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SEVENTH ANNUAL CONGRESS OF THE EUROPEAN UNDERSEA BIOMEDICAL SOCIETY AND SYMPOSIUM ON DECOMPRESSION SICKNESS

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9 SEPTEMBER 1981

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The Seventh Annual Congress of the European Undersea Biomedical Society and a Symposium on Decompression Sickness (the latter sponsored by the North Sea Medical Center, Great Yarmouth, England) was held at Churchill College in Cambridge, England on July 21-24, 1981. Approximately 50 presentations covered a wide variety of topics, with an emphasis on neurological decompression sickness. There was also a session on the medical aspects of amateur diving, and the program included a visit to the British Antarctic Survey for those interested. This report contains a brief summary of each paper.

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SEVENTH ANNUAL CONGRESS OF THE EUROPEAN UNDERSEA BIOMEDICAL SOCIETY AND SYMPOSIUM ON DECOMPRESSION SICKNESS

The Seventh Annual Congress of the European Undersea Biomedical Society (EUBS) and a special Symposium on Decompression Sickness presented by the North Sea Medical Center (NSMC) was held on July 21-24, 1981 at Churchill College in Cambridge, England. The meeting was sponsored by the Norwich Union Insurance Group. There were approximately 200 delegates from 16 nations from as far away as Australia and Japan for a meeting marked by superb organization, impressive international cooperation, and excellent scientific content. To avoid some of the Anglo-French language problems that have plagued past EUBS meetings, all speakers were asked (and the majority complied) to speak slowly and use a second screen as they gave their paper to show viewgraphs or slides containing a written summary of each point of the paper. This was an invaluable aid to understanding on both sides. Abstracts were made available in French and English.

Cochairmen of Session 1 were Dr. K. Seemann (West Germany) and Dr. J. Le Pechon (France). The first scheduled paper, which dealt with safety training and accident analysis for research diving over the past 20 years, was cancelled because the author, N.C. Flemming (Institute of Oceanographic Sciences, England) could not attend. His abstract indicates that the fatality rate among diving research scientists is 2 to 3 per 10,000 at risk per year, and decompression accidents due to poor medical conditions are rare. The first paper presented in Session 1 was by H. Örnhagen (Univ. of Lund, Sweden); he discussed the effect of body position in water on passive ear clearing and proposed anatomical and physiological reasons for confirmations of divers' impressions that passive ear clearing is better in the head-up than in the head-down position (air or water). R. Goad (Inst. of Naval Medicine, England) presented an investigation into relationships between chest dimensions and pulmonary barotrauma (in submarine escape trainees) that indicated that size and shape of the lungs and chest wall may prove to be influential in helping to predict which individuals might be at the highest risk in that particular training environment. J. King (London Hyperbaric Medical Service) reported on spirometric measurements taken on over 1,000 men during the course of commercial diving examinations, confirming previous impressions by Crosbie and Chemsit (on much smaller populations) that divers have higher values for forced expiratory volume in one second and forced vital capacity than nondivers (these are absolute values; unfortunately no data were presented on whether there is a change in the perhaps more important forced expiratory volume in 1 s as a percentage of forced vital capacity). There was a short audience discussion on the questions of whether present spirometric assessments are related to diver morbidity and mortality, whether current measurements are the appropriate ones, and whether a new set of standards is justified on the available evidence. C. Barnini (Sub Sea Oil Services, Italy) presented data collected during the course of working saturation dives over a 3-year period (92 divers examined). No long-term decrements that would lead to the conclusion that this type of diving is unsafe from a physiological standpoint were found, although few parameters—weight, blood pressure,

pulse rate, and subjective assessments were examined). Weight loss during saturation diving was reconfirmed, but an alternative explanation that this may have been due to actual work levels in industrial operations instead of hyperbaria per se was offered. The study was remarkable in terms of the amount of data collected in a field situation, a formidable task to say the least. M. Cross (Houlder Diving Research Facility, England) reported on some biochemical and hematologic responses observed in student divers participating in a 12-week basic air-diving course, including a slowly developing polychromesia, unassociated with increased reticulocytosis, which was thought to be due to erythropoietic inhibition in the first few weeks of the course. Audience discussion pointed out that a control population (i.e., nondivers living in the rather spartan conditions at the training center in question) might help to clarify some of these responses.

