Townsville, taking approximately 3 hours, is $3,000 plus staff salaries.

In overseas retrievals the cost is always borne by the diver or by his or her insurance company.

Communications

For all accidents occurring in Queensland, we are usually first contacted by the DES network, and as most of these divers are Australian, the Health Authority meets the costs. For divers who are not eligible for Medicare, and for those retrieved from overseas, the financial aspects are dealt with by one of the medical retrieval agencies, and they sub-contract the work to our unit in Townsville.

We have collected divers from Cape York to as far south as the Gold Coast, and offshore from PNG, Fiji, Nauru, Port Vila and the Solomons. With increasing diver awareness we expect this work to increase.

Treatment

In all patients our initial first aid is

1 85% or greater oxygen via appropriate mask and circuit;
2 the patient is positioned horizontally if air embolism is a possibility;
3 aggressive rehydration intravenously.

If we are using the Duocom, we initially compress to 18 msw with the intention of treating using a RN Table 62 profile of pressure and time. We have the option of going to 50 msw in deteriorating cases.

On arrival in Townsville, the diver is transferred, under pressure, to the main chamber in the Hyperbaric Medicine Unit of the Townsville General Hospital (Figure 3), where the treatment profile is completed. Follow-up recompressions are conducted as is required following repeated patient assessment.

Conclusions

Any retrieval service must satisfy the objective that it is “to improve patient care”. Stated more simply, the care given during transport must equal or better the management at the point of referral.

There can be no doubt that our service fulfils these objectives, and as it is yet to be determined whether early retrieval and treatment ultimately reduces morbidity, any caring society is obliged to support activities its community indulges in. This earlier treatment may reduce the incidence of some of the irreversible conditions related to scuba diving. As inexpensive insurance becomes readily available to all divers, the onus of cost will fall on the consumer, and clearly, the divers will want to be recompressed as early as possible, putting additional pressure on hyperbaric units to run efficient retrieval services.

Dr G.S. Gordon, BHB, MBChB, FFARACS, FANZCA, DipDHM, is a Staff Anaesthetist at the Townsville General Hospital. His address is P.O. Box 670, Townsville, Queensland 4810, Australia.

The photographs were taken in October 1991 by Dr John Knight during a visit to Townsville. Then retrieval organisation was then the North Queensland Emergency Response Group.

REPORTS OF OTHER DIVING AND HYPERBARIC MEETINGS

HYPERBARIC TECHNICIANS AND NURSES ASSOCIATION (HTNA) FIRST ASM

Hyperbaric nurses and technicians in Australia founded the Hyperbaric Technicians and Nurses Association at their first Annual Scientific Meeting in Adelaide on August 28th and 29th 1992.

The Association aims to promote and encourage the exchange of information between members; to standardise protocols and practices within the technical and nursing communities affiliated with the HTNA; to educate and inform the recreational diving community of developments that affect safe diving practice and standards; and to establish training requirements for hospital based chamber technicians.

The HTNA encourages members to join SPUMS and is closely affiliated with the Australian and New Zealand Hyperbaric Medicine Group, which is a Standing Committee of SPUMS.
Full membership is open to nurses and technicians currently working in the field of Hyperbaric Medicine.

Associate membership is for individuals or groups who have an interest in the aims of the Association.

Corporate membership is available for those companies or organizations that wish to support the aims of the HTNA.

Officers for the 1992-1993 year are:
President: Christy Pirone
Secretary: Steve Goble
Treasurer: Andrea Jones

State representatives:
New South Wales Barry Spiers
Victoria John Houston
South Australia Bob Ramsay
Western Australia Sharon Keetley
Northern Territory Dave King
Queensland Allison Mann
Tasmania Sean Rubidge

The Annual Scientific Meeting was attended by 40 hyperbaric nurses and technicians from nine different hyperbaric units around Australia.

