

*FITNESS TO DIVE*

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Undersea and Hyperbaric Medical Society Workshop

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*Recommendations found in this workshop are not intended to imply that justifiable deviations from them by physicians experienced in diving medicine under appropriate circumstances represent a breach of a medical standard of care. Furthermore, they are not meant to imply that new knowledge, critical research data, clinical experience, or clinical circumstances may not provide sound reasons for alternative approaches.*

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## CHAIRMAN'S FOREWORD

This workshop on Fitness to Dive brought together approximately twenty Undersea Medical Specialists, all physicians, most board certified in a medical specialty representing a variety of medical disciplines, and all currently active in the day-to-day care of divers. Most significantly, all are continuously faced with the decision-making process for determining individual divers' fitness to dive. These physicians were chosen by the chairman to participate in the workshop because of these specific qualifications.

Although the special categories of Military Diving and Commercial Diving did come up in the course of discussion, the main thrust of the conference was the development of a set of Consensus Standards for UNRESTRICTED SPORT DIVING, in terms of an individual's safety to be exposed to increased ambient pressure in an aquatic environment.

Diving for the "disabled" was not considered as an appropriate topic for this conference, as the types and degrees of disability are essentially unlimited and it was unanimously felt that it would be more appropriate for each "disability category" to establish its own special standards and policies, training methodology, and fitness criteria, perhaps, if requested, with the guidance and assistance from the Undersea and Hyperbaric Medical Society.

It was also agreed unanimously that these Consensus Standards are presented as guidelines, and are not to be considered in any fashion a "regulation" or formal policy of the Undersea and Hyperbaric Medical Society.

The recommendations listed under separate categories hereafter should be considered to represent a consensus of informed opinion from physician specialists active in the practice of Diving Medicine, and most of whom are active divers themselves.

A brief statement as to why any medical standards should even be considered is appropriate. An individual who enters the underwater hyperbaric environment imposes on himself a variety of physical, psychologic and pathological stresses that have been identified, well described in most cases, and are common knowledge to most diving professionals including diving instructors and divemasters. When this individual places himself in or under the water he is at great disadvantage with regard to his ability to propel himself through the dense medium and to protect himself against excessive heat loss, both of which require high energy expenditure rates. The individual must breathe gases of increased density in or through appliances to maintain adequate gas exchange of oxygen, carbon dioxide, and inert gases and must utilize a cardiorespiratory system that has been altered as a result of exposure to the direct effects of hydrostatic pressure. The individual must be able to adequately accommodate changes in gas volume and pressure in his anatomical and pseudo-anatomical spaces to prevent morbid changes. Lastly, he must function within narrowly defined limits imposed by the indirect effects of pressure,

i.e., the partial pressures and solubilities of gases, which cause toxic, narcotic, or stimulatory alterations to bodily functions.

Because of these obligatory stresses of underwater exposure, an individual must possess certain physical and physiological attributes and must be devoid of certain limitations or conditions in order to function safely in this unique environment. In this workshop we considered the physical requirements and limitations with reference to two major categories. The first concerns those requirements and limitations that affect everyone exposed to pressure, no matter if one is dry, wet, in a deep sea-rig, in a caisson, or enjoying "wall-dive" at a Caribbean resort or making a boat dive in the kelp off Southern California. The second major category concerns limitations or influences the requirements that an individual needs to perform diving in or under water, which clearly is not man's natural environment.

The group clearly realized, that unlike the United Kingdom, Australia, New Zealand, and other countries which require a "medical clearance" to engage in diving activity, no such standard exists in the United States. Most United States recreational diving training agencies do not require an individual to have clearance by a physician before entering training. Most diving training agencies rely on a brief health questionnaire and any positive answer or reasonable doubt on the trainee's part as to how to answer the specific questions may require clearance by a physician. It was not within the scope of this conference to discuss the training agencies philosophies regarding the need for or lack of need for physical evaluation before entering training. We felt this was strictly their business, and they are well qualified to seek their own advice on this subject. However, it was unanimously felt that some sort of improved health screening to engage in unrestricted recreational diving was appropriate and it was hoped that the guidelines herein recorded might serve as a basis for improvement.

The subject of the American Philosophy of, "I have the right to do what I want to do," came up several times during the discussion. The group unanimously felt that impaired or handicapped divers do NOT have the right to inflict on others the risk of rescue nor to inflict liability on their trainers, transporters, or providers of equipment without them being fully cognizant of the diver's detriment or handicap.

The consensus standards may be considered too strict by some, too lax by other; too specific by some, and incomplete by others. The reader is reminded that these recommendations are offered as a consensus of informed opinion of physicians active in diving medicine concerning the health and safety aspects of persons exposed to increased pressure in an aquatic environment. For the discussion and reasons for these consensus opinions the reader is encouraged to study the specific papers in this report as well as the attached limited discussion for each subject.

SUMMARY OF THE CONSENSUS STANDARDS FOR UNRESTRICTED DIVING  
SPECIFICALLY MEANING EXPOSURE TO INCREASED AMBIENT  
PRESSURE IN AN AQUATIC ENVIRONMENT

The term "contraindicated" is equivalent to the term disqualified. Other categories or phraseology imply a judgmental decision based on the individual condition and experience, the training status of the diver, or medical judgment which is in the best interest of the diver.