J. Paciorek (Kings College, London) presented some highly sophisticated work demonstrating that pressure exposure in men appears to increase the rate at which erythrocytes age. Her elegant work demonstrates that the formation of echinocytes in humans at pressure is due to degradation of rbc cytoskeleton protein. I. Thomas (Royal Victoria Infirmary, England) reported on work carried out to determine whether compression of bone marrow blood vessels occurs as a result of the volume changes in fat cells during exposure to compressed air that were previously demonstrated by this Newcastle-upon-Tyne group. The study demonstrated that while there was no change in total blood flow to the intact rabbit femur as a result of compressed air exposure, there was proportionately more flow to the cortex and less to the marrow. So this decreased marrow flow, relative to normobaric conditions, could theoretically permit entrapment of inert gas bubbles and be an important predisposing factor in the initiation of dysbaric osteonecrosis. Caution in extrapolating these findings to man was urged. M. Gennser's paper (Univ. of Lund, Sweden) about a study looking at the interaction between pressure and nitrous oxide on the contractility of rat atrial muscle concluded that, in the concentrations that might be required for anesthesia, significant decreases in contractility would be encountered. Submarine escape was treated in the next paper, given by P. Bell (Admiralty Marine Technological Establishment, Physiological Laboratory, England). While the current French approach to research into submarine escape from deeper water is to avoid decompression sickness (DCS) by slowing ascent rates with deployable drogue devices of some kind, the British feel this would make postsurfacing pickup more difficult, as survivors would be spread over a wide area of sea, and so they are looking instead at the possibility of using different gas mixtures without changing the current descent and ascent profile significantly. One of the gases that might be used is pure oxygen, assuming time-to-OHP (oxygen high pressure) convulsions is shorter than the escape profile (it also would have the advantage that oxygen bubbles, if produced, would resolve far faster than air bubbles). To this end rats were compressed in heliox-filled chambers, breathing pure oxygen, to depths of 350-400 msw (meters of seawater) on extended submarine escape profiles. The results demonstrated that above 10 bar, there appeared to be some protective effect against convulsions, thought to be due to the narcotic effect of oxygen, which might postpone the onset of fits by increasing the dosage required to produce a convulsion. However some disturbing late (and irreversible) convulsions were also seen in a few animals after

return to the surface from the deepest exposures. Work is continuing on animals by means of exposures to both pure oxygen and helium/oxygen.

The last paper of Session 1 was given by Dr. P. Bennett (Duke Univ. Medical Center, NC), and described the Duke "Atlantis" series of deep saturation dives designed to establish the relationship between nitrogen percentage and compression rate required to prevent the High Pressure Nervous Syndrome (HPNS). The most recent dive in this series was the milestone Atlantis III dive in January of 1981, which combined a slower compression rate with a "trimix," (helium, oxygen, and 10% nitrogen), and which was considered highly successful in terms of the functional abilities and work output of the three divers who took part. What was thought to be mild narcosis was seen at the record depths attained on this dive and the next logical step in the program would appear to be substitution of a somewhat lower nitrogen percentage at very great depths. Following his presentation, Bennett showed a 40-min. film entitled "Atlantis Three: 686 meters," a documentary style synopsis of the Atlantis program with major emphasis on the most recent dive in the series.

Cochairmen for Session 2 were Dr. H. Örnhagen (Sweden) and Dr. T. Nome (Norway). J. Norman (Inst. of Environmental and Offshore Medicine, Scotland) led off with a presentation on thermal comfort and deep-core temperatures in Antarctic scientific divers. During air diving under ice in 10 mm. wetsuits at depths of 6 to 36 msw for 20-25 min., the divers had an average core-temperature fall of 1 to 1.5°C with a further drop during transfer back to the base camp where they were rewarmed in hot water baths. Conclusions were that while they did not become dangerously hypothermic, subjective assessment of thermal status appears questionable, although "symptomless hypothermia" is generally not felt to be a problem as long as the skin is cold. There was a brief audience discussion highlighting the theoretical thermal problems in divers operating in the North Sea using hot water suits. The next presentation was given by J. Le Pechon (Compagnie Générale Doris, France), who described the empirical development of a commercial saturation diving schedule for operational use. Two hundred and thirty operational decompressions have been undertaken on this schedule and there were four cases of Type I DCS and one case of dysbaric osteonecrosis (though not all participants could be followed long term). It was not clear whether the schedule was developed in the laboratory or the field, but one would hope it was validated, man-tested, and accepted in the laboratory before being used operationally. There is a great temptation to change decompression procedures to fit operational requirements or economic realities, but the wisdom of doing so before long-term results of properly tested and established schedules are certain is questionable. The next paper, by M. Le Fur (Comex, France), described doppler detection of bubbles following no-stop air diving, a study that showed significant correlation between doppler signals and the percentage of body fat. Also, the greatest air depth at which saturation (defined here as 36 h) would not give rise to any detectable bubbles upon direct decompression to the surface was 6 msw (at 9 msw all subjects had bubbles, at 8 msw 50% had bubbles and at 7 msw one subject had bubbles; there were six subjects per dive). The next two papers also came from Comex, France. The first, given by B. Gardette, described a study designed to evaluate several different published air