The first day opened with a keynote address by Professor Bill Runciman, Head of the Department of Anaesthesia and Intensive Care at the Royal Adelaide Hospital and Chairman of the Quality of Practice Committee of the World Federation of Societies of Anaesthesia. He gave a superb account of the role of oxygen in evolution, entitled, “Oxygen - The Fire of Life”.

Dr “Fred” Gilligan, Director of Retrieval Services and “Forefather” of Hyperbaric Medicine at the Royal Adelaide Hospital, gave a particularly interesting history of Hyperbaric Medicine in Australia. Fred is an excellent source of not only hyperbaric and retrieval knowledge, but also hyperbaric historical facts and trivia.

Scientific papers were presented by Mandy Wilson (Hypobaric decompression illness - a case history), Jodie Perris (Differential diagnosis: DCI vs Irukandji stings), Henri Bource (The role for a private hyperbaric unit), Bob Ramsay (An alternative to steel for hyperbaric chamber construction), Sue Sheeran (Monitoring in a hyperbaric environment, Sydney style), and Dr Frank Quigley (Diagnostic and prognostic tests for ulcers; prediction and outcome).

A Safety Forum included an introduction to the Hyperbaric Incident Monitoring Study by Christy Pirone and a most “enlightening” presentation by David McGowen on “Fire safety and the hyperbaric environment”.

An Open Forum was held after the Annual General HTNA Business Meeting which produced much constructive discussion about dive tables, diving community education, hyperbaric research around Australia, and hyperbaric unit databases.

The quality of presentations and the discussion generated at the meeting was of a very high standard. Most attendees felt the meeting was very useful and long overdue.

The next meeting is to be held in tropical, warm sunny Darwin on August 6th, 7th and 8th 1993. Plan your next holiday for Darwin with the HTNA!

Membership application forms are available from
Steve Goble, HTNA Secretary,
Hyperbaric Medicine Unit, Royal Adelaide Hospital,
North Terrace, Adelaide,
South Australia 5000.

GLEANINGS FROM MEDICAL JOURNALS

SELECTED ABSTRACTS

Reprinted from the Program and Abstracts of the UNDERSEA AND HYPERBARIC MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING 23rd-27th June 1992

The address of the Undersea and Hyperbaric Medical Society, Inc. is 9650 Rockville Pike, Bethesda, Maryland 20814, U.S.A.

DIVERS AND ASTHMA

Observation on asthma in the recreational diving population.

Asthma is considered a contraindication to diving because of concern for pulmonary barotrauma due to lung over inflation during ascent from diving. However, asthma is extremely prevalent in the US (estimated to be 5-8% of the population). It seems likely therefore that a sub-population of sport divers may have asthma, and clinical experience
indicates that some asthmatics are participating in sport diving. Evaluation of the rationale for considering asthma as a contraindication to diving requires data on the role of asthma in diving-related illness to determine if asthmatic divers are at increased risk for diving accidents. Through a screening study in the consumer publication Skin Diver Magazine, (Petersen Publishing Co, Los Angeles, paid circulation 211,041), 10,422 responses to the question “have you ever had asthma” were returned from readers of the magazine. 870 (8.3%) answered yes, 343 (3.3%) indicated that they currently have asthma, and 276 (2.6%) stated that they dive with asthma. This screening study confirms field observations of asthmatics participating in diving. While this initial study does not support an exact projection of the number of asthmatic divers, it has isolated a group of asthmatic divers for further study.

From Temple University, Philadelphia, Pennsylvania and the University of California, San Diego, California, U.S.A.

A survey of diving asthmatics.  

