammatory agents such as indomethacine or ibuprofen.

It is clear that the bubble-triggered inflammation which occurs in DES is not reversed by recompression, and although the ischaemic component is resisted by hyperbaric oxygen, use of oxygen and recompression will not halt the inflammatory responses already underway. The process of inflammation encompasses a collection of responses which act normally to counter invasion of the body by foreign organisms (eg. bacteria, viruses, parasites). To gain insight into the use of drugs in DES therapy, we will examine the components of inflammation and the drugs which affect them.

Permeability

The vascular system retains fluid by holding protein molecules in the blood vessels. The osmotic effect of the protein holds water in the vascular compartment. Occasionally, it is expedient for proteins to leave the plasma and enter the tissue spaces outside the blood vessels. The most important need is when antibody diffuses into tissues invaded by bacteria or other organisms. To move protein from the vascular system, a gating mechanism is present in the post-capillary venule. The endothelium in this region can contract, separate cell junctions and allow protein to leave the vascular system. Control of the gating mechanism is through the

inflammatory process. Agents such as histamine, serotonin, bradykinin and certain prostaglandins stimulate contraction of post-capillary venular endothelium and cause an increase in permeability. Direct vascular injury with loss of normal endothelial integrity also increases permeability. When inflammation is triggered locally, by local injury, infection, etc, the reactions which occur locally cause release of vasoactive mediators, protein leakage locally and the mounting of a local attack against the invading organism. The local effect is a normal, necessary, response for protection against a wide variety of organisms. Consider now, how DCS will interact in this process. We have shown in a series of experimental studies that the presence of bubble surfaces in the blood activates Hageman factor, which in turn triggers the clotting cascade, and the complement activation chain. The same processes activate kinins and fibrinolysins. The complement chain produces substances which ultimately release histamine from most cells, serotonin from blood platelets, and activate kinins and prostaglandins. These reactions occur bodywide. There is no localization. Thus protein and water (plasma) leak from the bloodstream into tissues everywhere. Lung oedema is one consequence of this reaction in DCS, and significant arterial desaturation will occur when lung oedema is severe enough to compromise oxygen transport. The massive plasma leak also reduces blood return, haemoconcentrates the blood and will produce hypovolemic shock when severe enough. The entire reaction, triggered by bubble surfaces in the blood, can become self sustaining when tissue injury further activates inflammation. Thus recompression will not reverse this process and intervention with drugs is necessary.

Histamine release stimulates the endothelial cells which contract and open up little spaces between them which is a physiological response. In decompression sickness histamine release occurs all over the circulation. In addition white cells are activated by a number of things. Usually the beginning of the process is a bubble Hageman factor interaction. Hageman factor activates complement, then one of the products of complement activates the white cells. In other words, the signal goes to the white cells saying that there is an inflammatory process going on in the body. The white cells develop pseudopods and dig their way through the endothelium in the area where injury or inflammation has occurred. There are chemotatic proteins that tell the white cells where to go. You get an injury to a tissue which releases a chemotactic material. There is Hageman activation. The white cells are told to go to that place and leave the vascular system and go out and attack something. But in decompression sickness, the chemotactic compounds are distributed all over the vascular system, so that the white cell is getting a signal to dig through the epithelium everywhere. That is a different process to the physiological process. The white cell will damage the vessel wall and that is a component of the increased permeability. Another is the histamine reaction which is a physiological one. Histamine combined with direct injury gives a more prolonged permeability response.

In decompression sickness we have the physiological responses to injury in the micro-circulation, but it is a larger diffuse reaction than usual. Not just a local reaction, but the whole micro-circulation is stimulated. In addition to that we get vessel endothelium damage in many places, because the white cells are being told to go to work. So you get a response which includes the histamine response, and the direct vascular injury response. It is the direct injury response that damages endothelium and causes the more prolonged leakage.

We would like to treat the histamine induced response, which is the physiological response. We can block that, with steroids for example. At the same time we want to tell the white cells to stop digging up the endothelium. You can do that with steroids as well. Once that has been done, the histamine reaction will stop quite soon. The direct damage will not go away. It will have to heal and it takes several days for that to occur. When you are dealing with permeability changes you want to stop the process and replace the fluids. Then keep the fluids balanced until the system heals itself. Direct injury response may take twenty four to thirty six hours to recover. The histamine permeability response, if you stop it, can revert in half an hour. They are two different mechanism.

Clotting

Two different systems are involved with blood clotting in DCS. The intrinsic clotting system, triggered by surface activation of Hageman factor produces thrombi at gas-blood interfaces. When significant bubbling occurs in the blood (eg. in severe DCS) massive clotting can occur. Our previous work indicates that thrombosis of the vertebral venous plexus contributes to spinal DCS. When DCS occurs following blowup, it is likely that massive blood clotting contributes to the high mortality which these accidents produce. The second component of clotting which must be considered is the blood platelet. Bubble surfaces also activate platelets causing them to aggregate and form platelet thrombi on vessel walls and in the microcirculation. Besides the occlusive properties of platelet embolic or local thrombi, platelets release several vasoactive compounds which increase permeability (eg. serotonin) and cause vasoconstriction (eg. thromboxane). Platelet released thromboxane not only causes vaso-constriction, but also increases permeability, further stimulates platelet aggregation, and may activate leucocytes to migrate through vascular endothelium, causing further damage to the blood vessel walls. Once again, recompression will not reverse this process, once it has begun, and drugs which specifically interfere with these inflammatory processes are necessary for treatment.

If you look at clotting in the presence of bubbles, you will activation of the clotting system. That can be in the plasma (intrinsic) and with platelets. There are two different components. The platelet is directly activated by the bubble surface. It releases platelet factor 4 and activates

clotting. You can still activate clotting without platelets. The only protein in the clotting chain that is sensitive to surfaces is Hageman factor. It is sensitive to the lipid monolayer formed around the bubble. The centre of some of the lipo-proteins in the blood become denatured and adhere to the bubble and change the polarity so that there is a lipid monolayer around the bubble surface. That may be the link that gets Hageman factor activated. The fact is that you will find intrinsic clotting activated by bubbles and as far as we know the only component of the clotting system that is sensitive to bubble is Hageman factor. It may not be the surface per se which causes it, it may be the lipid layer that forms from the lipoproteins around the bubble.

These briefly described processes can be altered by agents which interfere with one or more of the components of the inflammatory reaction which has been triggered by bubbles in the blood or by tissue injury caused by gas expansion or by local ischaemia.

THE TREATMENT OF DECOMPRESSION SICKNESS

AA Bove

The treatment of the diver or aviator with decompression sickness (DCS) should always be based on recompression therapy. Recompression is the most important modality, but for the best results it should be combined with oxygen, adequate fluid therapy, corticosteroids, other anti-inflammatory drugs and, in selected cases, anticoagulants. When no chamber is readily available, the other measures used for therapy, even when successful, must be considered as inferior or palliative. Recompression treatment should always be provided even if a significant delay is anticipated prior to recompression.

RECOMPRESSION

Standard recompression therapy can still be based on the somewhat artificial, but practical division of DCS into minor and major types. Minor DCS involving joints is treated with a US Navy table 5 or equivalent [60 ft (18m) intermittent air/O_2] lasting two to two and a half hours. Major DCS is treated with a US Navy table 6 or equivalent [60 ft (18m) intermittent air/O_2] lasting about five hours. Other useful tables such as the Comex 30 [100 ft (3Om) intermittent air/O_2 and air/50% Nitrox] should be made available when a deeper treatment depth is required, and saturation is impractical. Special adaptations of the 60 foot oxygen table can be made for a monoplace chamber where pure oxygen is used to compress the chamber. In-water recompression can used with extreme caution and careful planning, when nothing else is available and the diver's life is threatened.

OXYGEN

The next most important treatment is oxygen. The use of 100% oxygen at 60 ft, 50% oxygen at 1OO ft and 100% oxygen at 1 ATA have all been established as useful therapy for DCS. Two benefits accrue from oxygen: ischaemic tissues may be provided with enough O2 to prevent cell death, and inert gas will be removed from bubbles and replaced by oxygen which will be metabolized. Although administration of oxygen appears simple, several precautions should be mentioned. The oxygen must be supplied with a tight fitting mask to ensure that the necessary concentration is provided. An overboard dump must be used in multiplace chambers to prevent oxygen build up in the chamber atmosphere. The hazard of fire with high pressure oxygen must be borne in mind. Oxygen should be provided to divers with DCS, while at one ATA awaiting transportation and during transit to a treatment chamber. Oxygen toxicity will not be a problem at one ATA unless oxygen breathing is prolonged. (eg. four or more hours continuously). At 60 ft on 100% oxygen however, signs of CNS oxygen toxicity must be watched for diligently.

FLUIDS

Fluid therapy is the next treatment consideration. The diver with DCS is likely to be hypovolaemic because of plasma loss into tissues, and may be dehydrated because of inadequate fluid intake, excess fluid loss or vomiting. The resulting hypovolaemia and haemoconcentration can aggravate the microcirculatory perfusion defect associated with DCS, and produce permanent tissue injury. Thus an important goal of therapy in DCS is to prevent haemoconcentration and hypovolaemia. The best replacement fluid is a crystalloid solution such as normal saline. The use of plasma or dextran does not appear to provide additional benefit. Also these solutions can result in fluid overload which is not readily corrected. Conversely, an overload of crystalloid is easily dealt with by giving a diuretic. Enough fluids should be given to maintain urine output at 100 cc/hour. Calculations based on the standard estimator for fluid deficit can be used successfully. Be sure the patient has normal lower urinary tract function, since bladder paralysis from cord injury may result in urinary retention and an improperly estimated fluid need. One should start an intravenous line in divers with DCS whenever practical and infuse normal saline to reverse or prevent hypovolaemia. One should monitor the urine output and blood pressure. It is essential that the state of bladder function is known.

CORTICOSTEROIDS

Corticosteroids should also be used in treating serious DCS. The glucocorticoids interfere with the inflammatory process. In DCS where the inflammatory reaction is inappropriate and detrimental to the diver, aborting

inflammation is a useful therapeutic goal. Steroids block the action of vasoactive mediators on post capillary venules, they prevent prostaglandin synthesis, stabilize the leucocyte lysosomal membranes, and have other actions which interfere with inflammation. The diver with serious DCS, such a spinal cord injury, or with brain injury from a pulmonary overpressure accident should be given high dose steroids for several days along with other therapy. Dexamethasone 10 mg IV initially followed by four to six mg every six hours for three days will provide optimum benefit. There will be minimal steroid side effects from this regimen.

OTHER ANTI-INFLAMMATORY DRUGS

Non steroidal anti-inflammatory agents such as aspirin, indomethacin, isobrufen also may be useful in DCS treatment. The important actions of these drugs involve platelet inhibition, and blockade prostaglandin synthesis. Prevention of platelet aggregation will reduce the possibility of intravascular clotting and reduction in thromboxane release will prevent local vasoconstriction and subsequent ischaemia. Excess aspirin can block vasodilator prostaglandins. Thus doses of aspirin in excess of 600 to 900 mg/day may have detrimental effects. A reasonable dose is 600 ml/day for several days after the DCS injury.

ANTI-COAGULANTS

Anti-coagulants should not be used routinely but reserved for severe DCS cases. Heparin in full anti-coagulant doses can be used when intravascular coagulation is a major component of the illness. Normally, this type of anti-coagulation is not needed in DCS therapy, and anti-coagulation with heparin is reserved for complex, life threatening cases of severe DCS. Low dose heparin (5000 units every eight hours) should be considered in DCS cases with spinal cord injury because of the risk of venous thrombosis and pulmonary embolism from prolonged immobility. Late anti-coagulation (4-6 days following injury) with anti-vitamin K agents (warfarin) may also be useful in preventing pulmonary embolism in severe, prolonged DCS cases. Nevertheless the occurrence of haemorrhagic areas in the spinal cord in DCS makes one very chary of using anti-coagulants during the acute phase of DCS.

FUTURE DRUGS

Other agents may emerge which will be useful adjuncts to pressure and oxygen therapy of DCS. These include antihistamines, anti-kinin agents and specific prostaglandin inhibitors. These drugs will be developed for use in inflammation occurring for other reasons. As the process is the same regardless of the trigger, they can be used for treating DCS.

TREATMENT OF DCS

The full battery of treatment which includes pressure, oxygen, fluids, steroids and aspirin should be provided for all divers with CNS involvement from DCS. Anti-coagulants and other anti-inflammatory drugs should also be considered in difficult or prolonged cases.

When a chamber is not readily available, all therapy except recompression should be provided. Even then delayed recompression therapy should be carried out to prevent permanent residual injury.

Question:

Why do you say that 500 ml of colloid may be too much? That is only 10% of the blood volume.

Dr Fred Bove

We are not talking about one unit of dextran of 500 ml . If you are going to make a commitment to treating a diver with dextrans, you may use five or six units or more, in the same way that you would use serum. You have an injured diver somewhere at a remote site and you tell the EMTD, Emergency Medical Technician (diver), to start an IV and to run in fluids until the patient is stable. You may now have a diver at a remote site, loaded with dextran, who may be in pulmonary oedema, and there is nothing you can tell the EMTD to do to relieve the pulmonary oedema. You could tell him to do a phlebotomy but that really complicates the issue. Whereas if you have some IV lasix in the kit, and if he has been given too much saline, then all you have to do is tell the EMTD to give him 20 mg of lasix and watch him. Saline is cheaper, it is usually more available and it is easier to administer from the point of view of worrying about allergy and things like that. I would recommend saline, because you get the same basic effect. You get the haemodilution effect with saline. It does not seem to warrant the use of the more expensive and more complicated fluid if you can get the same response from saline. Most of us will start an IV with saline and run it. If the patient is getting better, fine. If he is not getting better, then we go back and reassess. Then we decide whether we want to use a colloid or not. The first response should be to get a needle in the vein and start a drip. The drip ought to be a crystalloid solution rather than a dextran solution.

Question:

Is there a late resorptive phase to decompression sickness as in other illnesses with increased vascular permeability?

Dr Fred Bove

I do not know. You would expect that there would be

because the process is the same. It is a breakdown of the normal vascular integrity, which allows protein to leak. As the fluid gets pulled back into the vascular system you should, in decompression sickness, find that phase. A problem is that we do not have enough of a population of those kind of patients to start doing good clinical trials where we can observe them. There are some efforts in the States to get some of the centres together to try to decide on a couple of protocols so that we can make some sense out of the anecdotal information that everybody has. There have been no clinical trials and there are probably only six or seven a year of the really complicated cases where you can gain some experience. I think you are right, theoretically, because the process is the same. We ought to see a resorptive phase, and yet that has not been described so far. I think only because there have not been enough patients to make the observation.

Question:

Is there a possibility that the aspirin and steroids could cause haemorrhage?

Dr Fred Bove

There is a chance that you can get excess bleeding if you use aspirin and steroids. But that seems to be less of a problem than if you use heparin. In the States most of us would rather hold heparin in reserve and use it for a complicated, difficult case, rather than give it as an initial bolus for the first round of treatment.

Question:

Should not pressure always be part of the treatment of decompression sickness? After all pressure will reduce the size of the bubbles and presumably reduce the surface area activating clotting.

Dr Fred Bove

The point that you made about pressure reducing bubble size and then reducing the surface that activates things is valid, I think that pressure still ought to be on the top of the list. Every effort should be made to use pressure. In the States we insist on not bypassing the hyperbaric chamber for the sake of all the other things. You can use the other things on the way to the hyperbaric chamber. The chamber has to be on your list of things to use. It is still important. It is very important when you have somebody from a deep depth. If you have a blowup from two or three hundred feet, there is no way that you can save them unless you put them back under pressure. You can not stop bubble formation any other way. These people have such a large gas load that they will soon be like a Swiss cheese without pressure. You have got to get them into a chamber and back to pressure within minutes to get them to survive. Pressure is always important and should always be on top of the list.

THE EDMONDS UNDERWATER OXYGEN TREATMENT FOR DCS

John Knight

If you follow the tables closely, as do the USN, serious decompression sickness has an incidence of about 7% of the cases (Table 1). The USN actually normally dive more conservatively than the tables as supervisors add on an extra increment of both depth and time.

TABLE 1

PRESENTING SYMPTOM OF DECOMPRESSION SICKNESS. US NAVY
From Rivera (1963) 900 cases

Cerebral (including inner ear)	6.4%
Spinal	0.2%
Cardiorespiratory	0.4%
Pain only	82.7%
Other	10.3%

However, if you treat sports divers who have deep water to dive in, the picture is very different. (Table 2). Edmonds worked in Sydney and Erde in Hawaii. They treated 100 people and over 50% had serious decompression sickness.

TABLE 2

PRESENTING SYMPTOMS OF DECOMPRESSION SICKNESS. SPORTS DIVERS
From Erde & Edmonds (1975)

	100 cases
Cerebral (including inner ear	33%
Spinal	13%
Both Spinal and cerebral	5%
Cardiorespiratory	1%
Pain only	33%
Other	15%

TABLE 3

HOW TO AVOID DECOMPRESSION SICKNESS

Always do “no-stop” dives
Stay well within the tables
Know your maximum depth
Have an accurate depth gauge
Watch your time
Ascend at 18m a minute or slower
Always do a stop at 5m

If you must do a decompression dive decompress for the next depth and time

Use a shot rope
Have extra air on the shot rope

Do not fly or cross mountains for at least 12 hours

How to avoid decompression sickness ought to be emphasized to divers. Table 3 is a simple collection of information.