diving tables. It was concluded that mean doppler bubble scores were not statistically significant for the schedules examined for any given dive (except when cold-water dives were compared to dry-chamber dives), there was great inter-subject variability (though, in general, the lowest bubble scores were seen in the fittest subjects with the lowest pulse rates), and diving on a regular basis seemed to decrease bubbling after a dive. The next Comex paper reviewed the Janus dive series and the development of the decompression tables for them. In general it was felt by the investigators that it was possible to relate bubble scores to delta P in dives up to 300 msw but not deeper. Dives deeper than 300 msw (for humans) and deeper than 800 msw (for animals, including several recent dives using baboons to as deep as 1,030 msw) call for a new method of determining acceptable decompression profiles, which will have to await a better understanding of physiological phenomena occurring during such very deep exposures. A paper by R. Guillerm (Toulon, France) reported the effects of local compression (via a specially constructed knee cuff designed to apply pressure to the joint without impeding arterial or venous circulation) on limb bends during saturation decompression. Pain disappeared immediately at a cuff pressure within the cuff of about 10 k Pa but reappeared after deflation. Basically the work was interpreted as supporting Brian Hill's hypothesis concerning the tendon site of bubbles in Type I DCS and the mechanical vs ischemic nature of the pain. The French also pointed out that Type II bends, in their experience, are well correlated with intravascular bubbles (a correlation not present with Type I DCS). A member of the audience mentioned that the same results might be expected if the pain were caused by subperiosteal bubble formation.

N. Rogberg (Foundation for Occupational Safety and Health in the Construction Industry, Sweden) contended that the more severe low-frequency hearing loss in divers (repeatedly exposed to noise levels of about 100 dB) as compared to nondiving construction workers (routinely exposed to similar noise levels) might be due to repeated bouts of aural barotrauma. As only air audiograms were made, it was not established whether the loss in his subjects was conductive or sensorineural. A presentation by W. Fraser (Defence and Civil Institute of Environmental Medicine, Canada) reviewed some remarkable micrographs of full-thickness fractures of the bony layers surrounding the semicircular canals resulting during decompression of squirrel monkeys from deep heliox dives (on a schedule designed to produce vestibular DCS). It was hypothesized that isochoric gas exchange (similar to isobaric gas exchange but in a rigidly confined space) is the most likely explanation for the generation of the extreme pressures (approximately 17 ATA) that would be required to cause bone failure. The possible relevance of these findings to dysbaric osteonecrosis was noted.

T. Hennessy (Admiralty Marine Technology Establishment [AMTE] Physiological Laboratory, England) reviewed the development of British saturation decompression profiles from depths in excess of 180 msw, culminating in the current 28 m/day rate, which is similar to the current U.S. Navy rate, though arrived at by a different experimental design. B. Hills (Univ. of Texas Medical School) elaborated on an hypothesis that the size of extravascular bubbles is influenced to a large degree by circulation in their proximity (i.e., they tend to grow in underperfused areas and shrink in

highly perfused areas) as well as by decompression. Furthermore it is probable that the intermittent flow observed in capillary bundles is a widespread physiological phenomenon and these facts taken together may help to account for the occasional case of bends in even the most conservative decompressions. J. Desola-Alá (Centre de Recuperacio: d'investigacions Submarines, Spain) next reported on a wide range of biochemical and hematological parameters studied in 67 cases of Type II DCS and 11 cases of lung barotrauma, in the hope that this information might prove to be of value in differential diagnosis and prognosis. In the end, the only conclusion drawn was that patients with the fewest parameters showing abnormalities responded best to recompression therapy. There was, however, no correlation between symptoms and either the number or severity of parameters altered. The final paper of the day by A. Palmer (School of Veterinary Medicine, Cambridge, England) reported on pathological findings in a single human spinal cord. The findings suggested that residual corticospinal tract damage in a diver who has previously sustained an episode of serious DCS might be greater than would be expected from a clinical neurologic examination. The implication of this is fairly important, but until more human material is correlated with reliable history, little more can be said, other than that this is an extremely important area.