Asthma has traditionally been considered a risk factor for decompression illness in scuba divers. In order to characterize scuba diving asthmatics, in August 1991, a questionnaire was placed in Alert Diver, of which approximately 75,000 were mailed. By January 1992, 279 questionnaires were returned. Sixty-three percent were male. Mean age was 37.4 years (range 15-75). Two-hundred forty-three (88.7%) responders took medication for asthma, and 55.8% took medication pre-dive. Seventy-three (26.4%) individuals had been hospitalized for asthma, of which 20.7% had been hospitalized 1-5 times, and 5.8% had been hospitalized 6 or more times. Onset of asthma began under age 14 in 57.4%. Of those who wheezed, 13.3% had symptoms daily, 17.3% weekly, 26.2% monthly, and 43.1% annually. There were a total of 56,334 man-dives reported by 269 individuals. There were 11 cases of decompression illness reported in 8 individuals. The calculated risk of decompression illness in questionnaire responders (1 in 5,100 dives) significantly exceeds the estimated risk for unselected recreational divers¹, with an odds ratio of 4.16 (χ²=119.4, p=0.00001). We conclude that the risk of decompression illness is higher in the surveyed asthmatics than in an unselected recreational diving population. Further investigation will be needed to quantify this risk according to the degree of severity of asthma.

Reference 
1 Wilmhurst P. Prog Underwater Sci 1990; 15: 31

From the Divers Alert Network and F.G. Hall Hypo/ Hyperbaric Center, Duke University Medical Center, Durham, North Carolina, U.S.A.

DECOMPRESSION ILLNESSES

Concordance: a problem with the current classification of diving disorders.  

Using the current classification of the decompression disorders¹, a diagnostician is frequently required to make a diagnosis which assumes a disease mechanism or site of injury. To examine how consistently this can be done, 47 diving physicians agreed to “diagnose” 50 case histories selected from INM case records covering a two year period. The participants were asked to make a differential diagnosis, allocating an index of confidence (0-100%) to each diagnosis, or to state that there was insufficient information on which to base a diagnosis. Five cases were excluded from the analysis because “inadequate information” represented more than 10% of responses. Five cases had predominantly non-DCS diagnoses. In 3 cases the predominant diagnosis was from the “Type I” DCS category, but in 11 cases, there was inconsistent discrimination between “Type I” and “Type II” DCS. Some observers chose as alternative diagnoses conditions from both the “Type I” and “Type II” categories. In 7 of the 22 “neurological” cases, there was poor discrimination between AGE and “Type II” DCS (i.e. >20% mean confidence ascribed to each diagnosis). This inconsistency was found both within and between individuals. Of the four cases diagnosed as “audiovestibular”, inner ear barotrauma was not clearly differentiated from labyrinthine of cerebral DCS. Concordance was calculated as the mean of the confidence ascribed to the “most likely” diagnosis for each case averaged over the 45 cases. It was calculated to be 52%. While it is recognised that it can be difficult to make diagnoses based upon case histories alone, each diagnostician was given identical information. These initial results indicate that, using the “current” classification there is poor agreement between physicians diagnosing the decompression disorders.

Reference  

From the Institute of Naval Medicine, Alverstoke, Hants PO12 2DL, U.K.

Climatic and environmental factors in the aetiology of DCI in divers  

Decompression illness (DCI) may occur unexpectedly after “safe” dives and in the UK unexplained clustering of cases has been observed. It was hypothesised that the
weather and environmental factors may influence the occurrence of DCI. INM diving accident records were searched and all cases of DCI for the 6 years 1984-1989 extracted. Cases with historical features traditionally associated with arterial gas embolus were excluded. Records were required to contain clinical details, date and time, location, depth, and duration of the dive(s) preceding DCI. 177 records fulfilled these criteria. The climatic and tidal conditions prevailing at the time and place of each dive were ascertained from Meterological Office archives and Admiralty Tide Tables. Dive profiles were compared with an arbitrary standard, RN Air Decompression Table 11, and depending on whether dives conformed with or exceeded the time/depth profiles recommended, they were allocated to an index “safe” group or control “risky” group.