This story was published in the British Medical Journal in 1973. A 34 year old diver who dived quite deep on air was a little short on decompression time. He used the usual treatment that divers use for their pain - beer or whisky or some other alcoholic beverage. He turned up at the hospital on an island in the English Channel 15 hours after surfacing. He complained of a pain in his right shoulder, that he was giddy, that he could not walk very well, and that he could not move his right arm. The doctors discovered other things wrong with him. Treatment was rather difficult as he was drunk. They could not get the patient over to England for recompression until the next day. They thought that they really ought to try something in the interim. They used Dextran 40 to decrease the sludging and correct the haemoconcentration and stop lipid emboli. They used some heparin to try and slow down the clotting process. They used Mannitol to reduce the cerebral oedema, and they used fructose to sober him up. They gave him aminophylline for the bronchoconstriction. Probably the most important thing that they did was to give him oxygen. The point is that he improved with that treatment, which is pretty close to Fred Bove's suggested therapy.

TABLE 4

PROBLEMS AND TREATMENT

Problem	Treatment
Bubbles	Reduce size by a. compression b. breathing oxygen
Platelet aggregation	Aspirin
Haemoconcentration	Fluids
Circulatory stagnation	Fluids
Tissue Anoxia	Oxygen

When you are away from home and recompression chambers you still have got the same problems with decompression sickness (Table 4). You have got bubbles that have got to be squashed. You want to get the gas out of the bubbles and you use oxygen for that. You can re-hydrate the patient by putting liquids in. You can improve the stagnation of the shut down circulation with Dextran 40 and fluids. Ordinary electrolyte fluids are probably just as good. The Dextran 40 does work miraculously in people who have got shutdown areas and it seems to unclump and free the red cells. But you must give them plenty of fluids with it otherwise you get a jelly in the terminal renal tubules. And you cannot do much about that in the wilds.

Table 5 lists the factors affecting susceptibility to decompression sickness. Age and physical condition and hydration all affect us. The morning after the night before is not a good time to go diving but we all do it. Those of us who have not dived for a year, are a little anxious when we fall into the water. Those of us who are diving without adequate protective clothing are prone to get chilled. Those of us who have a headache may have taken aspirin and be improving. Those of us who have got anxieties and

TABLE 5

FACTORS AFFECTING SUSCEPTIBILITY

Previous Dives

Acclimatisation	Age
Obesity	Physical Condition
Alcohol	Exertion
Fatigue	Hydration
Chilling	Intercurrent Illness
Anxiety	Drugs

are taking Valium do not know what will happen under-water. As for acclimitization, those of us who do not dive very often are more at risk than the bloke who is working under pressure every day.

TABLE 6

ADVANTAGES OF OXYGEN THERAPY
IN 9 METRES OF WATER

No nitrogen added to tissues during treatment
Bubbles approximately halved in volume Diameter reduced approximately 20% Increased tissue oxygenation
Large nitrogen pressure gradient
No risk of CNS oxygen toxicity
No risk of DCS for attendants
Wet suits still effective insulation

Carl Edmonds' underwater oxygen treatment is considered to be controversial, but is in fact only taking the 60 feet oxygen tables out of the chamber and popping them in the water at 9 metres (30 feet) (Table 6).

It has advantages, you are not adding nitrogen to the tissues, which you do if you recompress somebody on air. The bubble diameter goes down and there is a large nitrogen pressure gradient while you are breathing oxygen at 9 metres (30 feet). You improve tissue oxygenation. You should have no risk of CNS toxicity. You are certainly not going to bend the attendant. And the wetsuit still keeps you reasonably warm. I am not going to say that this treatment is ideal. But when you are faced with a 12-24 hour delay before getting the patient to a chamber, and he has got to pay for the aircraft, I think it is probably worthwhile considering, if you have got this equipment with you, treating him, because you may cure him and save him that Journey.

Table 7 shows what is needed. The patient needs to have a full face mask so that he can vomit or go unconscious without drowning. The patient must wear protective clothing and insulation, a wet suit. You have got to have a shot rope, so you know where the patient is. You need to have ropes tied to the patient so that he cannot drop deeper than 9 metres. They have got to be supported because trying to stay at one depth in the water is extremely difficult.

The patient must be overweighted. Sitting him in the bight of a rope is a perfectly adequate way of supporting him.

TABLE 7

REQUIREMENTS FOR EMERGENCY RECOMPRESSION IN WATER USING OXYGEN

E size oxygen cylinder
Oxygen reducing valve
12m high pressure hose
Full face mask
Depth water 9m
Wet suit
Shot rope
Support
Attendant in the water
Communication system

But the patient will not thank you. In a trial of the equipment at Truk, Janene got very fed up after about 20 minutes sitting in the bight of the rope. We had weighted her around the waist, so her feet were light and floated up tipping her backwards. Also the bight of the rope cut into her bottom. It will cut in even more uncomfortably if it is tied around the waist. These people are going to be in the water for nearly three hours. You have got to have an attendant down there to make sure that the patient is getting better. It is best to have two attendants. This allows an efficient communication system. The patient can speak into the full face mask and the attendant can hear. A yank on the line to get the second attendant down to take over and the first attendant to take a message back to the surface.

TABLE 8

TREATMENT PLAN

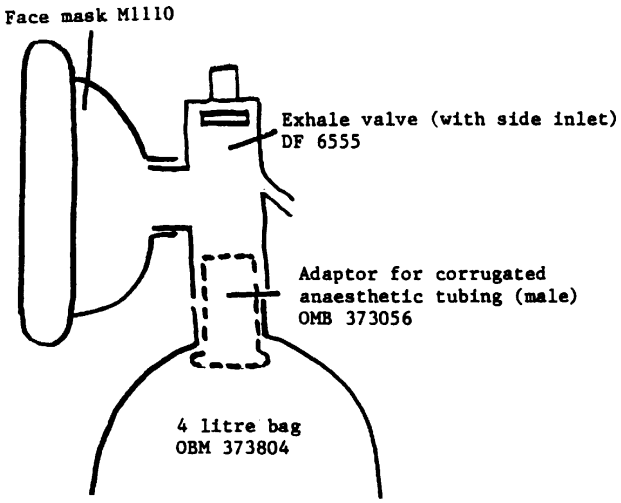
Patient to 9 metres on oxygen (hose length 12m)
Attendant breathing air.
30-90 minutes at 9 metres then ascend at one metre every 12 minutes (or 1 foot every 4 minutes).
If symptoms recur, halt ascent for 30 minutes.
If oxygen runs out bring the patient to the surface.
Do not give the patient air underwater.
After surfacing give 100% oxygen alternate hours for 12 hours.

It is a good idea to have a non-return valve at the bottom of the oxygen line so that if anything goes wrong the face mask does not suddenly fill up with water. The mask needs to be a tight fit. If it is not tightly enough applied it will leak.

The hose is marked off in feet or metres because the treatment is extremely simple. (Table 8). You take him down to 9 metres. You leave him there for 30 minutes and then if he is better, you pull him up at the rate of a metre every 12 minutes or a foot every four minutes, which is probably a better method of reducing pressure than coming up in metre steps every 12 to 15 minutes. Marks on the hose are a great help in controlling the ascent.

If he has a neurological bend he gets at least 60 minutes at 9 metres before being brought up. The stay at 9 metres can be safely extended to 90 minutes. The E size cylinder will allow 90 minutes at 9 metres and the 120 minutes of decompression as well as having some left for oxygen at the surface.

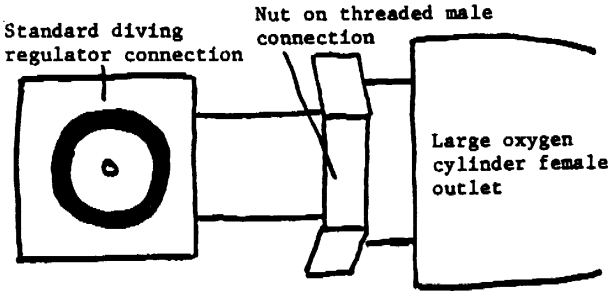
Figure 1



One must continue to give him oxygen once he has been decompressed. For oxygen at the surface one can either use a simple bag and mask set up (Fig. l) made from readily accessible anaesthetic equipment or the diver's regulator as recommended by Dr Mike Davis of Christchurch, New Zealand. To use a regulator with a D or E cylinder one needs an adaptor (Fig. 2) to screw into the cylinder outlet in place of the reducing valve used for the underwater treatment. The bag and mask has the advantage that you can watch the bag moving with each breath and see that the chap is breathing properly.

FIGURE 2

ADAPTOR TO FIT A DIVING REGULATOR TO A LARGE OXYGEN CYLINDER



Dr Bove has published a paper on the above topics, THE BASIS FOR DRUG THERAPY IN DECOMPRESSION SICKNESS. Undersea Biomedical Research. 9(2): 91-111. It is an expanded version of the papers printed in this issue.

Dr A Reid

In Rabaul we have used this equipment to treat a diver. Luckily we had had a few practice sessions, otherwise we would have had trouble.

There are a few things that I would like to share with you. You must have an organisation. If somebody gets bent in a place like this it is hard to get hold of people. We have to have three or four people who know all the organisation, and who can round up people and equipment. The only place we can do in-water oxygen recompression is a certain dock in the harbour. We have to get the Harbour Master to open up the dock. We have all this worked out ahead of time, otherwise when you do have somebody in trouble, you are going to be chasing all over town getting things organised. You must have a master list of your staff. You have to have somebody in charge of the operation (who is not the doctor at the site) who manages all the equipment and stuff, and the people going in and out. It is enough for the doctor just to think about the patient. We use a breeches buoy from a sailing boat instead of a rope for the patient. It is much more comfortable. We hang the weights on the bottom of the breeches buoy.

Although timing sounds simple here, we had distractions from people coming and going. So we have one person in charge of timing. He is also the person who tells how many feet to come up at each time interval. You have to have your rope properly marked. You must have exactly the right number of markers on it. You can easily get confused after five or six metres. The person who is timing has to have a proper sheet to write everything on. We found that people get cold and hungry, so hot drinks and things like chocolate bars are very helpful for the attendants. If it gets dark, as it does here at six o'clock, you have to have lights. Two or three lights are required. We have two or three people going up and down communicating and one person with the patient. So there was a communicator at the bottom if they wanted to send a message up, and there was a communicator on the surface to send a massage down. They rotated. They also had slates and pencils either to play noughts and crosses with the patient or to give communications from the patient.

I think the most important thing that I have found is that you have to have practice. It is not simple. It is simple in theory but there are a million things that can go wrong. Connections that do not fit the oxygen cylinders, something does not work, something else is broken. You should get together every now and then and practice. I found that this practice was very, very helpful.

Dr John Knight

That reminds me that I forgot to mention the great contraindication to this treatment, cold water. If you have got a cold diver and you put him back for another three and a half hours, you have got a hypothermic, very sick diver on your hands. Carl Edmonds designed this equipment for places where you could not get to a chamber quickly. Quickly meant about twelve hours. In Madang, unless there happens to be a Lear Jet at the airport, to get a bloke to the nearest chamber, which is at Townsville, you have got to fly the RAAF or a Lear jet from Australia to here, and then back to Australia. By which time you may well have made the man better by putting him back in the water.

Hypothermia is a very real contraindication to this treatment. When I am asked by people in Victoria and New South Wales whether they should buy these kits, I say very firmly that they should not. They are only useful in warm water. Heron Island is not warm enough, because they had to abandon one treatment at Heron Island, because the patient said he was too bloody cold and that he was coming up.

Dr Carl Edmonds

It does work OK in the Antarctic. All you need is oxywelding equipment. Use the oxygen regulator from that and industrial oxygen. Anyone who has an oxywelder could do the job very well. You do not have to use diving equipment to make it. We used an oxywelding regulator in the Antarctic and it was actually more effective than the

SALT WATER ASPIRATION SYNDROME MISDIAGNOSED AS OXYGEN CONVULSION

Carl Edmonds

There is one thing that worries me. I have noticed that people are using a case reported in the SPUMS Journal (0ct-Dec 1981, p23) to claim that there was a convulsion on oxygen at 9m. I do not doubt that you can convulse on oxygen at 9m, obviously it can happen. The original work done by Haldane has a report of a diver who did.

However, that case report is a terribly bad case report and if you are going to use oxygen toxicity as an argument, you should not use that case. The man firstly had salt water aspiration syndrome before he went on oxygen which means that there is no way he could have got his arterial oxygen up to normal let alone hyperbaric oxygen levels. They had to treat the man for salt water aspiration afterwards, which shows how seriously ill he was.

I think that what was described was something that they would have recognised, had they had a bit more experience, as a rigor from the salt water aspiration syndrome, not an oxygen convulsion. That case report should not be used by anyone as a case of oxygen convulsion.

DECOMPRESSION SICKNESS AND AIR EMBOLISM CASE REPORTS

Ian Unsworth

The first patient is not a diver. She was a 52 year old lady, of 51 kilos, who was admitted for the excision of a large solitary secondary rumour in the posterior quadrant, from an un-differentiated large cell carcinoma in the apex of the left upper lobe. She had presented with cerebellar ataxia. Diagnostic bronchoscopy and CAT scan had been done, which showed what the problem was. She was admitted for a sitting posterior fossa exploration. The pre-medication and induction of the anaesthetic was quite standard. She was monitored after she had been positioned head up on the table, with an arterial line, a right atrial catheter and an end-tidal CO_2 monitor. Induction occurred at 11.30. One hour 30 minutes after induction air was heard to be induced into the venous system, in the posterior fossa. The end expired CO2 fell from 2% to 1%. The blood pressure fell transiently to 60 mm mercury, from a very steady 150. The nitrous oxide was turned off. Halothane in 100% oxygen was turned on. A Valsalva was applied and about 30 mls of air was aspirated from the right atrial catheter. Peripheral vasoconstrictors were used and a large volume of fluids. I was not anaesthetising this patient, so this story is from the anaesthetist concerned.

Full recovery to the status quo was fairly quickly assumed and the operation proceeded. A further 28 minutes later, the air was again heard to be bubbling and hissing into the venous system in the posterior fossa. The end-tidal CO_2 was observed to fall by one half percent to 1.5%. There was not an alteration in the blood pressure, and no further air was aspirated from the right atrial catheter. On neither occasion was there any cardiac arrhythmias. The patient left the operating theatre, with the excision completed, at 14.20.

I happened to be near the recovery ward shortly after that, and I overheard a conversation about two episodes of air embolism in the theatre, so I was rather interested, The recovery room people said, “You might be interested in this case, perhaps you could advise us. What do you think we ought to do?” So I asked “What is the patient like?” They replied “We have given her 0.8 mg of naloxone because we wanted to see what she was like as she has not completely recovered from the anaesthetic and is not very well. She has a dense triplegia and she has not got much movement in her fourth limb either.”

The neurosurgeon was consulted and he agreed that hyperbaric oxygen might help the patient. She was given Decadron 60 mg intravenously.

I was at the hyperbaric unit to receive this lady, and I was absolutely amazed. She was fully conscious, she had no evidence of cerebral involvement whatever, no cranial nerve involvement, her speech was perfect, she had full appreciation of the situation. She was a charming lady. I was able to explain to her in simple layman’s terms what was going to happen to her. She said that it was fine by her, so there was certainly no apparent cerebral involvement. However, she had no movement in either of her legs, and she had no movement of any part of her left arm, and she could only just move the right hand, and wriggle the fingers very slightly. Sensation was apparently normal.

We put her into the chamber some two hours after the termination of the operation. There was some discussion about going deep. I did not. I took her only to 60 feet. I do not take patients off oxygen. I do not give air breaks, except in the very long treatments as in Table 6. It is useful to have the patient off the mask occasionally to have a drink. It also allows proper conversation with the attendant. I seldom have more than two breaks in four hours. I feel that if you interrupt the oxygen with air then you will get less effective nitrogen removal. She had 120 minutes on oxygen at 60 feet. She had intravenous dextran 70, at 125 ml per hour. We decided that Decadron 60 mg 8 hourly might be satisfactory.

Following the first session we found that the only improvement was that there was some slight movement in her right leg. No more than that. Everything else remained as was. So we were not all that optimistic. We put her in the chamber next morning, 14 hours after the first treatment. She had the same 60 feet, 120 minute treatment. After that treatment, we could move her right arm and her right leg on command. I wondered about treating her again. But before I could get around to giving her a third treatment, she improved in an almost miraculous way. She regained all limb movements. She could move all the small muscles of the hands and feet. The day after that not only was she sitting out of bed, but she was able to walk with support. Certainly there was no-one more impressed than the neurosurgeon, who had been talking to the relatives about unfortunate spinal damage, and this sort of thing.

I would like to hypothesise that air entrainment probably occurred during the second episode and that the entrainment was into the vertebral venous plexus, up in the brain stem, and that it crept down and affected the cord. As you how, Fred Bove has done some elegant work with David Elliott and John Hallenbeck many years ago, on the influence of air, admittedly air released during decompression, in the aetiology of spinal decompression sickness. What this lady may well have suffered from was air entrainment into the venous plexus of the cord, or the brain stem, without any cerebral involvement at all. If you do get air into the venous plexus, of course this will result in a brain stem infarct.

At the 1980 Scientific Meeting, John Miller made the point that these cerebral and spinal problems, following neurological procedures or open heart surgery, are often apparently refractive to treatment. You treat them for perhaps 24 hours to 36 hours without success and then improvement occurs. This lady we treated within 2 hours or 2 1/2 hours of entrainment, but still she did not come good on the first treatment.

The next patient was a fit 21 year old male, a sports diver. His diving history was one year's experience of diving. He had full equipment, including a watch, depth gauge, buoyancy compensator, so he was quite well equipped. He had even had a diving medical, from myself as it so happened, the year before. He and three friends decided that they would dive off the heads in a spot known to be 170 feet or more in depth. Just before they got to the dive site, they all ingested very large amounts of Seconal. The idea being that this would be an extra "hit" and the "narcs" would really send them. It did.