Session 3 was cochaired by Dr. E. Hansen (France) and Dr. D. Elliott (England). The morning was devoted to papers coming out of "Deep Ex 80," the first comprehensive deep dive (300 msw) completed at the Norwegian Underwater Institute (NUI) in Bergen—a significant tribute to the NUI team, an organization that has grown rapidly and successfully in a country without a long history of previous involvement in the hyperbaric physiology field.

S. Tønjum (NUI) reported on a 10-h test of two commercial passive thermal protection systems for divers (rebreather-scrubber plus survival clothing and bags), both of which were adequate for this length of time at 8-10°C in a heliox environment at 31 atmospheres. The systems still have to be evaluated for the time they are supposed to keep a man alive in these conditions. A recent symposium at the Institute of Naval Medicine in England, to be reported elsewhere, cast doubts on the ability of current passive systems adequately to protect a man for 24 h. G. Bölstad (NUI) reported on the decrease of maximal isometric force in human skeletal muscle seen at pressure when cooled to a temperature not previously thought to be sufficiently low to have an effect on contractile force. A. Påsche (NUI) reported on the thermal condition of divers using hot water suits at 300 msw. The theoretical problem of "insidious" hypothermia (diver unaware of gradual cooling when skin kept warm but breathing insufficiently heated gas) could not be confirmed or demonstrated in this study. The study also pointed out once again the need for improvement in equipment when there is a long distance (a 90-cm long gas whip in this case) between the heater and the diver's mask inlet, because of the heat loss in the line (higher than thermo-neutral skin temperatures can occur when the breathing gas is heated to acceptable levels). Påsche also discussed body cooling rates at various temperatures and ambient pressures in hyperbaric heliox in 3 thermally unprotected subjects. Conclusions were that differences in cooling rates were most likely due to the variation in morphological characteristics

between the 3 subjects and that subjective evaluation of thermal status upon rewarming could be faulty. A paper on diver exercise capacity at 250 msw while breathing helium-oxygen or nitrogen-helium-oxygen from an underwater breathing apparatus was given by K. Segadal (NUI). Next there was a report by O. Molvaer (NUI) that suggested that the nystagmic response to caloric vestibular stimulation was transitorily reduced in Deep Ex 80, with return to normal noted upon surfacing, the cause being hyperbaric helium-oxygen (these findings are at variance with those reported by M. Rensink et al in 1977 at the Sixth International Congress on Hyperbaric Medicine in Aberdeen; however, the NUI workers plan to continue this work with improved controls and more finely tuned thermal conditions in the next deep dive).

The question of ferritin as a possible specific indicator of dysbaric osteonecrosis was investigated on Deep Ex 80 and the conclusions reported by E. Norild (NUI) were: (1) ferritin increased from the very beginning of a dive; (2) there are numerous causes of ferritin release in addition to osteonecrosis; (3) ferritin may increase as a result of compression as well as a result of induced embolization, so it is not a specific indicator of dysbaric osteonecrosis. Dr. R. Peterson (NUI) presented a synopsis of the compression-decompression trials done in conjunction with Deep Ex 80. Three divers were rapidly compressed on helium-oxygen to 250 msw, exhibited symptoms of marked HPNS but stabilized within 24 h, and went to 300 msw without further symptoms. The other 3 were compressed at the same rate on trimix (10% nitrogen) to 300 msw (where transient narcotic effects were evident), and then transferred to a helium-oxygen environment; there were no symptoms of HPNS. Investigators concluded (1) that rapid compression on heliox may still be a viable alternative for operational diving although the possibility of long-term effects of this procedure still needs to be investigated; (2) that 10% trimix eliminates the operational problems of HPNS but nitrogen narcosis and gas density problems still need to be overcome; (3) that no decision can yet be made about the feasibility of switching from trimix to helium-oxygen at depth, but further investigations are planned. In another part of the study investigators considered possible time dependence of unlimited duration excursions from saturation (i.e., will tolerance be lost if "crushed" micronuclei are allowed to regenerate). The results (three divers got mild type I DCS during an upward excursion 9 days after compression but had no problems on an identical excursion 4 days after decompression) indicated that further work on this question is warranted but that no changes to the US Navy procedures were recommended. The neuropsychological-neurological test batteries presented next by R. Vaernes (NUI) supported the subjective impressions reported by Peterson. The final paper in Session 3, given by Dr. A. Brubakk (NUI) resulted in a prolonged and spirited audience participation discussion. Brubakk reported on a pulsed doppler method used in Deep Ex 80 that reportedly detected more bubbles occurring during the first (4 days after compression) upward excursion than during the second (the opposite of what the "crushed micronuclei" theory would predict) as well as a remarkable number of "events" detected in the arterial circulation, including the aorta and carotid arteries. The investigators were convinced they were detecting bubbles although there were no signs of serious DCS. The post-paper discussion revolved around whether there were really bubbles and whether normal people frequently get