These groups were further subdivided depending on whether single or multiple dives had preceded the onset of DCI. Group numbers were: 42 “safe” dives of which 36 were single and 6 multiple, and 135 “risky” dives of which 80 were single dives and 55 multiple. Climatic and environmental data for each group was compared by χ² analysis of contingency tables for each environmental variate. It was found that “safe” dives were more likely to result in DCI if the surface environment was cold. Significant differences were found in air temperature and wind chill (p<0.01 for all dives, <0.01 for single dives) and for air minus water temperature (p<0.01 for all dives, <0.05 for single dives) when prevailing conditions at the time of the “safe” dives resulting DCI were compared to the “risky” dives. For surface water temperature the difference between the two groups failed to reach significance when single dives alone were examined but p<0.02 for all dives. No statistically significant difference was found in barometric pressure, change in barometric pressure between the time of the dive and symptom onset, wind speed alone, or tidal factors between “safe” and “risky” dives. The results imply that exposure to a cold thermal environment following diving, particularly when the air temperature is cooler than the water temperature, may be a more important risk factor for the development of DCI than has hitherto been assumed.

From the Institute of Naval Medicine, Alverstoke, Hants PO12 2DL, U.K.

A retrospective review of the epidemiology of diving accidents treated at Naval Station Roosevelt Roads from 1986 to 1991 and implications for improving diving safety in Puerto Rico.

One hundred and eight diving accidents treated with emergent recompression therapy at Naval Station Roosevelt Roads between 1986 and 1991 were reviewed. Eighteen per cent of these accidents involved military members, 13% involved recreational divers, and 69% involved commercial spearfishermen. The diving accident diagnosis was DCS II in 61% of cases, DCS I in 18% of cases, and AGE in 21% of cases, while the initial treatment rendered was 38% USN TT6, 43% USN TT6 with extensions, 15% USN TT6A, and 4% USN TT5.

Treatment was delayed over 6 hours in 71% of cases and was delayed over 12 hours in 29% of cases. Ninety two per cent of the injured Puerto Rico civilian divers were outside the tables. Furthermore, only 17% of the civilian divers were certified although 86% had over two years diving experience. Over 85% of civilian divers were diving repetitive dives deeper than 80 fsw for multiple days. Thirty per cent of the civilian divers treated had residual symptoms requiring repetitive hyperbaric oxygen treatments, and there was only one fatality. Of the 15 patients requiring repetitive treatments in 1990 and 1991, 9 recovered, 3 achieved independent ambulation with an abnormal gait, and 4 remained wheelchair bound. These figures indicate that the large majority of diving accidents treated in Puerto Rico are associated with inadequate training and improper diving practices which may lead to significant permanent disability. It is postulated that increased educational efforts and stricter regulation of access to compressed air would improve diving safety in Puerto Rico.

From the U.S. Naval Hospital, PSC 1008, Box 3007, FPO AA 34051-8150.

Gender-related risk of decompression sickness in hyperbaric chamber inside attendants: a case control study.

From 1976-1990, Virginia Mason Medical Center carried out 7,910 hyperbaric oxygen therapy treatments exposing approximately 8,424 inside attendants (IAs). In that time, 26 IAs have been treated for decompression sickness (DCS) symptoms (0.31%). A case control analysis showed that the rate of DCS was dependent upon the maximum depth of the exposure (p<0.0001) and that exposure ratio for males and females (0.38 to 0.62) was similar to their ratio of DCS (0.31 to 0.69). Of the 9 female IAs whose menstrual history is known, 5 were menstruating when they developed DCS. Assuming 4 days of menses in a 28 day cycle, the risk for DCS in IAs with menses was 7.6 fold (p<0.01). Two of 24 female recreational scuba divers treated in the same period were menstruating during DCS. Assuming 4 days of menses in a 28 day cycle, the risk for DCS in IAs with menses was 7.6 fold (p<0.01). Two of 24 female recreational scuba divers treated in the same period were menstruating during DCS (p<0.013). Comments on the type of IAs DCS symptoms are included. We conclude that for dry hyperbaric chamber exposures there is no gender-related risk for DCS, but significant risk related to the
maximum depth of exposure. Menses is a significant risk factor for female chamber IAs but not for female recreational divers in open water.