On the bottom, after an undetermined time, the patient lost consciousness. He was dragged up by his mates, and dragged backward into the boat. They returned to the shore and this boy was taken to the flat of one of his mates. He remained unconscious for 48 hours. His "mates" thought that he was sleeping off his drugs. When he awoke, he was, to say the least, befuddled. He had paraesthesiae in both legs. He had profound weakness in both legs. He was ataxic. Apparently there was no visual disturbance. His mates decided that they could not look after him any longer and they dumped him in casualty. One of them stayed long enough to give about 30 seconds of history to the nurse in casualty. Then they just beetled off into the night.

So I collected this boy, who spoke very slowly and in a particularly drugged fashion. He was feeling a profound muscle weakness in his legs, paraesthesiae in his legs, severe lower back pain, and paraesthesia in the palm of his left hand. Reflexes were increased in both legs. Interestingly the plantar response was down. He really had to be treated as a spinal decompression sickness. He was given intravenous dextran 40, half a litre in four hours. We had no history of any bladder problems. He did not produce any urine, so perhaps he had made no urine at all in the last 48 to 60 hours. He certainly was rather dehydrated. He had dextran and other fluids, but we did not give him steroids. He was given table 6, the four hour haul, with a very good result. He did take a little time to respond, but once he did, all function returned quite well. By the end of the treatment time he was approximately 99% which was very fortunate.

You might think that was the end of the story. He was thinking that it was. But he developed a reduced air entry and breath sounds at the right base. X-rays showed that he probably had a lower lobe pneumonia. He had a fever and purulent sputum. This cleared up with physiotherapy and antibiotics and it was probably due to lying immobile for 48 hours.

Our drug screen on his urine showed barbiturates, phenobarb, paracetamol, codeine, so he had a good run of the drugs. His past history showed that three years before he went diving, he had been admitted to Prince Henry Hospital suffering from fits secondary to barbiturate withdrawal. He had been on various drugs at that time, including barbiturates, for insomnia. His friends used heroin. A year later, in 1980, he was admitted to Prince Henry again with an overdose of Seconal, and admitted at that time the occasional use of marijuana and heroin sniffing.

We had a happy ending to an unhappy boy. What is the moral here? I believe that this could well be the tip of the iceberg. We have got to admit that there are an unknown number of divers who dive for kicks. We have also to admit that there might well be a number of divers who are on drugs or use drugs, or combine the two. I believe that we have to be firmer. It is difficult how in fact one can screen out addicts, because he did not admit to any drug taking at all when I saw him for his medical. This of course is what drug addicts are going to do.

We have to educate divers that it is foolhardy to use drugs and then dive. I have written in the Medical Journal of Australia that I consider if a diver dies after taking drugs such as narcotics, sedatives or psychotrophics it is suicide, and if his buddy dies then it is murder. Perhaps we have to be more careful in assessing our cases of decompression sickness. I wonder whether we should do urine screens routinely on all cases of all types of decompression sickness and air embolism.

Dr David Brownbill

Fred Bove spoke earlier about the two basic causes of oedema. We ought to extend that to the central nervous system, to both the brain and spinal cord. Steroids for example may well prevent new oedema formation, but will not magically reverse oedema that has already occurred. In the case that Ian Unsworth has described, damage had already occurred. There was central nervous system dysfunction, so that was cell damage. In this case, the patient improved after 24 to 36 hours and one may postulate that oedema had settled down in that time.

This does bring up one point that I would like to make. When people have central nervous system dysfunction either the spinal cord or the brain, where cerebral oedema is a problem, whether it is due to being hit with a 10lb sledge hammer, a stroke, a cerebral metastasis or decompression sickness, the one thing that can do untold harm is the well-meaning bolus of fluid. I appreciate the problem in decompression sickness as a general proposition, that you must keep up the fluid to prevent dehydration. But if there is central nervous system involvement, I would very strongly recommend not to over hydrate. Overhydration may well push the patient into a worse situation.

Dr Fred Bove

This problem of rehydration is always a difficult one to deal with. If the circulation is unstable you have got to rehydrate to get the stable circulation. I think that is the first indication for the need for rehydration. If you had a hypovolaemic shock type of syndrome, you would have to get that blood volume back up to where it belongs. One must restore normal circulation instead of worrying about the central nervous system or the lungs for that matter. The approach that I have always taken is to treat where the problem is most acute and deal with that first. If there is

circulatory instability then you need to treat with fluids. So go ahead and do it. But be ready to deal with possible complications. You have got to take care of one thing at a time.

If the patient is stable and has a normal haematocrit and you are concerned about cerebral oedema, I agree, we ought to hold back on the fluids as much as possible. But one must maintain an appropriate amount of rehydration so that the patient does not suffer from deprivation of fluids.

Dr Ian Unsworth

We all know about the conflict of interest in the intensive care unit between the neurosurgeon who comes in, looks at the patient and says to leave him for half a day and goes away. And then the intensivist comes along and says that those kidneys will never stand that dehydration. Usually there is some sort of compromise, to preserve both the brain and the kidneys.

A CASE OF PULMONARY BAROTRAUMA IN AN ASTHMATIC DIVER

David Clinton-Baker

I am a general practitioner from Wangarai in New Zealand. Last month I was called to the evacuation of a twenty year old, an asthmatic, who suffered from pulmonary barotrauma with air embolism. The story follows on quite nicely from the comments about cerebral oedema.

It was his first deep water dive, the first dive of the day. He spent about eight minutes at 100 feet, seven minutes at 80 feet, and then about fifteen minutes getting up to 50 feet, from where he was seen to make a more rapid than usual ascent to the surface by his two buddies. I have spoken to the patient about this since. He says that at 50 feet he had a bursting sensation in his chest. He also describes burping up air. He remembers getting back to the surface. He remembers, just, getting to the stern of the boat. He remembers nothing after that.

He was pulled semi-conscious into the boat, and he was not breathing. CPR was instituted. The boat took some three hours to reach the coast. On the journey he breathed oxygen enriched air. During this time he had four grand mal fits. I first saw him at the coast when he was semi-conscious. When I asked him his name he responded. Very soon after that, he had another grand mal fit. Examination was unremarkable. His reflexes were normal and there were no neurological deficits at all.

We evacuated him to Auckland, which was an ambulance drive of another three hours. There was a total lapse of six hours from the time of the accident to when he was received at the chamber in Auckland.

As soon as I saw him I started a dextrose saline infusion and gave him 10 mg of Valium IV. During evacuation to Auckland, he had three more grand mal fits, and for the last hour of the evacuation was fitting continually, initially generalised, but towards the end with focal right sided fitting involving the right arm. He was received by Tony Slark at the RNZN Hospital at Devonport. We tried to X-ray his chest but due to the fitting this chest X-ray left something to be desired. However we were almost 100% sure that there was no pneumothorax. He was put into the chamber. He was taken initially to 60 feet on intermittent oxygen. He appeared to be very sensitive to oxygen and did not really respond. So we took him down to 165 feet on air. He spent a total of sixteen hours in the chamber, and was then transferred over to the Department of Critical Care at Auckland Hospital. During the evacuation from the coast to the chamber, I gave him a total of 880 mg of diazapan and Tony gave him a further 60 mg of diazapan with virtually no effect on the fitting.

In the Department of Critical Care at Auckland Hospital, shortly after his arrival, he developed extensor spasms and it was decided to ventilate him to a PCO2 of 30 to 35. His circulation later became unstable requiring infusions of Stabilized Plasma Solution and Lactated Ringers Solution for maintenance of his left atrial pressure, later supported by dopamine and dobutamine. On this regime he stabilized after a septic episode requiring ampicillin, cloxacillin and tobramycin for control. Sedation was continued with phenobarbitone. An early EEG was reported as Grade 3. On the fifth day he was breathing spontaneously and over the ensuing seven days he improved. He was left with a residual right hemi-paresis, which affected his right arm more than his right leg. Over the last three days of his admission he rapidly improved, with increasing power and co-ordination of his right arm. He was discharged home after fourteen days. He had to be re-admitted a fortnight later with further grand mal fits. He was then put onto prophylactic dilantin. When I spoke to him about a fortnight ago, he still had some residual weakness of the right arm, but was improving.

A brief outline of his asthmatic history and how he managed to get into the diving course. He developed a wheeze aged eight. In his early teens he required continuous prednisone, 10 mgs a day, as well as inhaled ventolin and several intramuscular injections of adrenaline, but no hospital admissions for his asthma. The five years or so before this diving accident, his asthma had been quite good, only requiring occasional ventolin and nightly inhaled beclomethasone. He went to his university club about four months ago, asking to be accepted into the diving course. He was referred to his local hospital, which is the largest in New Zealand. He was investigated by a physician there, which involved three hours of interrogation, examination and investigations and was told that he would be fit to dive.

He then went into the diving course, and the dive I have detailed was his first deep water dive.

During the evacuation by ambulance I was giving him fluids, I could not see him improving, so I stopped the

fluids after about half of the ambulance journey. I was going to ask about cerebral oedema, but that question has been answered. Of course, this case also raises the problems about asthma and diving even if the asthma appears to be well controlled.

Question:

Why did you diagnose air-embolism?

Dr David Clinton-Baker

I think it was a diagnosis of assumption because he made a rapid ascent from 50 feet. There was no macroscopic evidence of pneumothorax. There was no emphysema. It was assumed that he had an air embolism. He never had any bronchospasm. There was a doctor on the boat, who auscultated his lungs and thought there were sounds on the left base but there was never any evidence of bronchospasm. He had inhaled salbutamol before diving. He had the salbutamol aerosol in his diving bag.

The bursting sensation inside his chest is the last thing that he can remember clearly. He thought that he ascended at a normal rate, but his buddies tried to attract his attention and they could not get any response from him. They both said that he ascended too quickly. But his memory of the accident is obviously quite blurry, and this feeling of an explosion in the chest may have happened after or during the last ascent. His memory is quite vague.

Question:

Should the absence of severe episodes of asthma for the previous three years indicate fitness to dive?

Dr David Clinton-Baker

I am sure that he had had virtually no trouble from his asthma, it had completely resolved by episodic inhalations of salbutamol for about four years.

But after this episode I would ask myself whether anyone who had a history of asthma requiring treatment is fit to dive.

Question:

There is no real evidence that he burst a bulla. Perhaps his asthma was co-incidental. Why did you diagnose an air embolism when he did not have a pneumothorax?

Dr Carl Edmonds

I am worried by the last question. Most cases of cerebral air embolism do NOT have pneumothorax or mediastinal emphysema. Why anyone should use the absence of pneumothorax as an argument against the diagnosis of cerebral air embolism is beyond me.

THE THEORETICAL BASIS OF THE US NAVY AIR DECOMPRESSION TABLES

Bruce Bassett

My topic is decompression. It is the area of physiology that I have spent most time with, starting with aerospace physiology and the problems of decompression of aviators and in later years, twenty years or so, getting involved with the diving side of it. I am going to discuss this and the theoretical basis of the US Navy tables and an analysis of their safety. In this first presentation, I will just cover the theoretical basis. My second talk is a proposed design for sport diver tables, and that is a natural offshoot from the safety analysis of the tables, so those will come together. The final presentation will be about the problems of flying after diving and diving at altitude.

I am going to limit my discussion to the US Navy tables. There is good reason for that, as the tables that we are using here in Madang, however they are laid out as sport diver tables, are all based on this standard. The US Navy tables are probably the most used tables in the world.

Your primary speaker next year is Brian Hills, who has another concept about decompression altogether. So I will be talking about the old historical stuff, while he will be talking about his theory and maybe the twain shall never meet.

US Navy diving tables are a masterpiece of design. They evolved over the years. They represent a cookbook for diving, with recipes for decompression. If you can read, you can follow these schedules. By design they have to be this way, they have to take care of the average layperson diver. You do not have to have a high school education or a university degree to understand them. If you follow the instructions they are pretty easy to use whether they are presented in the original format or in the sport diver format. The numbers are all the same. If you can follow instructions and if you can read, then you can follow the tables for decompression.

I like to go into the theoretical basis for the tables because it is somewhat obscure. Unless you can find somewhere the 1906 Journal of Industrial Hygiene from London, you do not find Haldane's original stuff. Haldane and Priestly published a textbook on respiration up until the 1930's, so you can find a little bit of this up to about then. The US Navy reports on the development of these tables, which was really just an offshoot of Haldane's stuff, are buried in the Experimental Diving Unit Reports, which are classified, and are not sitting around in most medical or other libraries. I will try to present in this paper a little about what Haldane found, why he developed what he did, and what the Navy did with that. In the next paper I will talk about the analysis of how good they are or how poor they are.

I do not like the term no-decompression, but that usage is very common (no-decompression is a dive that you never

come back from) although no-stop diving is a better term. If we draw a graph of the no-stop limits, the "no-decompression" dives of the US Navy tables are to the left of the line while to the right are those dives which require stops on the way up. We are told, as sports divers, that we should never get into decompression dives. We should always stay to the left of the line. There are some implications in that statement that I am not sure are truly valid. However, I will discuss that in the next session. If we go to the right of the line, we get into the requirement for staged decompression. That is decompression stops on the way up. If you are strictly a sport diver, you may have never graphed out the profiles. But if you have studied the tables a little bit, you realise that as your exposure gets longer the decompression penalty, or the cost of your exposure, becomes progressively longer. For example at 150 feet for thirty minutes, you spend about an equivalent time in decompressing. For thirty minutes of play, you spend thirty minutes of boredom. If you spend eighty minutes at bottom at the same depth the time spent coming up is about two and a half times the bottom time. During this discussion of the theoretical basis of the tables, I hope it will become clear why this works the way it does. In decompression dives in the US Navy tables, the first stop is always short, and they get progressively longer as you work towards the surface. This is because of the basis of the model.

The basis of the US Navy tables is based on a concept of supersaturation and allowable supersaturation. The concept of the US Navy tables is that you can have a degree of supersaturation. So what is supersaturation? Supersaturation exists when tissue nitrogen tension or pressure exceeds the total barometric pressure acting on the body. In physiological shorthand PN_2 is greater than PB. You have to think about that one for a while to see how it exists. Some divers have trouble with this because they know about Henry's Law. We know that nitrogen tension is only a fraction of the total pressure so how is it possible to have a nitrogen pressure greater than total pressure? Time is what is involved here. You go to depth, spend some time breathing compressed air, taking up nitrogen, increasing the nitrogen tension in the tissues, and then, particularly in the area of sports diving, you make a direct ascent to the surface. You have not spent as much time in decompression as you did exposing yourself to the increased nitrogen pressure. So you can, in fact, reach the surface with a nitrogen pressure in the tissues greater than the barometric pressure surrounding you.

That is a definition of the term "supersaturation". The tables are based on allowing a degree of supersaturation. There are three ways of expressing this degree of supersaturation. It can be expressed as a ΔP which is an absolute pressure difference between what is in the tissue and the ambient pressure. It can be expressed as a M value, which we will get to later on, which is a sort of tabular relationship of inert gas over pressure. Or it can be expressed as a ratio. There is a maximum tissue nitrogen pressure which is tolerated at any given total barometric pressure. If you express this as a ratio, the ratio is equal to the nitrogen pressure divided by the barometric pressure.

I will elaborate on this point, because it is essential to the understanding of the way that the tables are built. If we have a man in equilibrium at any pressure, he is said to be saturated. So if we have not been diving for some period in excess of whatever time it takes to reach equilibrium, at sea level, we have nitrogen pressure of approximately seven tenths of an atmosphere dissolved in all the body tissue and fluids. The surrounding barometric pressure on our body is one atmosphere, so under these conditions, barometric pressure is greater than nitrogen pressure. We are not supersaturated. We are merely saturated. We are in equilibrium. If we take a man to a depth of 33 feet of sea water, ten metres, two atmospheres absolute, and leave him there long enough to reach equilibrium, his nitrogen pressure will increase to 79% of this total pressure. Again, so long as he is maintained at that pressure, barometric pressure is greater than nitrogen pressure, and he is merely equilibrated at a new barometric and nitrogen pressure. Now take this individual instantaneously back to sea level. He now attains the condition that we have to define, the state of supersaturation. Upon reaching the surface, he still has 1.58 atmospheres of nitrogen in all of the body tissues and fluids. The surrounding barometric pressure is less. It is 1 atmosphere. Now we have nitrogen pressure exceeding barometric pressure so he is supersaturated. If you wanted to express this as a ratio then it would be 1.9.

The concept of supersaturation that developed from Haldane's time and then into the building of the Navy tables was that if you are at critical supersaturation or less, the excess nitrogen pressure finds its way via the circulatory system back to the lungs to be exhaled. So, as a function of time at the surface, you rid yourself of the excess gas. If you exceed the critical supersaturation relationship, then the force of excess gas pressure is great enough to cause the evolution of bubbles involving tissues and fluids. So that is the basic bubble theory as supersaturation describes it. It is essential to the way the tables were built.

By the end of the last century Royal Navy divers were having such a problem with decompression sickness, that the British Admiralty appointed a Commission to study the problem. The physiological representative on this committee was JS Haldane.

Haldane worked with the hyperbaric chamber at the Lister Institute. His primary animal model was the goat. Goats are still used in decompression research. They display bends quite nicely. I have done a little work with them in altitude studies. Haldane had a starting point when in 1900 he was appointed to this Commission, a starting point based on knowledge possessed by divers. The divers of the day knew that, if they did not have exposures greater than about 30 or 33 feet, for the times that they were occupationally exposed to, they had no problems when they came to the surface. They started to have their problems of decompression sickness when they exceeded this depth. It seemed to be a threshold depth.

Haldane's basic observations about decompression were based on this fact. He took his goats to a depth of 33 feet of sea water, 2 atmospheres absolute, which is a nitrogen pressure of 1.58 atmospheres. As best as he could estimate it, it would require his goats three hours to reach saturation, to become equilibrated at that pressure. By extrapolation, he felt that man would require about five hours to become equilibrated at that pressure or any other pressure. An important point here is that the time to reach saturation or any percent of saturation on this model, is the same regardless of depth. So that at 200 feet it still requires three hours for a goat to reach saturation. To reach some given percent of saturation (less than 100%) would require the same amount of time whatever the depth.