right-to-left shunts (with Valsalva maneuvers, etc). From the audience, Dr. Brian Hills (Univ. of Texas Medical School) pointed out that the brain can probably tolerate three or four times as much gas when bubbles are very small rather than large; nonetheless when bubbles are of the size that can be detected by doppler methods and are on the arterial side, in his experience physiological changes and symptoms would be expected.

The EUBS scientific program ended with Session 3 and with two poster presentations ("The Histology of Lesions found in Animals following Rapid Decompression" by R. Bennet, Dundee, Scotland and "Modification of Sleep Profile of Cat under the Hyperbaric Environment" by K. Seki and H. Nakayama, Japan Marine Science and Technology Laboratory, Japan).

The North Sea Medical Center (NSMC) Symposium began on Thursday afternoon. Session 1 was chaired by Dr. I. Anderson (NSMC, England); he opened by summarizing the history of the NSMC and the reason for the symposium. The NSMC is clinically orientated and has achieved a well-deserved reputation in the field of diving medicine and offshore support over the past decade. The center is equipped for medical examination of offshore workers; training courses (which have been very successful) for nurses, diving supervisors, and diver-medics; advising companies regarding employee health and national regulations; emergency topside medical coverage; periodic diver medical examinations; and medical coverage for divers in the North Sea (and on occasion, farther afield). The need for a symposium on neurological decompression sickness was put forward by the NSMC because of its increasing experience and need for more information and a wider range of opinions on this important condition, which is unique among neurological diseases in that (1) there is little information in standard medical text books concerning it and (2) it is a frequently reversible neurological disorder seen in a young and otherwise healthy age group. Anderson related that between 1966 and 1980 approximately 280 diving accidents were seen by NSMC personnel. Of these there were 136 cases of DCS, 9 cases of pulmonary barotrauma, and 9 cases of arterial gas embolism. Forty-two of the cases of DCS were classified as CNS DCS, of which 21 improved dramatically with initial treatment, and in 11 cases the affected divers could not return to diving. Therefore the scope of the problem was considered sufficient to justify the present symposium.

The leading paper in Session 1 consisted of a historical review of neurological DCS in which Dr. P. James (Ninewells Medical School, Scotland) explained much of the work that has attempted to establish the pathophysiology responsible for DCS affecting the Central Nervous System (CNS) (arterial occlusion, vertebral-venous occlusion, autochthonous bubble formation). There followed a superb review of the applied anatomy and physiology of the spinal cord by Dr. M. Swash (The London Hospital, England). Next, Dr. N. McIver (NSMC, England), presented three case histories of neurological DCS which were then analyzed by Swash. Any hypothesized pathophysiology of the condition must take four factors into account: (1) the patchy and diffuse nature of the lesions that are often seen; (2) the relative contributions of intra- and extravascular mechanisms of damage; (3) preference of the condition for certain areas of the spinal cord and (4) clinical observation of the patterns of response to treatment, of recurrences, and of residual effects.