From the Virginia Mason Medical Center, Seattle, Washington 98111, U.S.A.

Cerebral involvement in decompression sickness.

There is increasing clinical evidence to suggest that cerebral involvement in decompression sickness (DCS) involving the central nervous system is more common than is evident from all previous studies which rely on diagnostic criteria derived from the presentation, clinical examination and response to therapy.

The use of new functional imaging techniques has added a new awareness to this possibility and its importance. Using a cranial window in a canine animal model we have been able to visualize and record events in the pial circulation during the onset and development of DCS following a no-stop dive to 300 fsw for 15 min.

These events were related to changes in the cortical and spinal somato-sensory evoked potentials (CSEP and SSEP) generated by stimulation of the median and peroneal nerves. The response to recompression was also studied as well as the consequent histopathology. The results of these experiments indicate that, inter alia:

1. The initial event in altered cerebral function, as measured by CSEP’s, is the appearance of arterial gas emboli.

2. In this model, at least, the progression to complete occlusion by gas of the pial arterial and venous circulation is dramatic, often occurring in less than one minute. This process appears to be “fueled” by off-gassing of cerebral tissues into arterial gas emboli that have arrested.

3. Recompression rapidly restores perfusion but, in some cases, clearing of gas had not occurred at 60 fsw.

4. The CSEP’s were always affected before the SSEP’s suggesting that the brain is significantly more vulnerable to decompression stress than the spinal cord.

5. The resulting histopathology in cerebral tissues was entirely compatible with an ischemic process and correlates well descriptions of acute and chronic pathology in divers.

From the Naval Medical Research Institute, Bethesda, Maryland 20889-5055, U.S.A.

Femoral head decompression sickness as a concomitant of central nervous system decompression sickness.

Decompression sickness is typically divided into two categories, Type I, pain only disease, and Type II, that disease associated with the central nervous system (CNS). Deficits in sensory and motor function are the predominant signs of DCS of the CNS. The decreased pain sensation can be of such severity as to mask the presence of acute bone DCS. The presence of CNS DCS does not eliminate the possibility of the simultaneous occurrence of bone DCS. It is important to recognize the presence of Type I DCS because of the ramifications of chronic bone DCS, dysbaric osteonecrosis.

To look at the frequency of concomitant Type I and Type II disease, we compressed young pigs to 150-200 feet of salt water and then decompressed them in such a manner as to produce clinical DCS.

Pathological examination of the brain, spinal cord, and both femoral heads of the animals was undertaken. A significant number of the animals with gas bubbles in their CNS were found to have gas bubbles in the epiphyseal areas of the femoral head, these epiphyseal lesions being indicative of the presence of acute bone DCS. We conclude that acute bone DCS may occur simultaneously with CNS DCS in a significant number of patients and may go unnoticed. Given the possibility of concomitant Type I and Type II disease it would appear prudent that the long term management of patients with central nervous system DCS should include monitoring for dysbaric osteonecrosis the long term sequelae of acute bone DCS.

From the Hyperbaric Treatment Center, 42 Ahui St., the University of Hawaii School of Medicine, and Tripler Army Medical Center, Honolulu, Hawaii.

Presentation of concurrent decompression sickness and carbon monoxide poisoning - treatment with HBO.

This case presentation is a 32 year old experienced scuba diver successfully treated for combined decompression sickness and carbon monoxide poisoning four days post-dive exposure (day 0). He was a good health prior to the (only) dive, had moderate alcohol intake the night prior, but no other known risk factors for DCS. The patient, his friend and a Mexican guide all noticed an odor “like chemicals” or exhaust fumes in their air supply.