He took his goats down and repeated what was known at that time. He took them down to 33 feet and kept them there until he felt they were saturated, then brought them directly to the surface and found that they did not display any signs or symptoms or manifestations of decompression sickness. Presumably because they were not forming bubbles. If you look at the conditions that presumably existed the animals, if they were equilibrated, and they were pretty nearly saturated, had 1.58 atmospheres of nitrogen pressure in all of the body tissues and fluids. So when they hit the surface they were withstanding a ratio of nitrogen pressure to barometric pressure of 1.58-1.

The logical thing to do was to work the goats progressively deeper, saturate them and find out what the tolerance was to decompression. Obviously, if you were doing this scientifically, you would do it in stages. Haldane started at 2 atmospheres and gradually worked deeper. If we take the example of 5 atmospheres absolute, 132 feet of sea water, nitrogen pressure 3.95, keeping the animals there for three hours, until they were presumably saturated, he found that as long as he did not decompress them any shallower than 2.5 atmospheres they were fine. If he tried to get them any shallower they started to display symptoms of decompression sickness. So if you look at the ratio of nitrogen pressure to barometric pressure, under these conditions, it turns out to be the same ratio that he observed in the first series of animal exposures. He worked to a maximum depth of 6 atmospheres absolute, 165 feet. This relationship seemed to hold true throughout. As long as the animals were not exposed to a supersaturation ratio of greater than 1.58 to 1, they did not have any symptoms. That was his initial empirical observation which allowed him to start building some tables.

If you are at 5 atmospheres and you reduce the pressure to 2.5 atmospheres the total barometric pressure relationship is a two to one relationship, as with two atmospheres coming to sea level. So very often you will hear Haldane's finding expressed as the two to one relationship. But in fact the driving force for gas phase separation is the excess nitrogen or inert gas pressure compared to barometric pressure. This is the overpressure of gas trying to come out of solution. Normally the gas is breathed off in the lungs but if the pressure difference becomes too great, then the forces are such that gas phase separation occurs.

So Haldane's empirical observation was that there seemed to be a critical supersaturation ratio that could be tolerated. The next phase in the developing of safe decompression was to build some sort of decompression schedule from this. First of all from this observation you could assume that, whatever the pressure you were saturated at, it was safe to reduce the pressure by one half. If you were at 5 atmospheres, you could always come directly to 2.5 atmospheres, no matter how long you had been at 5 atmospheres. Then you would have to consider saturating at 2.5 atmospheres and then reducing that by half again. Eventually you would reach the surface that way. But that is not realistic. If in fact it takes three or more hours to reach saturation you would like to know where you stand in terms of percent of saturation at any given period of exposure time. So Haldane's next problem was how to estimate the rate at which you approached saturation, the rate at which you take up and eliminate nitrogen.

He had three major factors to consider and a whole bunch of other variables. We will just look at three factors that cause it to be a fairly complex job to estimate what percentage saturation you have under a given period of exposure at any given pressure. When you got to depth and started exposing yourself to increased inert gas pressure, you established a large pressure difference between the nitrogen pressure in the lungs and that in the tissues. This large ΔP, pressure gradient, causes a rapid initial uptake of the inert gas. The circulatory system carries inert gas out to the tissues as a function of time, so tissue pressure is increasing. You are exposed to a fixed nitrogen pressure so as a function of time, ΔP is decreasing. Which means that the rate at which you can expect gas to transfer to the blood from the lungs and from the blood to the tissues, would slow down. Initially with a large ΔP gas uptake would be rapid and as time went on and the ΔP decreased, the rate at which nitrogen uptake occurred would slow down. And all this says is that you would not expect a linear relationship between time and percent saturation. You would expect some sort of exponential relationship, where uptake was fast at first, slowing down as the pressure gradient decreased. That would be the physical consideration.

To complicate this a little bit, we also know that the solubility of the inert gas that we are breathing is different in different types of tissues. For example, nitrogen is five times more soluble in fat that in water. So if you have a great deal of fat, it will hold more nitrogen and, theoretically, this would take more time to reach a given degree of saturation. If we describe the decay of the pressure gradient as causing an exponential curve, we can visualise that we have at least two exponential curves. One that describes the nitrogen uptake in water and one that describes it in fat. The curve for fat being the curve with a shallower slope, taking longer to reach saturation than in water because fat holds more nitrogen. We all know that in the body there is no clear division into adipose tissue and water only tissue. Every tissue has a certain degree of fat in it. The proportion of fat in a given tissue is going to tell you that tissue has a different rate of uptake to another tissue.

It is not just a simple problem of two curves to consider, one for water and one for fat. It is a whole family of curves for nitrogen uptake and elimination.

If that is not enough to confuse us, then we have to consider the transport system for getting nitrogen to and from the tissues, that is the blood flow. The rate of delivery or removal of nitrogen is dependant on the perfusion of the tissues. If you look at the actively metabolising areas of the body in a resting man, things like the heart, brain, active bone marrow, kidney and thyroid receive 75% of output, yet they represent only 25% of body weight. At first approximation, ignoring the fat issue, we could say that this represents 25% of your nitrogen capacity. Yet you are servicing this 25% reservoir with 75% of your blood flow. You have got a big stream filling a small reservoir. In resting man skeletal muscle and adipose tissue receives about 25% of blood flow, yet is 75% or perhaps more of the nitrogen capacity. A very large reservoir being serviced by a small stream. So again we have got another two curves to interpose. In fact we really have a family of curves for all different areas of the body, related to the rate of blood flow, or the perfusion factor, as well as the percentage of fat in a given tissue or area of perfusion.

So, in 1906 Haldane had an empirical observation that you do not want to see a relationship of more than 1.58-1 in terms of excess gas pressure to barometric pressure. But you would like to know what percentage of saturation you have on a given exposure. What you seem to come up with, just considering three factors, is a whole family of curves. So how do you estimate where you are at the end of a given period of time and a given pressure, if you have got an almost infinite number of uptake relationships based on perfusion, physical factors and so on?

Well, in 1906 Haldane could not do it, and I am not sure that we can do it. So Haldane said "To hell with it, I can't do it" and he established a mathematical model. Haldane used the half-time equation which describes an exponential curve. This is a nice way of building exponential curves. You can produce a whole family of curves in this manner, by assigning either estimated, measured or guess work units of time to this equation. The half-time equation says that in one unit of half-time you reach 50% equilibrium. A unit of half-time is not defined in seconds or minutes or hours, it is unitless. It is the time taken to reach 50% equilibrium. With radioactivity 50% of what you are dealing with is decayed in one unit of half-time. If you are dealing with nitrogen uptake or elimination, you will take up or eliminate 50% in one unit of half-time. In the second unit of half-time, you go 50% of the remaining distance as it were, or a total of 75%, and so 87.5% in three units of half time, 93.75% in four, and right up the line. Of course the equation says that you never get where you are going. If you only go half the distance with each step you never reach 100%. But you get close enough for government work and diving and things like that. In practical applications six units of half-time brings you nearly to 99% equilibrium. Generally speaking, beyond six units half-time, you consider a theoretical compartment to be in equilibrium with the inert gas pressure that you are exposed to.

Haldane, using this totally theoretical mathematical model, and an empirical observation that animals could tolerate an overpressure of inert gas, expressed as a ratio of 1.58-1, now had the ability to build a decompression schedule. Haldane assigned time constants, theoretical compartments, to this model to build his tables. He assumed that there was a vast component, a vast tissue, that would have a half-time of five minutes. That is one unit of half time would be five minutes, two units would be ten minutes, and so on. Then he had slower compartments with half-times of 10 minutes, 20 minutes and 40 minutes and the slowest compartment in his model was 75 minutes. So that compartment would reach 50% saturation only after 75 minutes of exposure. Nobody has ever proposed that there was a distinct anatomical distribution for these half times. The general terms of fast tissues, slow tissues, medium tissues have been bandied around.

In an exposure to 4 atmospheres of absolute nitrogen pressure for 40 minutes, we take the half-time of five minutes and divide the time of the exposure, 40 minutes, by that half-time and that gives you the units of half-time for that particular compartment. Forty divided by five is eight units. This tissue has been exposed with a 40 minute exposure, eight units of half-time. Remember that beyond six you consider it 100% saturated. So the 5 minute compartment in 40 minutes would have 4 atmospheres of nitrogen pressure. The ten minute compartment in a 40 minute exposure would be 4 units of halftime, so that we would have 93.75% of 4 atmospheres, close to 4 atmospheres of nitrogen pressure. The 20 minute compartment after 40 minutes of exposure, 40 divided by 20 is 2, has been exposed for 2 half-time units. That is it would be 75% saturated so would have a nitrogen pressure of 3 atmospheres. The 40 minute tissue with a 40 minute exposure, 1 unit of half-time, so 50% saturated, which is 2 atmospheres of nitrogen pressure. The 75 minute tissue would obviously be something less than 50% saturated.

The equation allows you to determine, minute by minute, second by second, or even millisecond by millisecond, the percentage saturation of these theoretical compartments as a function of time. After your exposure for 40 minutes, to four atmospheres you are ready to decompress. The 5 minute tissue has 4, the 10 minute tissue something less than 4, the 20 minute tissue has 3, the 40 minute one has 2, and the 75 minute one has something less than 2, atmospheres of nitrogen pressure. You head towards the surface. Haldane said that you can continue upwards until the nitrogen pressure divided by the barometric pressure is no greater than 1.58. The tissue which has the highest nitrogen pressure as a result of this exposure governs your ascent. You do mathematical manipulation to find out what factor you divide by four to give you 1.58 and that is the barometric pressure that you must not exceed on your ascent. That is how Haldane built the decompression schedules in 1906, based on an assumption of half-times and an empirical observation that the animals withstood

this degree of excess gas pressure, this degree of supersaturation.

The deepest stop on the decompression schedule was a very short stop, because it was determined by the tissue with the highest pressure of nitrogen, which is always the fast tissue. Coming up from depth you reach a point where the five minute compartment has this critical ratio, so you come to a screeching halt. How long you have to stay there for that ratio to decay is determined by the rate constant, so the deep stops are always short because you are dealing with a fast half-time. You allow it to decay down to where it is safe for that compartment to make another ascent of 10 feet. Then you find that the next stop becomes a little longer, because the 5 minute tissue has decayed down to where it is not bothering you at all and now you are stopped by the next slowest compartment. As you get closer and closer to the surface, depending on bottom time, each stop is limited by one or two half-time tissues. As you get closer to the surface, you get slower and slower compartments. So the time that you have to spend at a decompression stop gets progressively longer, because the outgassing rate and the uptake rate are, in this model, equivalent. So it gets very lengthy. The longer that you are down, the more nitrogen you put into these slower compartments which is when the decompression profiles start to become disproportionate. The longer dives require progressively longer decompression because of the large gas flows into slower compartments.

In 1906 Haldane published his tables, and compared to what was going on before 1906, he solved a lot of problems. Prior to 1906, the decompression profiles were generally slow linear decompressions, as well as slow linear compressions. Much time in diving was wasted in getting to depth and getting back from depth and they were not solving the problems. Haldane introduced his tables and that seemed to solve the problems. It worked for a long time. I can not tell you exactly when the US Navy started diving using the Haldane tables but they did. They were satisfied with them for a while, but Haldane's tables were fairly limited in terms of the range of depth. He did not go beyond 165 feet or 6 atmospheres.

The US Navy had requirements to go beyond Haldane's tables in terms of depth and time. So working with the Haldane model, they started to explore beyond Haldane's tables and eventually produced a set of their own tables. I am not sure of the date of them, they were before the ones we now call the US Navy standard tables. Using the Haldane model, they produced a set of tables that were properly tested at the experimental diving unit to an end point of zero bends. If somebody bent they went back and looked at the model, found out which tissue had the highest calculated pressure in it compared to barometric pressure and did a little cut-back here and there. Then they tested again and if they got by with it that was an established schedule. Then it went out to the fleet. These tables that I am talking about are the old US Navy tables that were superseded back in 1955. If you come across them you should recognise them because they have an ascent rate of 25 feet a minute instead of 60 feet a minute. In the depth and time columns they had something starred which was called an optimum time for a given depth. In those days they did not have repetitive diving tables. For repetitive diving you simply added the total time of each successive dive.

In the 1950s the US Navy decided to look at the tables again. There were many factors involved, one of which was probably some percentage of bends cases occurring on the old tables, also scuba had come into the military hardware. Scuba although giving greater mobility is short on duration, so the only way to get work done was to build a repetitive dive system. So they had to go back to the drawing board, the Haldane drawing board, and generate new schedules of standard decompression tables and repetitive dive tables and exceptional exposure tables all about the same time.

The next paper will take you to the US Navy tables circa 1955. They found that they had to extend half-times beyond 75 minutes. Within the standard US Navy tables the slowest half-time considered is 120 minutes and that is also the basis of the repetitive dive tables. The exceptional exposure tables go up to a half-time of 240. In saturation diving I have seen some ridiculous numbers like 360 to 720 minutes used. It is beyond imaginable physiology to have a tissue that has half-time of 750 minutes. How can you provide enough oxygen for it to survive if that is the perfusion? The model starts to break down with saturation diving. Empirically it was found that there was a different ratio for different halftimes. That is what the current tables are based on.

You probably know more about the theory of anaesthetic uptakes and so on than I do and I am not sure that any half-time pattern model is a valid model at all. Things are too dynamic to handle by a half-time concept. There are different models, some work as well, some maybe work better. If you get conservative enough in your approach to decompression, then anything works. When you are working at a close edge it is interesting then that this model has as good a record as it does. I am not sure that we really know how to describe what happens with gas uptake and elimination in terms of diving exposure.

Navy tables are based on half-times for 5, 10, 20, 40, 80 and 120 minutes in the range of the standard tables. The repetitive dive system is based solely on the 120 minute tissue. The exceptional exposure tables include half-times of 120, 160, 200 and 240 minutes. Other models do not use half-times. The Royal Navy model is not based on halftimes. The Swiss tables are based on halftimes. The Canadian model, instead of having parallel compartments as in the USN tables is a series model, where one theoretical compartment bleeds into another, which complicates it. None of them are absolutely proven as being physiological. They are models by which they approach decompression, empirically tested.

Question:

How much fine tuning did Haldane have to do after he worked out his figures? How much changing did he have to do between the theory and when he actually ended up with tables?

Dr Bruce Bassett

The theory evolved from Haldane's empirical observations. He did not do a whole lot of fine tuning. The next presentation will show how the US Navy fine tuned it. From there is the practical side, how good are they, what do the statistics look like and, maybe, what is coming down the pike, maybe that will be next year's meeting with Brian Hills.

ANOTHER ALUMINIUM CYLINDER EXPLOSION

WARNING FROM SOUTH AFRICA
CYLINDER EXPLOSION INQUEST AND ENQUIRY

On 29th October 1981 there was an Inquest Enquiry held into the circumstances surrounding the accident on 18th March 1981 which involved JS Jolly. It was found that the aluminium cylinder which exploded had been damaged thermally due to excessive heat during a repainting procedure.

The investigation, held in the Durban Magistrate's Court, reminded those using or filling such cylinders that:-

1. Aluminium cylinders which have been subjected to the action of fire of exposed to temperatures in excess of 177°C must be condemned and destroyed. There is no reconditioning allowed.

2. Any aluminium cylinder which shows any defects in this regard requires verification by hydrostatic testing.

3. All concerned are advised, in their own interest, to adhere strictly to these rules.

(From Barologia Newsletter, March 1982)

DIVING MEDICINE IN DEPTH

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Brochures for each course will be distributed approximately five months before the starting date. Early registrations are encouraged as space is limited at each location. To ensure a place in the course of your choice, write to Human Underwater Biology enclosing a deposit of US$50.00 per person. The full course deposit of $200.00 per person is payable within ten days of receipt of the course brochure.

A CURRENT STORY

Officials at an aquarium in British Columbia are said to have two one metre long South American electric eels in one of their display tanks. This Christmas they decided to combine salesmanship with a practical application of the special powers of their exhibits. They placed two electrodes in the tank and connected the circuit to include the lights on a Christmas tree near the tank. Each eel is said to produce 100 watts of power, rising to 300 volts if they are particularly agitated. While calmly swimming the electric output is steady. Power increases as they actively hunt.

The electric discharge from the Australian Electric Ray are said to provide one diving instructor (at least) with innocent (?) amusement. He will suggest that diving pupils touch such creatures in his diving area, then shows that he suffers no ill effects, having waited till the ray's charges are much reduced. Former students wait patiently for him to pick the wrong ray one day.

SCUBA DIVING AND ASTHMA

A Proposed Investigation at Royal Prince Alfred Hospital Sydney.