Dr. A. Palmer (School of Veterinary Medicine, Cambridge, England) made a case for an essentially vascular phenomenon being responsible for spinal cord lesions of DCS (necrosis due to infarction from platelet aggregation and thrombus formation in both gray and white matter with an associated widespread vasogenic edema). The gray matter lesions appear to resolve faster than lesions in the white matter, so the phenomena of localization and preferential sites of spinal cord DCS may simply reflect the total amount of white matter at risk in certain areas of the spinal cord. In reaching these conclusions, Palmer studied 27 goats with spinal cord lesions produced by rapid decompression from 30 msw for 60-min. dives. Some animals were sacrificed immediately (both at depth and within a few minutes after decompression), others between 24 and 48 h after the dive, and finally, some at intervals of 5 days to 6 months. He was able to study some animals that, clinically, appeared to have recovered completely after 6 months but showed significant pathology at autopsy. Pathologist I. Calder (Health and Safety Executive, England) discussed spinal cord lesions in man, pointing out that most human material that is studied either comes from hyperacute cases (i.e., blow-up with complete mechanical destruction of material) or is poorly preserved, which makes the investigation difficult. *Chronic* material is also extremely sparse. Nevertheless, the material he has been able to study, both acute and chronic, tends to support Palmer's interpretation of the animal data and also tends to support the previous allusions to the "iceberg" phenomenon (i.e., that damage is likely to be greater than would be expected from clinical examination results).

Session 1 produced a prolonged disscussion. In general, all participants seemed to agree that a CNS damaged diver, even with minor residual defects, should always be barred from further pressure exposure, and many were skeptical of returning the "clinically cured" man to diving. There were one or two anecdotal stories of a diver with a spinal cord lesion treated successfully (with prompt and complete recovery) who many months later had a second "hit" in the same location as the first. Most would accept that cord damage can be more severe than one might think, but it was also pointed out that for years people have gone back to diving 6 to 8 weeks after prompt and full recovery from an appropriately treated cord lesion, and have successfully completed full diving careers with no further problems. It was further argued that residual ischemia, if present, might reasonably be expected to recur upon decompression and reversion to a normal oxygen partial pressure after a successful cure. Counterarguments ran along the lines that an organism might function well with small lesions but when redundancy of the CNS was reduced, a *second* incident could leave much greater deficits than the first, an important question to consider from viewpoints of litigation and fitness-to-dive decisions (the comparison being that many patients who suffer their second stroke or cerebro-vascular accident, even if it is a relatively trivial one from an anatomical point of view, do not recover nearly as much function as they did after their first accident). Discussion then went on to the consideration of long-term and repeat hyperbaric oxygenation (was it of proven value or would the end result be the same without it?). The only way to answer the question with certainty would be a prospective double-blind study; however, there appeared to be agreement that it *should* be used because marginally viable (and perhaps nonfunctioning) neurones might be saved that would otherwise not recover, making the final

deficit worse than it need be. Prof. C. Lambertsen (Univ. of Pennsylvania Medical Center) summed up the discussion and made his own observations with the following points: (1) the session had reaffirmed that CNS DCS is an infinitely variable disorder, ranging from being clinically undetectable to severely incapacitating and irreversible, and that no doubt this is due to the complexity of the human nervous system; (2) given this premise, we should apply the same type of thinking to our treatment approach, that is, we should not attempt to concoct a treatment highly specific for a single favored causative mechanism (i.e., a bubble in a vessel or in myelin or in an axon, etc.) but rather we should make rational use of the whole armament available to us: drug therapy, fluid and electrolyte balance and so on, in addition, of course, to pressure and oxygen.

Session 2 was cochaired by Sir J. Rawlins (England) and Dr. X. Fructus (France). W. Keatinge (London Hospital Medical College, England) discussed hypothermia in general, and in divers in particular. His presentation was oriented more towards the professional deep-water diver than to the recreational shallow-water diver, yet it aroused great interest and a fairly long discussion. One important point that was brought out (and which, I believe, is already being acted on by at least one diving equipment manufacturer) was that a combination of closed-circuit hot water suit and a small diver-mounted immersion heater could largely eliminate the problems of both skin burns and the possibility of hypothermia in deep-helium-oxygen-breathing professional divers. The next presentation by M. Harries (Guys Hospital, England) was easily the most entertaining (and one of the most thought-provoking) of the entire week. This gifted lecturer gave an extremely practical talk on drowning and resuscitation in far from ideal settings, and in view of his previous experience as a longtime lifeguard and member of the British International Surfing Team combined with his position as a lecturer in medicine at Guys, the conference organizers could not have made a better choice for this particular subject. The messages of airway management (difficulty with airways in over half of all drowning victims), how and why people drown (very quickly, and usually as a result of lack of responsibility), use of oxygen for all drowning victims (notwithstanding recent controversy over oxygen in ambulances), and his views on long survival times with (young) drowning victims in cold water (respiratory arrest but protected by intact circulation) were put across in a manner the audience is not likely to forget soon.