All dived to 100 fsw, but the patient recalls being caught in a current which forced him 20-30 feet deeper than
his two buddies. His ascent to join the other divers was difficult due to problems with his dry-suit, weakness and leg cramps, and necessitated a rest stop on a sand shelf at 60 fsw. By this time, low on air and with severe nausea since 15 min into his 35 min dive, he ascended to the surface without a required decompression stop (the other divers still had 1/2 tank of air left). His buddies complained of nausea during and after the dive. On surfacing, he vomited copiously and was so profoundly weak he had to literally be helped out of his gear and lifted onto the boat. He developed bilateral joint pains on day 1. He flew back to the U.S. on day 2 and developed a headache during the flight. All symptoms resolved by day 3 leaving him with residual patchy extremity paresthesias. He returned home on day 4, noted by family and friends to be mentally slow and unstable. He was treated with IV fluids and, on days 5-7, with two USN TT6 followed by three USN TT5. The paresthesias resolved, and he gradually improved. Psychometrics testing was done a total of four times showing gradual improvement, the last test was normal. This combined insult is a disordered decompression. The patient recovered fully.

From the Department of Hyperbaric Medicine, David Grant USAF Medical Center (MAC), Travis Air Force Base, CA 94535.

Radiographic imaging in neurological decompression illness.

All patients with neurological decompression illness (DI) evaluated at Duke University Medical Center who had CT or MRI evaluation of their brain or spinal cord were included in this study, a total of 66 patients. Brain MRI scans were performed in 46 patients, CT scans in 17 (15 with IV contrast) and both studies in 8. Twenty four patients underwent MRI scanning of the spine using surface coils. There were 52 males and 14 females (mean age 34.8 years, range 16-59). All patients were classified into B (brain involvement) or S (spinal cord only). CT scans were classified as normal (N), abnormal with lesion probably related DI (AR) and abnormality probably unrelated to DI (AU), e.g. cerebral atrophy. MRI scans were classified as normal (N), large cortical and subcortical abnormality (AL), small area of increased T2 signal in the centrum semiovale (UBO) or small area of increased T2 signal in a region atypical for UBO (AS). AR on CT or AL on MRI were only observed in the presence of an abnormal neurological exam. In the 8 patients who had both studies of the 6 with normal CT had had either AL or AS on MRI; both individuals with abnormal CT also had an abnormal MRI.

Only two individuals had abnormalities detectable within the cord (8.3%). We conclude that CT and MRI are frequently normal in neurological DI. Although statistical significance was not achieved abnormalities appear to be more frequent in the presence of B. These two imaging techniques are less sensitive than clinical evaluation in neurological DI.

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<td>Normal</td>
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<td>Brain involved</td>
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<td>Spinal cord only</td>
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From the Hyperbaric Center and Departments of Anesthesiology, Neurology, Pulmonary Medicine and Radiology, Duke University Medical Center, Durham, N.C. 27710 USA.

Demonstration and explanation of the breach of the pulmonary filter with transesophageal bubble contrast echocardiography.
Harch PG, Gottlieb SF, Van Meter K, Kerut EK and Swanson HT. Undersea Biomed Res 1992; 19(Supp); 41

In a companion abstract, a case was presented where Type II DCS was masked as mental illness. We were interested in understanding the mechanism of the CNS manifestations of the DCS resulting from a breath-hold ascent from 30 ft in cold, fresh water altitude diving. To simulate the cardiovascular and pulmonary mechanics of a breath-hold ascent, bubble contrast transesophageal echocardiography was performed. In the absence of a patent foramen ovale, significant numbers of bubbles were detected in the pulmonary vein and left atrium during phase IV of the Valsalva manoeuvre. Blood gas analyses failed to demonstrate significant a-v shunting. The unusual bubble patterns observed may be explained by the cardiovascular and pulmonary vascular dynamics associated with the Valsalva manoeuvre. It is thought that the overshoot of the blood pressure rebound resulting from the rapid surge of venous return and the subsequent increase in stroke volume results in capillary distension and transmission of otherwise filterable bubbles. Alternatively, one would have to postulate the existence of a subclinical, congenital abnormal anatomy.

From the Jo Ellen Smith Memorial Baromedical Research Institute and Jo Ellen Smith Medical Center, New Orleans, Louisiana. 70131, U.S.A.
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