It has long been the teaching of both diving instructors and of doctors involved with Diving Medicine that any history of "asthma" was an absolute bar to acceptance of such a person for exposure to hyperbaric conditions, in particular to compressed air diving. The reason for this was the belief that such people were at particular risk of suffering pulmonary barotrauma should they need to make an emergency ascent. There is reason to believe that many people have ignored such advice and dived, few coming to medical notice from asthma-related morbidity. Such cases as have been identified seem to indicate that danger, if it occurs, arises from suffering asthma in a diving situation rather than from pulmonary barotrauma.(1,2)

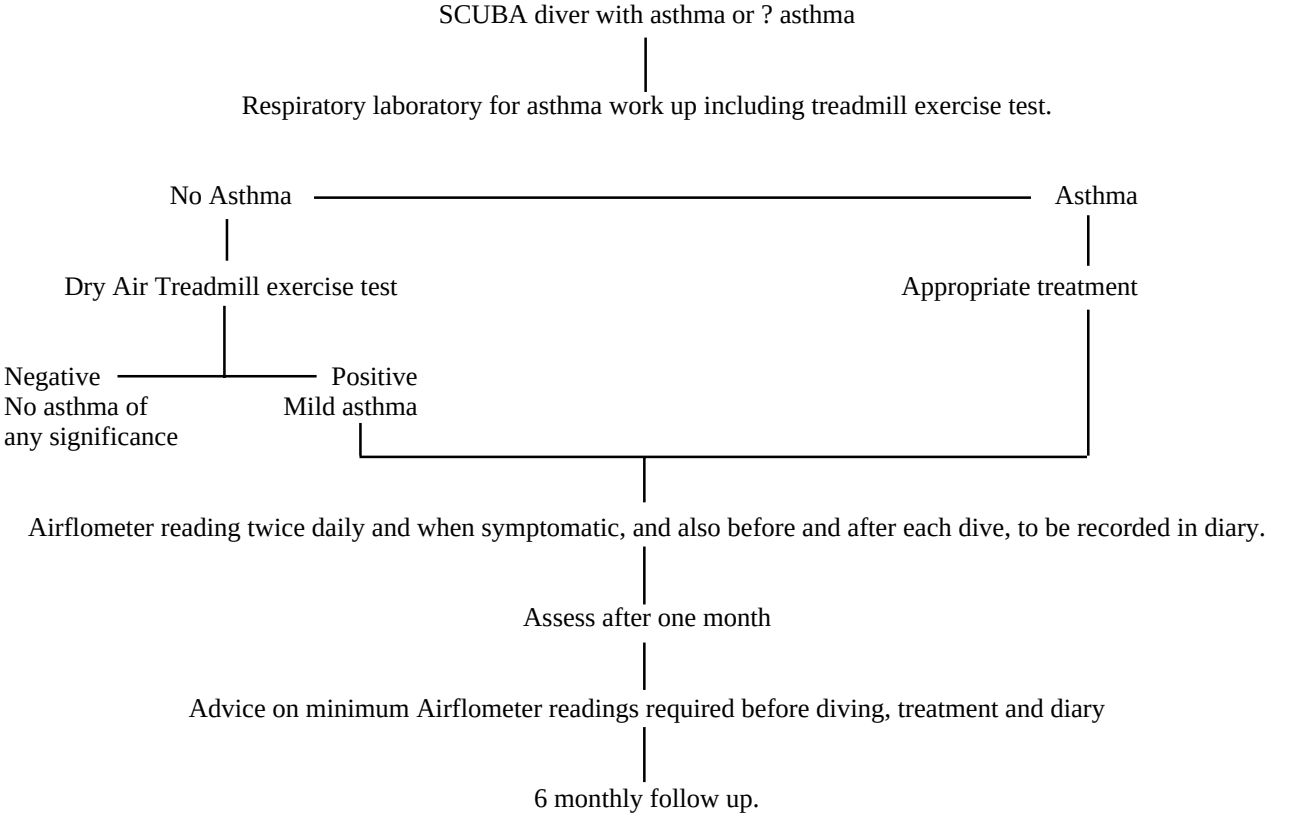
That some chest physicians have felt able to accept certain carefully assessed asthmatics as fit to scuba dive has not passed unnoticed:

> "The reprehensible tendency of some physicians, and often the respiratory specialist, to recommend that an asthmatic takes a bronchodilator prior to his dive, has nothing to commend it. It ignores the facts that (1) the asthmatic is far more susceptible to pulmonary barotrauma, (2) the aerosol bronchodilator may relieve some, but not all, areas of airway resistance, (3) the inhaler will be very effective in allowing the person to descend while breathing relatively normally, but is less effective at the end of the dive when the emergency free ascent is more likely, (4) despite the pharmaceutical literature, most sympathomimetic drugs do have arrhythmogenic effects, aggravating a small but appreciable hazard that already exists in the diving environment".(3)

However true some of these statements may be, they are unfortunately based on slender documentation of actual incidents and cannot be taken as putting the matter beyond legitimate debate. The acquisition of a greatly increased store of information is necessary before an accurate picture of the risks of asthma in a diver can be validly prognosticated.

It is proposed to offer asthmatic divers and would-be divers, a full assessment of their respiratory function and the apparent risks they run if they scuba dive. It is hoped that the CONFIDENTIAL nature of the project will encourage divers to make a full disclosure of their asthma problems and their diving experience, in particular any influence of one upon the other. The investigation is offered as a service to those presenting themselves and a basic phase in improving our understanding of the significance of the different types and severities of "asthma" in persons wishing to undertake scuba diving. The following protocol is presented for discussion:-

PROTOCOL FOR SCUBA DIVERS WITH ASTHMA



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Anyone with an asthma-diving history or who has knowledge of such persons (or of incidents where asthma and diving have co-existed) is asked to contact either Dr Peter Corte or Dr Douglas Walker.

Dr Peter Corte
Respiratory Lab.
Royal Prince Alfred Hospital
Sydney NSW

Dr Douglas Walker
PO Box 120,
Narrabeen NSW 2101

ASTHMA AND DIVING

Paul G Linaweaver

Because of conflicting opinions among experts regarding the asthmatic individual and diving, an in-depth discussion of the problem is presented.

One definition of asthma is as follows: "Bronchial asthma is a type of pulmonary incompetency due to constriction of the bronchi and oedema of the bronchial mucosa caused by the response of a susceptible bronchial tree to specific, allergic and/or non specific irritative stimuli".(1) Today there is no doubt that allergy plays an important factor in those patients whose asthma attacks are elicited by exposure to antigenic substances such as pollens, spores, animal hairs or dander (allergens). This type of asthma is often called "extrinsic asthma" and is common among young and otherwise healthy people. In many patients with asthma, however, an association of symptoms with allergen exposure cannot be demonstrated. Such patients, who suffer from this condition, are often classified as having "non-allergic asthma.(2)

In asthma, airway resistance, mainly reflecting large airway diameter, is increased and maximum expiratory flows, which reflect small airway diameter, are decreased. Thus, both large and small airways appear to be affected. However, it is probable that small airway obstruction predominates.

In a recent study from the University of California, San Diego, Wagner, et al. measured ventilation/perfusion relationship in asymptomatic asthma patients.(3) Their study showed that in some of the asymptomatic patients, as many as one-half of the lung units lay behind completely closed airways and had very low VA/Q ratios as a result of collateral ventilation. They demonstrated that even though standard pulmonary ventilatory function studies may appear grossly normal (because one is measuring predominantly the large airways or 80% of the ducting system), small airways may be closed. One cannot tolerate, in a diving situation, respiring with closed airways.

The possibility exists of developing acute, symptomatic or asymptomatic bronchospasm in presensitized or predisposed airways by a variety of stimuli, including specific allergens, infection, chemicals, physical stimuli, such as an aerosolized spray of water from a regulator or temperature changes. Psycholphysiologic effects affecting the vagus and other portions of the autonomic nervous system can cause bronchospasm (4). Hypocapnia caused by the hyperpnoea of exercise can cause bronchoconstriction and could be compounded by the hyperventilation of the panicking diver (5). Airway muscles hypertrophy and increased responsiveness exists in these typical individuals, and, indeed, an increase in tone of airway muscle causing airway constriction and development of asthma can occur whether the individual is aware of it or not.

In acute asthma, vital capacity will decrease even in the presence of a normal or increased total lung capacity due to increase in residual volume. At residual volume, we can assume that the small airways are virtually closed with a resistance approaching infinity. The individual is then forced to breathe at an increased functional residual capacity (FRC), closer and closer to the total lung capacity. This causes dyspnoea.

Overpressurization accidents require not only overpressurization, but overexpansion of the lung. The asthmatic, breathing at an elevated FRC near TLV, has already lost the margin of safety before overexpansion occurs. These factors result in increased susceptibility to overpressurization and the development of the triad of pathology; pneumothorax, mediastinal emphysema and traumatic arterial gas embolism.

I do not think there is any informed physician who would say that an active asthmatic should dive. Should the individual who "grew out" of his asthma be allowed to dive? Based on experience, the answer is NO unless the individual is equipped to carry his own recompression chamber wherever he goes.

The individual who has not had an asthmatic attack since childhood will, frequently, wheeze during forced expiration and not be aware of it. Pulmonary function studies will show decrease in the expiratory phase of the flow volume curve, and in particular, the maximal mid-expiratory flow (MMEF 25%-75%) will be diminished.(6) To re*peat the test with bronchodilators may prove fallacious as many of*

the smaller airways, smaller than 2 mm in diameter which have few muscle fibres, may not respond to the bronchodilators. It is always tempting to give in to these usually young, bright, enthusiastic individuals, but, the risk is too great. Once an asthmatic, always an asthmatic.

If the diagnosis of asthma is in doubt or if the patient is asymptomatic, a positive histamine and/or methacholine test substantiates its existence. In asthmatics, hyperreactivity to methacholine persists for years, even in the absence of active asthma. In proper hands, this test is a safe diagnostic tool.(7) A positive test contraindicates exposures to increased pressures which could lead to unacceptable risk for pulmonary overpressurization accidents.

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COMMENT

The case reported by Dr Clinton-Baker (page 17) suggests that asthmatics needing treatment to stay symptom free should not dive. Yet many do dive successfully. Should opinion or fact govern medical advice? It is to provide facts that the Royal Prince Alfred Hospital investigation has been started.

PATHO-PHYSIOLOGICAL CONSIDERATIONS OF HYPERTHERMIA IN HYPERBARIC ENVIRONMENTS

Ian M Calder

During the past decade hypothermia as a cause of death in the European sector of professional diving operations has become recognised. However, hyperthermia as a problem was reported by Cox et al.(1) in which six fatal accidents were cited, and undoubtedly a number of unexplained deaths could be attributed to this. The problem has been identified at present as occurring in the decompression chamber rather than in the working environment at depth.

HYPERTHERMIC SYNDROMES

Adolph (2) identified several heat disorders but for the purposes of the hyperbaric environment only three need be considered. These are:-

1. Water deficiency heat exhaustion, described by Black et al.,(3) and caused as a result of lack of drinking water intake in a hot environment.
2. Hypochloraemic (a salt deficiency) heat exhaustion due to inadequate replacement of salt loss described by Ladell et al.(4) and leading to dehydration and reduced blood volume.
3. Heat exhaustion due to physical exercise in a hot environment with sweating but without replenishment of salt or water.

PHYSICAL CONSIDERATIONS

To appreciate the dynamics of the induction of hyperthermia in a compression chamber it is necessary to examine the thermodynamics of the environment. The adiabatic mathematical formula gives the theoretical background to the induction of high temperature in a chamber. The equation for this is:-

$$T_2 = T_1 \quad \frac{(P_2)}{(P_1)} \frac{8 - 1}{8}$$

where T_1 is initial and T_2 is final temperature in degrees Kelvin (=°C + 273), P_1 is initial pressure and P_2 is final pressure. d is the ratio of specific heating; for air d - 1.4. Therefore:-

$$\frac{\gamma - 1}{\gamma} = 0.2857$$

Thus, if the initial temperature is 32°C

$$T_1 = 305°K \text{ (ie. } 32 + 273 \text{ degrees absolute)}$$

and if a fourfold pressure rise is induced

$$\frac{P_2}{P_1} = 4$$

$$T_2 = 305\ (4)\ 0.2857$$

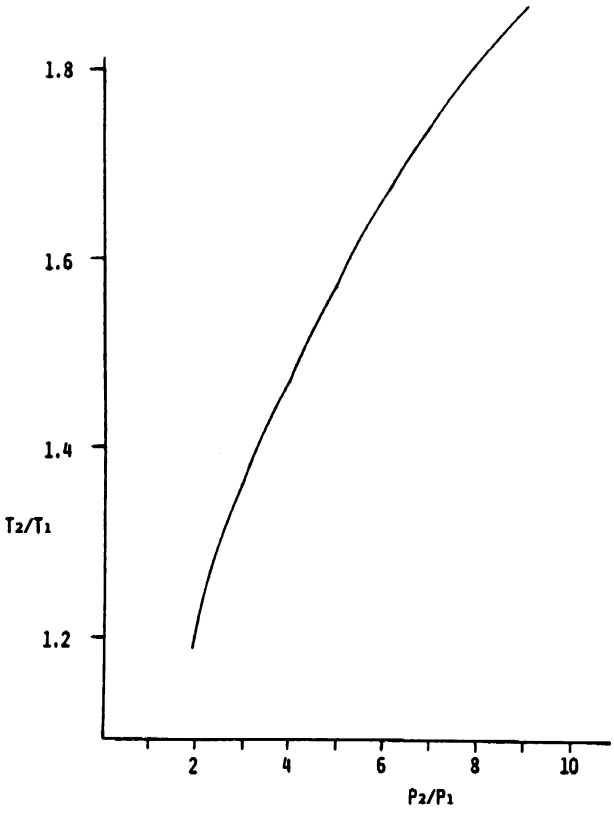
= 453K
= 180°C

When such changes are plotted on the graph, this shows the relationship between T_2/T_1 and P_2/P_1 which when expressed in actual terms shows theoretical temperature rise for example:-

Pressure Rise (in atmosphere)	Initial Temp. (°C)	Final Temp. (°C)
2	10	90
8	40	300

FIGURE 1

THE RELATIONSHIP BETWEEN INITIAL AND FINAL TEMPERATURES FOR MULTIPLES OF INITIAL PRESSURE



This calculation does not make any allowance for physical influencing factors which may cause considerable variations in the final temperature and environmental conditions reached. These are:-

1. The surface temperature of the chamber. In cold climes this will reduce the theoretical final temperature. However, a more serious aspect is the situation in which the chamber is exposed to tropical sun, thus giving an additional radiation heating effect.

2. The speed at which the chamber is compressed. This has a profound influence, in that a rapid increase leads to a large number of calories being introduced into the chamber with consequent environmental temperature rise, which cannot be reduced by radiation from the surface.

An additional dimension to be considered in connection with the physiological effects of these two factors, is that of relative humidity. When the environmental temperature reaches or exceeds 37°C this assumes greater importance. At and above this point, the control of the body temperature is dependant upon the loss of fluid from the body surface by evaporation and cooling is due to the mechanism of latent heat of evaporation. It necessarily follows that as the environment temperature increases above 37°C, together with that of the relative humidity approaching 80%, the cooling mechanism fails and body temperature rises which, outside narrow limits results in irreversible changes. Heat transfer to the body core is necessarily greater in an oxyhelium environment, which compounds the effective rise in body temperature.

CLINICAL ASPECTS

Hyperthermia may pass unrecognised and unappreciated unless it is considered in the differential diagnosis.

When considering the thermodynamics of the hyperthermic situation it is not necessary to have a high initial temperature to induce the unphysiological final temperature, as is shown by reference to the temperature pressure graph.

This has been shown by two deaths in the temperate climes of Northern Europe following rapid compression. However, mere emphasis has to be placed on the adverse effects of rapid compression in tropical and sub-tropical climates, both from personal experience and that of King (unpublished), which has resulted in the total of six deaths. The awareness of such a condition could in retrospect be an acceptable cause of two otherwise unexplained deaths during recompression.

All eight cases had shown signs which were clinically compatible with decompression sickness, and this was the rationale for recompression. The subsequent change and development of symptoms were not those of decompression sickness. These ranged from mental confusion and aggression, anxiety, absence of sweating (hot dry skin), to circulatory collapse. Although it is impossible to generalise from a small series of cases, in the three incidents where more than one person was involved, and there was a survivor, it is apparent that the obese have a smaller chance of survival. This would correlate with the reduction of weight to surface area ratio, with consequent lower heat loss.

Case 1

A medical assistant and a doctor entered a recompression chamber to attend a diver with pulmonary barotrauma. They were compressed to 60 m (equivalent depth sea water) on a 23/77 mixture of "Heliox". The ambient temperature was approximately 35°C and the humidity 85%. The medical assistant was an obese West African, 153 cm tall; the doctor was lean and 180 cm tall. The medical assistant remained lightly clothed, the doctor

removed all his clothing and drank copious quantities of fluid while the medical assistant declined to drink. After 26 minutes the medical assistant was so distressed that he entered the outer lock to decompress, but he still drank nothing. He became increasingly distressed, and was observed picking at his clothes in a confused state. When he left the chamber he collapsed. After about 15 minutes he was recompressed to 42 m, at which time he was stuposose with an irregular almost impalpable pulse of about 120/minute and a respiratory rate of 50/minute. He was given 500 ml 15% dextrose with 20 mg dexamethasone but he died some 5 and a half hours later. At necropsy the body and tissues were noted to be very dry with no interstitial fluid. The CSF sodium was 174 mmol/l (nominal mean 127 in Naumann's series and 131 in a personal series of ten cases). There were no other gross abnormalities.

UNLIKE HYPOTHERMIA, HYPERTHERMIA IS AN IRREVERSIBLE AND RAPID PROCESS, AND ONCE PROTEIN COAGULATION OF VITAL CENTRES HAS OCCURRED, DEATH IS INEVITABLE.

POST MORTEM CONSIDERATIONS

Specific tissue changes developing as a result of acute hyperthermia have not been established, although degeneration of crypt epithelium of the small intestine has been suggested as an indicator. The time sequence is, however, usually too short for the presence of any changes to be established. However, the effects are essentially biochemical, which would not produce morphological changes.

Blood chemistry is an ideal parameter to measure during life, but rapid and variable changes occur after death. Electrolyte measurements of vitreous humor by Hughes (5) and cerebro-spinal fluid by Naumann (6) have shown that these levels are relatively unaffected during the first few hours after death. Taken in the proper context, examination of these parameters may give some indication of the electrolyte status immediately preceding death. However, Cooper et al.(7) make the important point that the least affected is the sodium content, but potassium is subject to some variation.

The use of this technique has been usefully applied in six cases of suspected death caused by hyperthermia. The normal figure of 127 mg.% quoted by Naumann (6) closely agrees with the mean figure of 131 mg.% obtained from a personal random autopsy series of ten cases. All samples were by needle aspiration of ventricular fluid and preservation in lithium heparin tubes before analysis.