R. Pearson (Inst. of Naval Medicine, England) talked on the subject of diving accidents and accident management in the British sport-diving population and was sharply critical of the irresponsible attitude of certain divers and, at times, of their training organizations—a criticism which was certainly justified when he described several incidents wherein reckless, uninformed, or simply idiotic behavior resulted in accidents that should not have happened (1981 is thus far proving to be a banner year for similar accidents!). There was no one in the audience who could argue with the facts and statistics presented; it was encouraging that representatives of the two largest British diving clubs said that recognition of the things he was talking about was already leading to changes in attitudes and policies, and that a major goal of both organizations for the future was increased education of their members. Increased education

of the medical personnel who are potential health-care providers for divers and other groups exposed to hyperbaric conditions is equally important, and a step toward providing that education is provided by just such a forum as this. The last paper of the evening was given by R. Goad (Institute of Naval Medicine, England) and addressed the subject of fitness for amateur diving.

Session 3 was opened by Dr. N. McIver (NSMC, England) on Friday morning. Dr. T. Shields (AMTE, Physiological Laboratory, England) reviewed the development of the use of pressure and oxygen in the treatment of DCS as well as a summary of current therapy (concentrating on British, French, and American experience and purposefully omitting ancillary therapy, which was to be covered later in the session). Shields described two interesting cases of Type II DCS that initially worsened with hyperbaric oxygen (presumably due to vasoconstriction of marginal-flow areas in the damaged tissue), though both cases eventually showed good response to long-term hyperbaric oxygen. P. James (Ninewells Medical School, Scotland) then described some problem areas in therapy, concentrating on inappropriate treatment of deep heliox bounce diving-induced DCS with (1) inadequate recompression or (2) recompression on air, or both. Adequate recompression on helium-oxygen for this type of fulminant DCS from heliox bounces or blowups has long been standard therapy, but the cases are so few that at times the principles have been forgotten, with predictably tragic results. Hopefully the work Dr. James has done in presenting unusual cases will help prevent similar mistakes.

A paper titled "Scientific Considerations in Recompression Therapy" was given by Dr. B. Hills (Univ. of Texas Medical School). An important concept in this talk was Hill's hypothesis that the primary factor determining what effect a particular breathing gas will have on bubbles already present is the solubility of the inert gas or gases in the mixture (i.e., on the basis of solubility a primarily nitrogen bubble will shrink if you breathe helium, which is much less soluble in blood than nitrogen). On this basis, and referring to Dr. James's cases, he contended that one should not ignore a procedure (that is, breathing heliox as part of the treatment for air DCS) that seems to work well on clinical grounds, because of various theoretical counterdiffusion theories that don't seem to work in practice. For my part, this is an area of confusion, and Dr. Hill's arguments may be a step in the right direction. In the next paper J. Wolkiewiez (France) described his group's experience in treating DCS and concluded that both Type I and Type II cases have better end results when oxygen, aspirin, and fluid replacement are used enroute to the recompression facility. This is a view we have long shared and that I believe has gained widespread acceptance and more publicity over the past few years.

The final paper of the Symposium, the keynote address, was given by Dr. A. Bove (recently of Temple University, Philadelphia, PA, and now of the Mayo Clinic, Rochester, MN) and was perhaps the best scientific presentation of the week. Bove spoke on the basis of drug therapy in the treatment of DCS. The presentation was reminiscent of John Hallenbeck's review of fundamentals of HPNS at the Seventh Underwater Physiology Symposium in Athens in 1980 (the proceedings have recently been published by the Undersea