The environment results essentially in a pure water depletion with a consequent rise in CSF sodium ion content. From the proven series of six deaths from hyperthermia levels of the sodium ion ranged from 141 mg.% to 181 mg.%. With this range it is apparent that such measurements cannot be regarded as definitive scientific diagnosis of hyperthermia. Such results have to be considered in the overall findings of the case, and must be regarded as a useful adjunct. During dissection, the impression may be formed of some desiccation of body tissues, which taken with the clinical history can lead to a constructive clinico-pathological conclusion.

DISCUSSION

Hyperthermia in a hyperbaric environment is a condition easily produced even in the more temperate climates. However, without an awareness of the speed and ease which such a condition may be produced, this may be omitted from the clinical differential diagnosis. As a result the condition may be worsened by the very nature of the treatment, in which the bizarre symptoms may be judged as being due to decompression sickness rather than to electrolyte disturbance from hyperthermia. It is certain in one case that such misinterpretation has resulted in a further recompression resulting in further deterioration of the patient's condition.

Treatment, once diagnosis has been made, must be symptomatic. The object is to reduce the environmental, and consequently core temperature, and restore the electrolyte balance with appropriate intravenous therapy.

ACKNOWLEDGEMENTS

Thanks are due to Professor MMR Williams of the Department of Nuclear Engineering, Queen Mary College, London, for help with the thermodynamics, and Miss Mandy Collins of Department of Morbid Anatomy, London Hospital, Whitechapel, for help in the preparation of the typescript.

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THE DIFFERENTIAL DIAGNOSIS OF A CASE OF UNDERWATER EAR PAIN

Noel Roydhouse

This is a description in chronological order of the way an underwater diver may present to the diving specialist doctor (DSDr). It demonstrates the unwitting failure to disclose vital information, that is not uncommon in a lay person not medically educated and not uncommon in a diving professional (DP).

Day 1: 7.30 pm

Situation. DSDr in spa pool in backyard. DP in Singapore. Abbreviated conversation on telephone when DSDr summonsed by the blinking red light overlooking the pool.

DP: "The ENT surgeon wants to do a bilateral Caldwell-Luc (a radical antrostomy) on my sinuses because I am having difficulty in clearing my ears when diving. Do you agree?"

DSDr: "Describe your problem." Note that the description at this stage differs from what DP describes later.

DP: "My nose runs and it blocks and I have had increasing difficulty in clearing my ears so that I get a lot of pain."

DSDr: "What did the X-rays show?"

DP: *"I do not know really but it showed a lot of white."* This could be opaque antra on sinus X-ray and indicative of sinusitis. Note the confusion that exists between most lay people between "sinus" trouble which is actually a 1920 folk-medicine misnomer for nasal catarrh ie. nasal blockage and nasal discharge, and the condition of true purulent sinusitis. There is an enormous difference.

DSDr: "Well in New Zealand I do not advise a Caldwell-Luc operation, (a radical type of operation) at the first consultation."

DP: "What should I do?"

DSDr: "Use a nasal spray and Sudafed tablets and see how you get on."

DP: "OK. I will fly back if I am no better."

Day 5: 9.30 am

Situation: DSDr doing some minor surgery. DP at the Auckland International Airport asking for and appointment and given one for 2 pm.

Day 5: 2 pm

Situation: Both in surgery. Interrogation resumes.

DSDr: "What actually happens when you dive?"

DP: "When I clear my ears I can feel the air go into my ears but the pain persists in my right ear. I used to be able to clear my ears easily but it has become more difficult over the past few months. I do not get colds but my nose blocks up at times especially on the right side and it runs a lot."

DSDr: "What sort of stuff comes out of your nose? Thick or thin, clear or cloudy?"

DP: 'Thin clear fluid."

DSDr: (To himself) Aha, thin clear copious fluid you do not get with sinusitis, you should have some at least cloudy and there is not usually much discharge. Blockage is usually on the side of the sinusitis. DSDr then carries out an examination. The ear drums are both indrawn and more so on the right side. (This is a probable indication of spasm of the right tensor tympani muscle whose nerve supply is the same as the temporalis muscle and the medial pterygoid muscle, both elevators of the mandible against the maxilla). The medial pterygoid muscle is palpated, (a finger pressing medial to the ramus behind the last molar tooth) and the right one is very tender compared to the left side. There is a minor wear of the lower incisor teeth indicative of a tendency to grind the teeth.

DSDr: "Do you grind your teeth?"

DP: "Yes I do."

DSDr: A grunt, and then examines the nose which is very clear, ie. there is a good airway on both sides with a slight deviation of the nearly straight nasal septum to the left with compensatory hypertrophy of the right inferior turbinate, ie. the right inferior turbinate was larger than the left and this explained the complaint of greater nasal blockage on the right. The middle meati into which the maxillary sinuses open, are clean and clear, which state is not expected with sinusitis. There is no pus or purulent exudate in the middle meati, on the inferior turbinates or on the floor of the nose. Provisional diagnosis to this stage is vaso-motor rhinitis with a preponderance of the parasympathetic effect, ie. copious thin nasal discharge and the mandibular dysfunction syndrome (MDS), ie. referred subclinical pain to the right ear brought to subjective levels by exposure to cold water. Note that in Brunei at 30 metres the water is quite cold. Other symptoms elicited to back up the MDS were the blocked feeling in the right ear, popping noises in this ear and sometimes the feeling that there was water in his ear ie. that there was a feeling of water in his ear when he knew there was no water there. The proviso at this stage was that it was necessary to have his sinuses X-rayed especially as this examination was what had prompted the advice that he needed a radical antrostomy operation ie. a Caldwell-Luc operation in which a large opening is made between his maxillary sinuses and the lower part of the nose.

PLAN OF ACTION

1. Cauterize the right inferior turbinate, which was done. He said at this stage that in Hamilton an ENT surgeon had done this five years ago and that he usually fainted when he saw a needle.

2. Arrange for a sinus X-ray. This was carried out at 3 pm.

3. Arrange for a dentist to examine and treat any interferences. Lower left molar interferences were treated at 3.30 pm. Interferences are where natural teeth cusps are too upstanding and interfere with easy side to side movements of the teeth and promote grinding of the teeth which in turn brings on spasm of the mandibular muscles which in this case was the medial pterygoid.

Day 5: 4 pm

X-ray of sinuses (inexplicably at this stage) showed that the maxillary sinuses were opaque, this being the usual indication of a purulent sinusitis, so an antral lavage was set up to wash out the maxillary sinuses to determine whether there was pus or not in the sinuses.

DP: "So I have infected sinuses after all?"

DSDr: "Not until I see pus. Seeing is believing." (with a slightly lessened sureness of the provisional diagnosis). To do a wash out of the sinuses, in addition to the topical anaesthetic, a reinforcing injection of local anaesthetic is given into the mucosa of the inferior meatus under cover of the inferior turbinate, using a 5cm gauge 26 Schimmell needle on a dental syringe. At this stage some difficulty occurred in finding the bone of the naso-antral wall.

DSDr: "What sort of operation did the Hamilton ENT surgeon do to you?" (The undisclosed information).

DP: "I do not really know, but my nose bled for days afterwards."

DSDr: (To himself.) Aha, and so ho. What is going on here? Did the surgeon do it or did he not? So a probe was passed into the inferior meatus and sure enough on both sides were found the antrostomies or the holes made by the ENT surgeon from the nose into the maxillary antra for the operation of intranasal antrostomies. Even before the penny could drop the X-ray result was explained. After any antrostomy operation, be in intranasal or a Caldwell-Luc, the sinus mucosa becomes thickened and shows up in X-rays as an opacity. Every ten years I get caught out, but only temporarily. A re-examination of all the X-rays substantiates the thought and the findings of a previous sinus operation.

DIAGNOSIS

1. Vaso-motor rhinitis.

2. Mandibular dysfunction syndrome.

3. Previous intranasal antrostomy.

PLAN OF ACTION

1. Cautery, already done on the right.

2. Interferences treated, already done.

3. Jaw muscle stretching exercises.

4. Librium.

5. Atrovent (Ipratropium Bromide) nasal spray.

6. Cautery of the left inferior turbinate.

7. Weekend with his parents down country.

REASONS FOR PLAN OF ACTION

1. Cautery of the inferior turbinates stops them from swelling up so much and improves the functional airway, ie. prevents nasal blockage. In four out of five divers it makes it easier to clear the ears.

2. Interferences promote tooth grinding and clenching causing spasm and incoordinate movements of the mandibular muscles. Referred sensations from the mandibular muscles are felt in the ears.

3. Jaw muscle stretching exercises promote normal movements and normal coordination of the jaw musculature. In this case he could not open his mouth without the lateral pterygoid coming into action and pulling his mandible forwards with a jerk, an unnatural movement. So he was trained to open his mouth without the forward protrusion of his mandible.

4. Librium has a specific effect on the chewing centre and reduces or stops nocturnal grinding and clenching of the teeth. It is given for a short (5 day or night) course to break the habit.

5. Atrovent (Ipratropium Bromide) is a parasympathetic paralyser as is its relative, atropine. Locally it has a strong effect on reducing nasal discharge. The other procedure to stop a rhinorrhoea is to do a Vidian nerve resection in the pterygo-palatine fossa behind the posterior wall of the maxillary antrum. The nerve carries the parasympathetic nerve fibres to the nasal mucosa.

6. Cautery of the left inferior turbinate to improve the nasal airway and cautery sometimes reduces the nasal discharge. With a non-congested nose, the patency of the normally cleaned Eustachian tube is improved. He had carried out the Toynbee test previously and this showed, at that time, normal Eustachian tube function.

7. Down country is a good place to relax and would give him time to carry out his exercises.

Day 8:

He returned from the country stating that his jaws were ever so much better though he had not complained before that his jaws were tight. The inferior turbinates were healing and his nose was clear of mucus. An audiogram showed that his hearing was normal. He flew back to Singapore the next day after a trial in a pressure chamber to check that he could clear his ears easily. He has reported no problems since then.

TRACING NITROGEN IN DIVERS' BODIES

Mary M Matzen
Naval Medical Research Institute

Development of a method for computing safer decompression schedules for Navy divers is a major mission of the Hyperbaric Medicine Program Centre at the Naval Medical Research Institute (NMRI) in Bethesda, Maryland.

To improve air decompression schedules, diving researchers need a better understanding of nitrogen uptake and elimination in the human body.

As part of a study of nitrogen exchange, NMRI researchers combined their expertise with that of researchers at the Lawrence Livermore Laboratory (Livermore, California) of the University of California in the fall of 1980. The purpose of the experiment was directly to measure where and how fast nitrogen enters a diver's body. The protocol required that the subjects breathe trace amounts of radioactive nitrogen.

Personnel and Rationale

Designers of the experiment were LCDR PK Weathersby, MSC, USN; Dr LD Homer and Captain ET Flynn, MC, USN, of NMRI; and physicist P Meyer of the Livermore Laboratory. The subjects were investigators and divers from NMRI: Captain Flynn; LCDR Weathersby; HCMS (DV) PC West; HM1 (DV) RJ Ebsen; HT1 (DV) M Gingrich; HT1 (DV) M Washington; and ET2 (DV) PA Holden.

The subjects were highly motivated, yet a bit apprehensive about the radiation exposure. Divers are accustomed to taking big risks in their work - they have been trained and are knowledgeable and competent in the hazardous diving environment. Breathing radioactive gas was a novel experience for all, but a source of particular concern to LCDR Weathersby and Captain Flynn, whose role was to ensure that the procedures were safe.

Many divers have at some time suffered decompression sickness (bends), resulting from bubbles formed in the tissues after rapid decompression. The offending bubbles are assumed to be caused by the inert gas in the diver's breathing mixture. The rationale around which most decompression schedules have been developed assumed that, under increased pressure, the diver's body absorbs excess nitrogen and that this excess nitrogen generates bubbles that form in the blood stream if the pressure is rapidly reduced. Adequate decompression schedules allow the excess nitrogen to be exhaled; thus inhibiting bubble formation and preventing the occurrence of decompression sickness. The crux of the problem is to define exactly how fast inhaled nitrogen enters and leaves parts of the diver's body.

Experimental Procedures

Previously, medical researchers have had to approximate the tissue uptake and elimination rates of nitrogen. To get a more accurate picture of nitrogen exchange in the body, researchers have had to use other gases which are more easily detected in the body (such as radioactive Xe 133) and assume that these gases behaved like nitrogen. The few experiments that attempted to measure gas uptake and elimination have generally used anaesthetized animal subjects and have used a short measuring time, such as 30 minutes. More importantly, gas exchange in a particular region of the body has not been studied because the techniques measured either expired gas or venous blood samples that contained mixtures of gas from all organs in the body. NMRI investigators sought to eliminate many of the assumptions which have to be made in these types of experiments by trying to measure nitrogen exchange directly in humans.

Nitrogen is hard to measure. It has no colour and no odour. It has no optical, magnetic, or nuclear properties that make it easy to identify. In the Livermore experiment, the subjects breathed a mixture of 20 percent oxygen and 80 percent nitrogen that included a small fraction of radioactive nitrogen ($^{13}N_2$). This nitrogen, which behaves chemically the same as normal $^{14}N_2$, could be traced anywhere in the subjects' bodies.

Physicist Paul Meyer has assembled the equipment and expertise that makes the Livermore Laboratory one of the few sites in the world where $^{13}N_2$ can be produced and studied in humans. The nitrogen for this experiment was prepared on-site because $^{13}N_2$ has a radiologic half-time of only 10 minutes. This means that a one hour delay in transporting would reduce the amount of radioactive gas by 98 percent. The $^{13}N_2$ is prepared by bombarding pure nitrogen with gamma rays (see explanation in Figures 1 and 2). This procedure was accomplished by remote control in a well-sealed accelerator area. Intensity of the rays was such that 1/300 second of exposure to the direct beam would be fatal. To get sufficiently "hot" nitrogen to serve as an effective tracer, a 50 minute irradiation time was required. A danger inherent in the procedure was the chemical risk that could arise from the possible creation of noxious gases during preparation of the radioactive nitrogen. However, gas monitoring showed that creation of such gases did not occur.

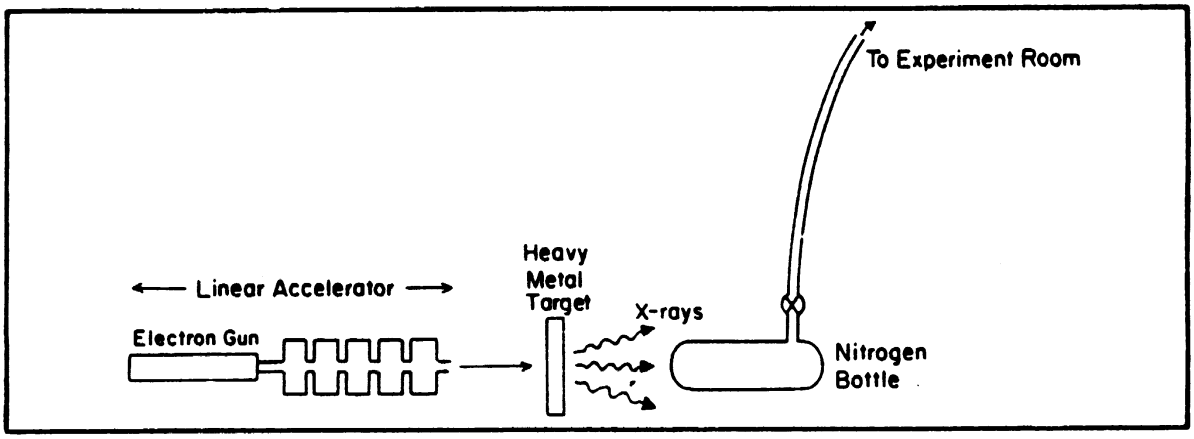
An unavoidable risk in this experiment was exposure of the subjects to ionizing radiation as they breathed the $^{13}N_2$ mixture. In fact, a major part of the researchers' time was spent in designing and monitoring the experimental procedures to keep the exposure at an acceptably low level.

During the 30 minutes that the subjects breathed the radioactive gas (at 1 atmosphere in these first experiments) and for up to 90 minutes thereafter, the location of the $^{13}N_2$ was identified and measured in their bodies as they lay inside a radiation device known as a positron gamma camera. The areas in the body of special interest were those most often affected by decompression "hits"; the shoulder, the knee and the inner ear.

In ideal circumstances, the information (image) obtained from a positron camera is three-dimensional in nature. However, these experiments were limited by the maximum

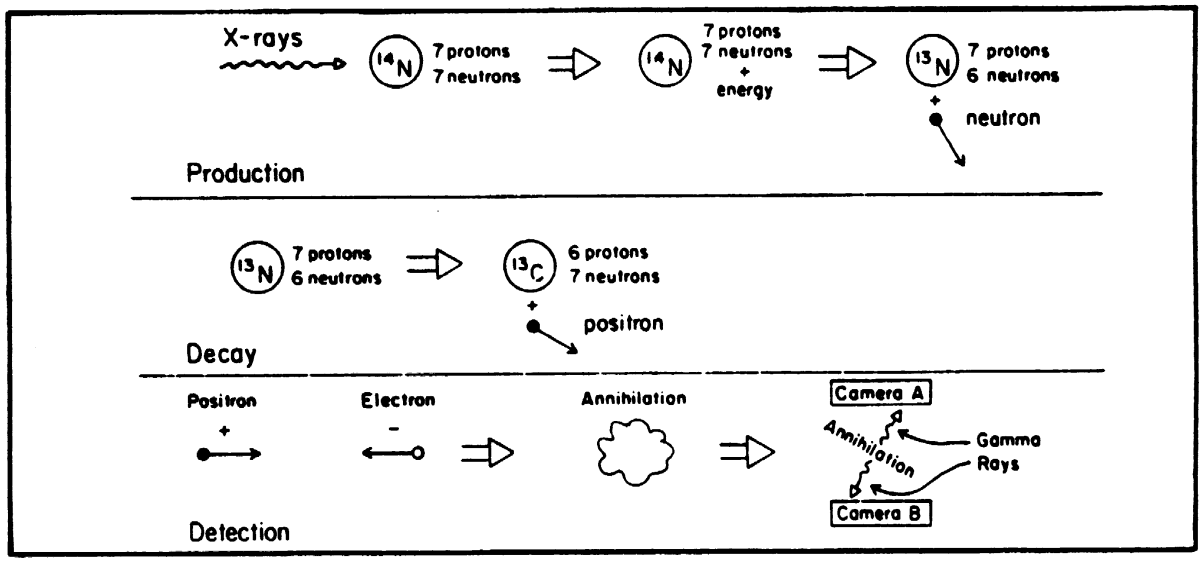
FIGURE 1

PRODUCTION OF RADIOACTIVE NITROGEN



Pure (stable) $^{14}N_2$ is pumped into a gas bottle in the accelerator cave (about 30 feet beneath the experiment room). Electrons are produced and accelerated to nearly the speed of light and to over fifty times their rest mass before they are scattered and their energy converted to X rays ("Bremsstrahlung"). The X rays bombard the nitrogen bottle and cause the nuclear reaction that produces radioactive $^{13}N_2$.

FIGURE 2
PRODUCTION DECAY AND DETECTION OF $^{13}N_2$



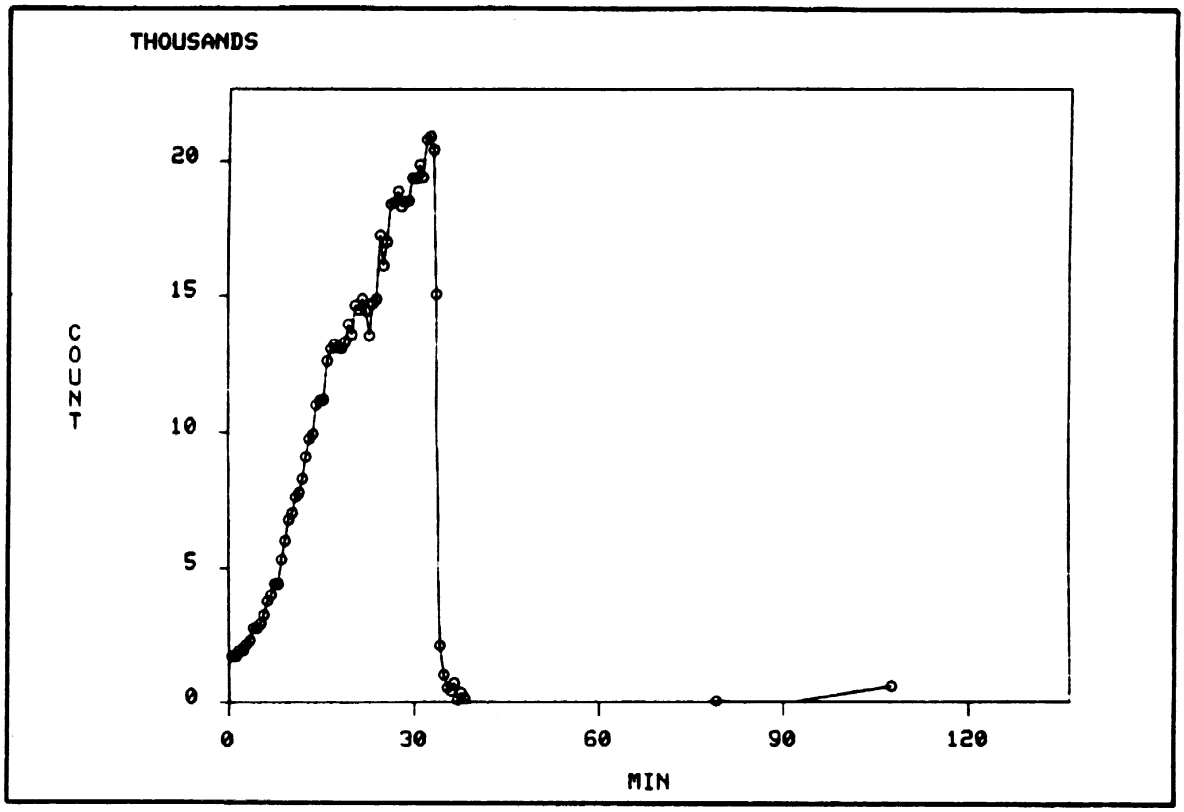
High energy X rays strike a stable $^{14}N_2$ nucleus, causing the ejection of one neutron to produce the radioactive isotope $^{13}N_2$. Within an average of 14 minutes the unstable $^{13}N_2$ decays to eject a positron - an electron with a positive charge. Because the positron is the corresponding anti-matter particle to the electron, it soon interacts with an electron, and both are annihilated. The positron and electron masses are converted into energy in the form of two gamma rays emitted in opposite directions. When their direction of travel causes both to hit the faces of the two gamma cameras, the location and time of the annihilation is recorded as one data point.

specified radiation dose to each subject. The "mere" 500,000 or so radioactive disintegrations recorded for each subject do not provide a perfect reconstruction of the "image". Extensive computer programming and operation are needed to "crunch" (or assimilate) the data points into a useful form such as shown in Figure 3. The data should provide estimates of comparative nitrogen capacity in the shoulder, knee and inner ear, as well as the average time and variations in the amount of time that nitrogen stayed in these areas.

Results

This long overdue and important study will provide real information about the way air behaves in the diver's body. NMRI researchers who are attempting to devise a better model for computing air decompression tables await this vital information. Conclusions can not be drawn until the data is analyzed; but the results of the experiment could change old theories or add new ones.

FIGURE 3



Graph of amount of radioactive nitrogen (count of number of $^{13}N_2$ disintegrations) plotted against time since the start of the experiment. This information was obtained from the shoulder of one of the NMRI subjects and has been corrected for the radiologic half time of ^{13}N. The sharp fall at 30 minutes followed the switch from inspiring the $^{13}N_2$ mixture to inspiring room air.

Comment

On a lighter note: The diver subjects reported disappointment that Livermore was not a bustling naval port. Their disappointment was off-set, however, when they found the local winery was suitable for an occasional "culturally inspiring" visit - an activity dear to a diver's heart.

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UTILIZING THE TEST OF PRESSURE IN SUSPECTED DECOMPRESSION SICKNESS

C Gordon Daugherty

Like many other diseases, decompression sickness in its earliest stages often presents an incomplete or ill-defined picture which may be difficult to distinguish from other medical problems or everyday nuisances. It is in this setting that the use of pressure may provide both the diagnosis of the disorder and its treatment. This is similar to the use of nitroglycerine as a diagnostic test in a patient who presents with chest pain. Should the chest pain be relieved, this medication not only helps determine the nature of the problem but also serves as the beginning of treatment. Similarly, a test of pressure is a means of diagnosing the disease of decompression sickness at the earliest possible moment, and helps avoid unnecessary treatment.

A typical situation may follow a dive in which a minor musculo-skeletal strain could have been produced. The diver often thinks he has "pulled a muscle" when the discomfort first begins. This discomfort produces difficulties for everyone concerned. From the diver's point of view, treatment of his minor discomfort will involve another long period of time in a chamber. However, he is also uncomfortably aware that decompression sickness can become quite serious, sometimes in rather short order.

From the standpoint of the supervisor, the diver's tentative complaint may represent an unwelcome distraction from his main task of keeping the work moving right along. He shares the diver's awareness that failure to treat decompression sickness early may lead to more serious problems later. The essence of the problem lies in the fact that many medical disorders begin as just-noticeable or minor symptoms. Wouldn't it be nice if there were a simple, reliable test taking only twenty minutes to perform which would distinguish a serious from a trivial problem?

In the case of early decompression sickness there is such a test.

The test consists of returning the diver to the chamber and taking him to a depth of sixty feet while breathing pure oxygen. Given the mild, early nature of the symptom in question, if it is due to decompression sickness it should be relieved rather quickly. If the symptom is not gone, or significantly improved after a period of twenty minutes, then it is probably not decompression sickness. Remember that the symptom in question is rather vague, elusive, or otherwise suspected not to be decompression sickness. Usually there is the suspicion that the diver is suffering from a simple muscle or joint strain and needs no treatment. If the diver has obvious pain in a joint there is usually no question about the nature of the problem and no need to do any sort of test. In my experience, the relief often comes well before sixty feet is reached, perhaps even at just a few feet of depth. If this occurs, then the question as to the nature of the symptom has been settled and the diver is treated according to the appropriate table. If recompression for twenty minutes has no significant effect on the symptom in question, it is probably another type of problem and the diver is returned to the surface. Since he is breathing oxygen throughout the test period, his decompression status is not affected adversely.

Some commercial diving supervisors have a strong belief that any recompression should be nothing less than a complete table. This may be because, in earlier times, treatments were often concocted on the spot. The diver received sufficient recompression to relieve all or most of his discomfort, but without resorting to scientifically formulated tables. This sort of inadequate treatment should not be confused with the test of pressure. The manoeuvre is not treatment when it is instituted and should not be interpreted as treatment by anyone reviewing the records of the dive. As mentioned earlier, it is analogous to the diagnostic use of nitroglycerine in a puzzling case of chest pain. Should the test relieve the symptom in question, it may then become part of the treatment which will be carried out. Should the test be negative - thus allowing the conclusion that there is no decompression sickness present - this is not inadequate treatment or any sort of treatment at all. Rather it is a direct and logical method of solving a puzzling and potentially serious problem. It reflects alertness and awareness on the part of the person utilizing the test.

A recent case in the Gulf of Mexico illustrates the use of this test. A commercial diver sustained a mild, pain-only bend on a Tuesday which was quickly and appropriately treated by his supervisor. He was then held out of the diving rotation for twenty-four hours (a matter of company policy) and returned to diving on Thursday, completing the job. He returned to shore the following day and noted mild but definite pain in his shoulder late Friday night which was still present upon awakening Saturday morning. In this case, the question was not the nature of the discomfort but rather the timing of it. Mild pain after this long a delay is supposedly not typical of decompression sickness. Nevertheless, a test of pressure was elected and the patient experienced total, near-instantaneous relief of pain as he was passing a depth of 12-13 feet.

Based on my experience with the disorder, I think atypical presentations of decompression sickness are more common than one sees with other diseases generally. I am much more suspicious than I used to be and much more ready to either treat, or at a minimum, utilize the pressure test. In the case just described, there was a time when I probably would have pronounced the pain muscular and not recommended decompression.

The thing that has educated me the most is that, the more I use the test, the more peculiar things I see being relieved by recompression. I specifically include divers reporting mild problem with mood or orientation. It is precisely these types of soft, subjective complaints, unaccompanied by any objective neurologic abnormality, which may lead to the diver being labelled as a malingerer and make him reluctant to discuss his symptoms at all. While one cannot exclude a placebo effect, we should remember that the diver, while wanting to remain healthy, does have a reluctance to re-enter the chamber unnecessarily.

The test of pressure is direct, logical, and does no harm to the diver. I have found it extremely useful in my own practice. I can say that, in times past, I probably should have used it more often. I believe that diving physicians who use it aggressively will find, as I have, that mild decompression sickness is more common than most of us realise.

DIVING TREATMENT TABLES WHEN IS ENOUGH TOO MUCH?

Marcel Johnson

State of the arts diving techniques and equipment mean big returns in all facets of the diving community, sport, research, scientific, and commercial. These returns may vary according to the goal orientation of each segment; however, one particular factor never varies. Man under hyperbaric pressure is subject to decompression sickness.

The past decade has marked a radical development period for the diving community. Men have gone to deeper depths and remained there longer than ever before, probing further and further, seeking to define our limitations. And in the process, as in all other facets of diving, divers got bent from time to time. In dealing with these cases in the real world, treatment techniques for decompression sickness have also advanced. These advancements involve not only tables but also procedures for use of equipment and diagnostic techniques.

Let us look at a commercial dive job.

The first diver in the water is to clear loose debris from an area over a pipeline, use a water jet to remove the mud from the area and then cut a section of pipe out for repair using an oxygen arc cutting torch.

After working for forty minutes at 250 feet of sea water, breathing a mixture of 10 percent oxygen and 90 percent helium, being roughed up by crane slings, chains and jet hose, shocked and abused by the oxygen arc cutting lead and ground cable, in pitch black water, it is time to leave the wonderful world of zero visibility, jagged-sharp metal, and cold water. But this leaving takes a while because now the diver has to hang onto a down line at in-water stops while decompressing for the next 83 minutes while the cold gets colder, for now he is not working. The extra heat of work and the concentration used for the job are replaced by greater heat loss and boredom. By the time he climbs into the recompression chamber on the surface to recompress and breathe oxygen from a mask for 177 minutes at 40 fsw, even chamber time feels like a weekend vacation. It is hard to suck oxygen from the mask. Just the continuous working of a man's diaphragm and lungs against the mask diaphragm makes him sometimes want to give up breathing; however, he is now on a mattress with sheets, blankets, a pillow, magazines, coffee, juice, food, and maybe even heat. So just remember to breathe, breathe, that's it. Keep breathing ...

The washed out look and feeling of a man when he comes out of the chamber is something hard to describe. He looks as if he has been in the hospital under treatment for a long time, one step beyond tired and beat - more like used up.

Now the next man takes his turn and the first waits 18 hours. Then, get up and do it again. Amen.

How does a man feel before, during and after this sort of exposure? Depending on the individual and the job, that will vary somewhat, but following a dive like the one described here, he is going to have pain in his body, a headache sometimes, be tired, and his lungs will be tired and sore. He will have cuts, scratches, bruises, trains, and possibly even decompression sickness.

It would seem that symptoms of decompression sickness might be easy to miss or ignore when mixed in with all of the other sensations the body enjoys during hardworking dives and decompression. And it sometimes is, unless it is a very serious case with paralysis and unconsciousness. For this reason, it is a growing consensus among diving physicians that all bends cases, not matter what their degree, should be treated on a USN Table 6 or 6A, when the case is being handled in the field by diving personnel. If the dive team is inexperienced and ill-equipped, this consensus has its merits. However, this is, in most cases, not the situation.

If the diver being considered for treatment has just completed two or three hours of chamber time, breathing oxygen from a mask, essentially he has just undergone a routine treatment built into surface oxygen decompression tables. (Tables may vary according to the employer).

The diver may be physically unable to undergo further prolonged treatment on any oxygen table because of lung fatigue or oxygen toxicity problems. He may not be bent, or his problem may be resolved by use of USN Table 5. One thing is sure, he must go back under pressure to sixty feet on oxygen.

A complete and thorough neurological examination must be conducted as soon as possible, either on deck prior to recompression or at sixty fsw in the chamber. This examination will provide a road map of symptoms. The initial examination followed by frequent subsequent examinations throughout therapy, enables supervising personnel to pinpoint, trace, and alleviate suspected bends symptoms.

In cases where symptoms are subtle or doubtful, it is easy for the diver/patient to take this examination as a joke. When the Emergency Medical Technician (Diver)(EMT-D) has him wagging his tongue, rolling his eyes and fingering his nose, the diver may possibly feel he is auditioning for a slapstick comedy routine. Therefore, it is important that the examiner be well trained in what he is looking for and why. It is also helpful if the diver has had the basic concept of neurological examinations explained to him at some prior time. Check too for oxygen toxicity signs, and find out if the diver/patient can tolerate oxygen therapy.

Another tool used in diagnosis of suspected pain-only bends, and in conjunction with a neurological examination, is the test of pressure. Basically, this is when the diver enters the chamber and begins breathing oxygen at the surface, as he is recompressed at 60 fsw. He then continues to breathe O_2 for 20 minutes. If the symptoms are not relieved or improved by recompression and the neurological examination is negative, we can assume that the diver is not bent and he may be decompressed with no further treatment. This is not a treatment table. It is, however, a diagnostic tool.

During this evaluation period, critical decisions must be made regarding treatment table selection and logistics. If the diver is bent, most situations may be adequately resolved with supplies on hand using USN Treatment Tables. However, there are occasions when these are not enough and nitrox saturation techniques are necessary, as when a patient is at the end of his treatment table but needs more time at depth, or he needs to remain under pressure but can no longer tolerate breathing oxygen from his mask. The supervisor must recognise these facts and understand that he does not have to decompress. The patient may remain at his depth of relief indefinitely if emergency nitrox saturation techniques are instituted immediately, stabilizing chamber atmosphere within acceptable saturation limits.

Emergency nitrox saturation may be conducted in a standard double lock decompression chamber. The patient and tender are kept at treatment depth while all equipment, supplies and medical assistance are transferred, installed

and administered under pressure. Oxygen toxicity is a prime concern during this manoeuvre, therefore, if the situation indicates saturation, a positive decision must be made and carried out with all dispatch.

Lowering the recompression chamber partial pressure of oxygen to 0.5 atmospheres absolute by dilution with 100% nitrogen and installing a portable carbon dioxide scrubber will provide the necessary additional requirements. Because gas and equipment are not always readily available on site, it is a good idea to have planned for this eventuality ahead of time, so that one radio or phone call can mobilize all of the necessary people and supplies.

There are many factors to be weighed when dealing with a possible or known bend, no matter what the degree. It is important to have well-trained people on each dive site who are trained in diving medicine, such as an EMT-D and an experienced diving supervisor. These professionals should be able to recognise when a neurological examination and test of pressure are negative and the patient may be decompressed without treatment. They must determine how much treatment a diver/patient may require and how much he can physically tolerate.

There is no one magic answer to diving medical problems, and any oversimplified rule of thumb can lead to more problems than it resolves. Training, experience, and planning are our greatest allies during normal operations and emergency situations. By using a logical, well-planned response scenario, we can respond, without over-reacting and manage to a successful conclusion any diving medical problem that might arise on site.