Medical Society and are invaluable for the comprehensive review articles alone, review articles by Hallenbeck and others prefacing each major topic). Bove's lecture began with the presumption that no ancillary therapy can replace recompression in the treatment of DCS, but there are a number of adjunctive measures that can help. His task was to review the use of oxygen, fluid therapy, steroids, various anti-inflammatory agents, anti-coagulants, and other miscellaneous agents, all of which alter the inflammatory response in some way (and so lessen the effects of DCS, which is presumed to trigger an inflammatory response both by direct tissue aggravation by nucleated bubbles and by indirect blood-bubble interactions). There followed a review of the general principles of inflammatory responses and pathways and of relevant drug actions. It was pointed out early in the presentation that mild DCS, as well as more serious forms, could be expected to benefit from adjunctive therapy (i.e., inflammation produces pain, so it is not always necessary to postulate bubbles in joints to explain Type I DCS pain). Many recurrences of DCS fit the time frame of edema formation due to permeability changes and vascular leakage in the inflammatory response (supporting the French experience of using anti-inflammatory adjunctive measures early in all cases of DCS). In serious DCS, total blood volume falls within 2 1/2 to 4 h (easily measured by a concomitant hematocrit rise) and platelets fall as well (responses which are not seen in mild DCS, probably because inflammatory responses are kept localized whereas in more serious cases the response is widespread). Recompression alone will not reverse the presence of microthrombi and edema (demonstrated by some beautiful SEM pictures) so other measures are needed: measures that combat the important aspects and mediators of inflammation (i.e., increased vascular permeability, chemotaxis and leukocytic infiltration, tissue damage by lysosomal products, etc.). Bove made a plea for people in our field to keep abreast of and utilize the vast amount of anti-inflammatory research that is going on all over the world.

Initially, the most important aspect of therapy is the optimal application of pressure and oxygen. More work is needed to determine the optimal levels of these two variables, but, in the meantime, schedules such as COMEX Table 30 are useful backups, especially when recompression deeper than 18 msw is required. In this case the more optimal oxygen content (as opposed to compressing deeper on air alone) can help reduce ischemia and remove inert gas. Fluid replacement (IV started on site, at an adequate rate to keep up a 100 cc/h urine output—catheterized if necessary) is important in replacing depleted volume, restoring hematocrit, preventing blood sludging, and improving tissue perfusion. It is important to remember that while plasma is lost as a result of DCS, divers are also frequently dehydrated even *before* the dive, a situation that usually worsens during transport to a treatment chamber. So fluid shifts plus inadequate intake generally mean that replacement with crystalloids is preferred over colloid replacement. Saline or Ringers solution (not lactated Ringers because lactate levels are already rising) are the fluids of choice. Problems with the Dextrans include the possibilities of allergy, of increased bleeding, and of fluid overload (i.e., pulmonary edema—the danger being that Dextrans cannot be removed rapidly by diuresis as can saline or Ringers solution).

Steroids are useful in opposing permeability increases, augmenting catechol responses, inhibiting leukocyte "stickiness", and stabilizing lysosomal membranes. Glucocorticoids should be used in preference to salt-retaining steroids. Dr. Bove noted that steroids only oppose the extracellular edema produced by increased vascular permeablility and that cellular CNS swelling is best treated with oxygen. It is rare to get side effects with a short course of steroids, therefore Bove believes that, despite theoretical arguments, it is always best to give the diver the potential benefits of steroids. The recommended dosage was 10 mg IV of Decadron immediately, followed by 4-5 mg every 6 h. I would add here that, personally, I always cover seriously ill divers, even on short courses of steroids, with antacids, as they are often in a high state of physical and physiological stress over and above the side effects produced by the steroids. Bove next discussed the four main classes of non-steroidal anti-inflammatory agents (aspirin, indomethacin, phenylbutazone and ibuprofen), all of which act as inhibitors of platelet aggregation, blockers of prostaglandin synthesis, and anti-inflammatory drugs. He urged the audience always to consider the benefits vs the side effects for all these drugs and to try to keep abreast of new drugs with fewer side effects, which are constantly being introduced.

The anticoagulants discussed were intravenous or subcutaneous heparin and the antivitamin K agents, which have the advantage of oral administration but take several days to wear off. A recommendation was made that anticoagulants *not* be used initially as they might worsen hemorrhagic spinal cord lesions (even in a hospital setting, heparin often causes bleeding and is frequently hard to control). Low-dose heparin (5,000 units every 8 h) may be beneficial over the long term to the diver immobilized in the hospital with paraplegia to prevent pulmonary embolism and thrombophlebitis. Finally, a discussion of other miscellaneous agents included the use of prostaglandin inhibitors with specific anti-inflammatory properties, antihistamines (their side effects include drowsiness, but they should still prove useful), anti-serotonins, and antibradykinins (i.e., Trasylol—efficacy has not been proven). The audience was urged to consider all new agents introduced in the future that have anti-inflammatory properties.


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