THE DECISION TO "EMERGENCY SATURATE"

Marcel Johnson

Occasions sometimes arise in the treatment of decompression sickness when the normally used USN Treatment Tables are not adequate for a particular situation. A patient may require more time at his depth of relief than allowed by these tables, or he may need time at depth on O_2 and be physically unable to tolerate O_2 therapy. For whatever reason, if a patient needs to remain at his depth of relief for a prolonged period or needs deep recompression therapy, two items must be addressed immediately: Oxygen toxicity and decompression commitment.

The possible need for emergency saturation should be discussed and planned for at an earlier date by persons connected with diving operations, because when emergency nitrox saturation therapy must be conducted, time is very important. Because of the high level of oxygen the chamber occupants may have already been exposed to, the chamber environment must be brought within acceptable saturation limits and maintained there until such time as saturation decompression is indicated and completed.

The following is a step-by-step example of such a procedure, as used in actual field emergencies. It is offered only as a reference to those interested. The author assumes no responsibility for its use by others.

On the job site a double lock DDC is used. Should a decompression problem arise, it is treated in this chamber. The patient and inside tender go into the chamber and are pressurised to treatment depth and then undergo therapy as indicated. If during the course of events saturation therapy is indicated, saturation procedures are initiated while the patient and tender are at the patient's depth of relief. They will remain under pressure until proper decompression is completed.

1. Load into outer lock the following items as soon as possible and pressurise outer lock to inner lock depth.

   a. Emergency Medical Technician (Diver) (EMT-D).
   b. Medical kit
   c. CO_2 scrubber
   d. Portable heater/chiller unit
   e. Chamber lights
   f. Two crescent wrenches
   g. One pipe wrench
   h. Teflon tape
   i. Wiring harness for scrubber, heater and lights
   j. Wiring harness installation-under-pressure tool
   k. Thermometer, hygrometer

2. After blowing the outer lock down to inner lock depth with air.

   a. Inside the chamber, the EMT-D conducts a thorough neurological examination and administers medical assistance as necessary.

      Outside the chamber, the topside crew installs a 100% nitrogen supply whip to the outer lock blow down valve. Air or treatment mix is plumbed to the chamber built-in breathing system supply (BIBS), and a chamber atmosphere analysis tube and flow cap are installed.

   b. The EMT-D transfers from the chamber inner lock to the outer lock. He then passes all of the previously loaded items from outer lock to inner lock.

3. Close the inner lock hatch and commence decompression of EMT-D on the appropriate table.

   a. The inside chamber tender installs the wiring harness installation-under-pressure tool in a through hull penetrator.

   b. The outside tender installs the through hull penetrator with its pig tail resting in the installation-under-pressure tool.

c. The inside tender may now remove the installation-under-pressure tool revealing the through hull electrical penetrator's pig tail, which is now accessible inside the chamber.

d. The wiring harness for the scrubber, heater, and lights, which was passed inside during Step 2 may now be installed to the through hull electrical penetrator pig tail.

e. Activate the heater, scrubber, and lights. Check each component for proper operation.

4. The outer lock reaches the surface and its hatch is opened.

a. EMT-D exits chamber.
b. Load into outer lock the following items:
   1. Plastic bags
   2. Toilet paper
   3. Bucket
   4. Towels
   5. Blankets
   6. Drinking fluids
   7. Food
   8. Any miscellaneous items deemed necessary at this time
   9. BIBS overboard dump panel if available

5. Chamber occupants go on BIBS breathing air (or treatment mix).

6. Pressurise the outer lock to the same depth as the inner lock with 100% nitrogen and open the inner lock hatch.

a. Allow the chamber atmosphere to settle and adjust same as necessary until a PPO_2 of 0.5 ATA is reached.
b. Chamber occupants may go off BIBS at this point as desired and/or directed.
c. If a BIBS dump panel is to be used, this is the time to install it.

The chamber occupants may remain at this depth indefinitely without ill effects from O_2 or other contaminants as long as proper saturation procedures are adhered to.

The chamber temperature should be maintained at a level comfortable to the occupants. The patient should remain supine as much as possible and force himself to consume fluids beyond the level called for by the normal thirst reflex. It is important that the whole body circulation and hydration be optimal in order to facilitate inert gas wash out from the patient's body.

A competent doctor knowledgeable in diving medicine should be called to the site if possible and/or the chamber with its support equipment should be loaded onto appropriate transportation and transported to a proper hyperbaric treatment facility where therapy is completed and the patient is thoroughly examined by the attending hyperbaric physician. Follow up examinations are conducted as deemed appropriate by the physician.

PERSONAL PROFILE

Michael D (Marcel) Johnson has trained in diving and diving medicine at the US Naval School of Diving and Salvage, the US Navy Experimental Diving Unit, the US Naval School of Deep Diving Systems and Saturation Diving, the Professional Association of Diving Instructors and the Daniel Freeman Hospital's Emergency Medical Technician-Diver Program, under the auspices of the Commercial Diving Center in Los Angeles, California. Mr Johnson has been active on the Association of Diving Contractors (ADC) Medical, Safety, and Technical Committees. He has served as Director of Diving Safety, Western Hemisphere, to Oceaneering International Inc., in Morgan City, Louisiana, and is at present an active commercial diver.

UNDERSEA MEDICAL SOCIETY INC. ANNUAL SCIENTIFIC MEETING JUNE 1-5, 1982, NORFOLK, VIRGINIA

Noel Roydhouse

The UMS started off in 1967 when a group of about 200 underwater medical research scientists wanted to create a forum where they could exchange views, discuss problems and obtain an overview of the research going on in the various research centres. UMS has expanded considerably and now has a membership of 1928 members in 47 different countries. Affiliated to UMS, and each having a member on UMS National Executive, are the South Pacific Underwater Medical Society (SPUMS) and the European Underwater Biomedical Society (EUBS).

When it comes to talking, those research workers who apparently lock themselves up or are locked up in their laboratories, really let themselves go. The locking up is in reference to the various naval laboratories where secret research is carried out, little of which is permitted to reach the public eye. In fact, to get into these laboratories one has to have a naval connection and even then certain areas are restricted.

Hence the programme was heavily oriented towards pure research with such papers as "Free Radical-induced lipid peroxidation and ion flux as a mechanism in oxygen convulsions" and "Muscle-twitch contractions appear to be slower and stronger in man during deep saturation diving". For the physician interested in the Bends and Decompression there was one whole day and another day of poster presentations containing much valuable information.

From the exercise physiology point of view the "Underwater Ergonometry with a US Navy Team" was interesting. They used the principle of a cycle ergometer moved by the hands which was mounted on a frame so that a person could be exercised lying in any plane. The brief synopsis was that the maximum oxygen uptake did not vary with the position of the diver. In the clinical session on "Exercise Limitations in Diving", the belief was that, contrary to

surface exercise where circulatory system and oxygen diffusion is the limiting factor, in hyperbaric circumstances it is the respiratory system that limits exercise. At 1 ATA (atmosphere absolute) there was an increase in Max VO_2 by about 10% due to the increase in PO_2. However the endurance time increased by about 50%. However with the denser gas the hypothesis was that there is a high gaseous inertia. In addition with rapid expiration a "choke" point develops which may be physical or functional. The hypothesis is that when exercising the expiratory flow is limited and hypoventilation occurs which stimulates the respiratory centre leading to over-ventilation with a burden on the inspiratory muscles leading to their fatigue. Carbon Dioxide (CO_2) builds up, causing further respiratory stimulation. At this stage, before CO_2 narcosis comes on, dyspnoea occurs related to the muscle fatigue and the tension in the lung tissue. Dyspnoea is a known condition in divers exercising hard. The author now knows to assume an upright position and to slow down and not to blame his old regulator.

The meeting went on in a serious manner until Friday afternoon when J Dwyer, Professor of Paediatrics and an expatriate Australian, delivered a dynamic paper, UNDERSTANDING MODERN IMMUNOLOGY, THE SECOND MOST FASCINATING SYSTEM IN BIOLOGY. He introduced much humour to the proceedings and even the title was a take-off. If he had called it as he believes THE MOST FASCINATING SYSTEM he could become involved in argument with other specialists. Like his mother, he takes the soft-line and when she is asked what her son specializes in, she says he is a cardiologist, thus letting herself out of explaining what a paediatric immunologist does. One wonders what an immunologist has to offer Hyperbaric Medicine. He is a consultant on immunology to NASA because they found out that on return to earth, the astronauts appeared to have lost their T-lymphocytes. Now you only have one lot of T-lymphocytes and their life-span is about 60 years and you do not get any more. However, they found the T-lymphocytes back in the bone marrow to whence they had migrated. As he said, "heaven knows what happens to lymphocytes in divers".

And so the meeting came to an end on Saturday afternoon with a case-history on an unusual but not unknown cause of air embolism. The paper was entitled "Orogenital sex as a cause of non-fatal air embolism in pregnancy". There are ten recorded cases in the literature and they all died but this one survived. The modus operandi is the husband blowing into the vagina which leads to almost immediate collapse and loss of consciousness due to massive air embolism. Eventual treatment in a recompression chamber led to some recovery but rehabilitation was necessary at the time of the reporting.

There were 247 registered at the meeting with people from many different countries. The programme covered 3 and a half intensive days when 54 papers were delivered, 18 tutorials given, 35 poster displays shown and 2 demonstrations given. The only criticism, and this was well known to the organisers, was that there were not enough clinical presentations for the clinicians present who were about 75-80% of those attending. All in all another fine meeting of which UMS can be justly proud.

LETTERS TO THE EDITOR

SHELL UK LIMITED
Shell-Mex House
Strand
LONDON WC2R ODX

The Editor,
SPUMS Journal

I accept the apologies that were offered in your December editorial for any transcription errors in the verbatim reporting of one of my talks at last year's annual scientific meeting. On the contrary, I would like to congratulate your editorial team on catching the theme so well.

However, let me please cerrect one of my own statements (Oct-Dec 1981; 11(4): 27) following a case reported to me within the last few weeks. Unilateral pallor of the tongue (in fact LIEBERMEISTER'S SIGN) was demonstrated at the post-mortem of an undoubted case of air embolism (RR Pearson, personal communication). I hope none of you has cause to report another.

David Elliot

NOTES TO CORRESPONDENTS AND AUTHORS

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

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

REPRINTING OF ARTICLES

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

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

26th April 1982

DIVING SAFETY MEMORANDUM NO 11/1982
DIVER/BELL UMBILICALS

Underwater inspections and maintenance tasks often require divers to operate on long umbilicals.

The choice of lengths of the diver/bell umbilical and the distance which the diver is permitted to operate from the diving bell must depend on a variety of factors, not the least of which is the size of the diving bell and stowage space within that bell, taking into consideration the problems of the bellman having to go the full extent of both umbilicals to assist a diver in distress and the problems of return, recovery and stowage. Care has also been taken in the upward and downward excursion which the diver is permitted to make. The size and content of the bail-out cylinder must be considered.

Diving Memorandum No 5/1980 dated 8 April and amended in June of 1981 attempted to advise on a maximum diver umbilical length. Due to the variety in size of equipment and techniques employed, this advice has not necessarily been of value. The circumstances prevailing at the time plus the equipment in use establishes the maximum safe distance that a diver can horizontally excursion from the bell. The establishment of this distance is the responsibility of the diving contractor and the on site supervisor remembering that the diver should always be placed as close to his task as is reasonably practical.

Diving Safety Memorandum No 5/1980 is cancelled.

17th May 1982

DIVING SAFETY MEMORANDUM NO 12/1982
KIRBY MORGAN BAND MASK/HOOD
RETAINING BAND SCREWS

Recent accident investigations have identified a post dive procedure employed by some divers which is potentially dangerous.

It would appear that some divers employ the practice of slackening off the hood retaining band screws on the Kirby Morgan band mask during post dive servicing and leave them slack until pre-dive checking on resumption of diving when the tender or diver tightens them prior to the dive.

One near fatality can definitely be attributed to the failure to tighten the screws before diving and may have been responsible for several other incidents.

Should it be considered desirable to slacken off the hood retaining band screws during post dive procedures the band and hood should be completely removed.

28th May 1982

DIVING SAFETY MEMORANDUM NO 13/1982
MEDICAL CASUALTIES TRANSFER UNDER
PRESSURE

The Diving Operations at Work Regulations 1981 require diving contractors to provide facilities for transferring diving casualties safely under a suitable pressure to a place where treatment can be given safely under pressure.

As most diving contractors are aware, a hyperbaric evacuation facility, intended primarily for medical evacuation of divers under pressure from an offshore facility to a so-called “hospital chamber” has been established for some time. This facility consists of the permanent shore chamber owned by the Grampian Health Board and the portable transfer chambers owned by a consortium of operators.

Diving contractors should ensure that if the operator for whom they are working is not a member of the “consortium of operators” that they have taken the necessary action to conform with the requirements of legislation. The use of the transfer facility should not be relied on in the event of a requirement to evacuate divers under pressure if their vessel or support platform is at risk. Under some circumstances the system could be used but diving contractors should have an adequate primary method of coping with this contingency.

Request for use of the TUP facility should come from the “on-site” qualified medical practitioner.

The initial point for communication in the event of an emergency is the Aberdeen Royal Infirmary, Foresterhill, Aberdeen, in which is situated the central telephone exchange servicing the Aberdeen Hospital and which is manned on a 24 hour basis. The number is Aberdeen (0224) 681 818. The telephone operators, having been given the essential details of the incident, will contact the Duty Community Medicine Specialist who in turn will then contact the hyperbaric consultant.

The Scottish Home and Health Department accepts responsibility for the treatment of casualties transferred under pressure when the casualty is locked into the main on-shore chamber.

PROJECT STICKYBEAK

Send incident reports to

DR DG WALKER,
PO Box 120
NARRABEEN NSW 2101

INTERNATIONAL UNION OF PHYSIOLOGICAL SCIENCES
XXIXth INTERNATIONAL CONGRESS

Sydney 28th August - 3rd September 1983

Although the first international physiological congress was held in the last decade of the nineteenth century, this meeting in Sydney, is the first such International Union of Physiological Sciences Congress to be held in Australia. The members of the Australian Organising Committee are anxious that every Australian clinician and biological scientist in whatever field or speciality is aware of the Congress and of the fact that all such workers and practitioners are welcome to attend and participate.

The Congress is essentially in two streams:

1. Free Communications. Everyone is invited to submit one of these.

2. Invited Lectures and Symposia which will be grouped around a variety of themes.

THEMES AND THEME CO-ORDINATORS

A. Perinatal Events (2 Symposia): GD Thorburn
B. Reproduction (4 Symposia): DM de Kretzer
C. Execution of Voluntary Movement (5 Symposia): R Porter
D. Adaptation to the Environment (5 Symposia): GB Sharman
E. Blood Flow and Function (2 Symposia): E Lumbers
F. Peripheral Autonomic Nerves (3 Symposia): ME Holman
G. Excitation and Contraction (5 Symposia): A Dulhunty
H. Membrane and Epithelial Transport (6 Symposia): JA Young
I. Respiratory Gas Transport/Respiration (5 Symposia): A Woolcock
J. Metabolism and Nutrition (4 Symposia): FJBallard
K. Regulation of Body Fluids (6 Symposia): AZ Gyory
L. Channels in Excitable Membranes (4 Symposia): P Gage
M. Physiological Basis of Perception (5 Symposia): I Darian-Smith
N. Neural Communication (3 Symposia): S Redman
O. Structure and Function of Circadian Pacemakers (3 Symposia): M.C Moore-Ede
P. Endocrinology (5 Symposia): H Niall
Q. Developmental Neurobiology (5 Symposia): MR Bennett
R. Cardiorespiratory Control (5 Symposia): DI McCloskey

SINGLE SYMPOSIA AND CHAIRMEN

1. Mechanisms of Cardiac Arrhythmia: E Coraboeuf, France
2. Pain: WE Willis, USA
3. Cardia Mechanics: G Elzinga, Netherlands
4. Pathophysiology of Large Arteries: R Ross, USA
5. Comparative Physiology of Hearing: R Klinke, GFR
6. Platelet Functions: B Firkin, Australia
7. Comparative Physiology of Vertebrate and Invertebrate Vision: RW Guillery, USA
8. Autonomic Nervous System in Man: J Ludbrook, Australia
9. Motivational Behaviour State Control: P Karli, France
10. Learning and Memory: ER Kandel, USA
11. Construction and Testing of Models of CNS Action: DM MacKay, UK
12. Metabolic Aspects of Endothelial Function: D Sherpo, USA
13. Capillary Transport: C Crone, Denmark

Every registrant will receive a comprehensive book of the abstracts as well as tickets for two special Congress Receptions. A special Congress Concert, with the Sydney Symphony Orchestra conducted by Sir Charles Mackerras, has been arranged and a notable programme has been devised for accompanying members. There is a concessional rate of registration for students.

The Organising Committee hope that large numbers of students - at all levels will take advantage of the great opportunity that such a congress offers of hearing and meeting so many famous physiologists. So often the high cost and inconvenience of international travel virtually eliminate such opportunities for students (and many graduates).

The second circular, with complete organisational details for the Congress, as well as the instructions for the submission of abstracts is available now. The abstract deadline will be at the end of December. To go onto the list for distribution of this circular you are asked to write to:

The Secretariat,
29th Congress of the International Union of Physiological Sciences,
Post Office Box 783,
CANBERRA CITY, ACT, 2601
AUSTRALIA

The registration fee is expected to be in the region of $200.00.

MEMBERSHIP OF SPUMS

Members pay $20.00 yearly and Associate Members $15.00. Associated Membership is available for those neither medically qualified nor engaged in hyperbaric or underwater related research. Membership entitles attendance at meetings and the Annual Scientific Conference and receipt of the Journal/Newsletter.

Anyone interested in joining SPUMS should write to:

Dr Janene Mannerheim
Secretary of SPUMS
22 Frank Street,
Box Hill South VIC 3128