I. General

- A. Age: It is difficult to set up a minimum age for diving because of wide individual variation in physical development, strength, psychologic maturity, and intelligence. From a practical standpoint, several established diving training agencies will train 12-year-olds in the company of a responsible family adult 18 years or older, leading to a *junior diving* certification. This is considered reasonable. Individuals over age 45 deserve careful medical evaluation, and the application of the principles of physiologic age and performance criteria is more appropriate than chronologic age.
- B. Sex: An individual's sex, per se, should not affect election for any type of diving. Pregnancy in diving is a consideration and is a subject expressed elsewhere in the medical literature as well as in previous Undersea and Hyperbaric Medical Society workshops.
- C. Body Build: There are no specific standards except to remind the public that obesity represents a hazard to divers because of the affect on inert gas exchange and its relationship to decompression sickness as well as being a reflection of general physical fitness and total general health.

II. Neurologic Conditions:

- A. Epilepsy:
  - 1. If epilepsy is required to be controlled by medication, diving is contraindicated. (This includes childhood seizure disorders requiring medication.)
  - 2. A possible acceptable risk would be a patient with a history of febrile seizures in infancy, apneic spells or seizures attendant to acute illness such as encephalitis and meningitis, all without recurrence without medication.



B. Migraine Headaches:

May be disqualifying if frequent, severe, or with complications of visual field loss, vomiting, hemiplegia, or loss of consciousness.

C. Head Injury:

1. Disqualifying if prolonged unconsciousness or post-treatment amnesia or residual neurologic symptoms or findings prevail.
2. Possibly an acceptable risk if the unconsciousness was less than one minute and if the post-traumatic amnesia was less than one hour.

D. Spinal Cord Dysfunction:

Probable increased risk of decompression sickness exists whether the myelinopathy results from trauma, demyelinating disease (multiple sclerosis) or from vascular compromise.

E. Spinal Surgery:

1. Probable increase of risk from structural overload:
  - a. Anterior interbody fusion
  - b. Lumbar laminectomy
2. Possible increased risk for decompression sickness from lumbar laminectomy.

F. Stroke and transient ischemic attacks will be disqualifying.

G. Intracranial aneurysm, arterial-venous malformation or tumor will all be disqualifying.

H. Craniotomy for any cause will have attendant possible increased risks for decompression sickness or mechanical trauma.

I. Decompression Sickness Type II CNS Involvement:

1. Disqualifying if residual neurologic symptoms or findings persist.
2. Possibly increased risk if symptoms remit after single standard treatment table or after 24 hours postaccident (concept of increased susceptibility at site of previous injury).

III. Ear, Nose, and Throat

A. External Ear:

1. Pinna--Any infection of the pinna or injurious states such as unhealed lacerations or frostbite would be disqualifying until resolved.
2. External Auditory Canal--Otitis externa, seborrhea, cerumen obstruction blocking more than one-half of the external auditory canal cross sectional area, and obstructive exostosis all will be disqualifying until medically or surgically resolved.

- B. Middle ear--Recurrent otitis media or recurrent otorrhea within the past three months should be disqualifying because of the possibility of attendant poor eustachian tube function. Absolute contraindications would be a tympanic perforation. If the tympanic perforation were repaired by tympanoplasty it would still be disqualifying. However, if the simple myringoplasty were performed with success, the patient could be cleared for diving. Absolutely disqualifying would be the presence of tube myringotomies. If the patient had a history of previous mastoid or middle ear surgery, this would be disqualifying. Specifically, stapedectomy would be disqualifying, as would mastoidectomy with permanent fistula.

Further, the patient should be excluded from diving if a prevailing facial nerve paralysis persists for any reason. If the patient has idiopathic facial nerve paralysis with resolution, then the patient may be cleared for diving if there is no sign of deficit or recurrence after an appropriate wait.

THE PATIENT SHOULD BE SUSPECTED OF HAVING ABNORMAL MIDDLE EAR FUNCTION IF THE PATIENT GIVES A REPORT OF HAVING TROUBLE DURING DESCENT FROM ALTITUDE IN AIRCRAFT AND SHOULD BE REFERRED TO A DIVING PHYSICIAN FOR EVALUATION, SPECIFICALLY TO A DIVING PHYSICIAN SPECIALIZING IN OTOLARYNGOLOGY.

- C. Inner Ear--If the patient is found to have a history of tinnitus or deafness, fluctuating hearing or vertigo, he should be sent for evaluation to a diving physician in the ear, eye, nose and throat specialty for further testing with audiometry by air and bone conduction and for evaluation by electronystagmography. Disqualifying will be a history of any inner ear surgery which involves shunts, cochlear sacculotomy, or diagnostic evidence of temporal bone or posterior fossa tumors. Further disqualifying conditions would be the diagnosis of Meniere's disease, labyrinthine round or oval window rupture, benign postural vertigo, sudden hearing loss, vestibular neuronitis, or otosclerosis. All of these diseases would be disqualifying unless completely resolved.
- D. Nose and Paranasal Sinuses--Temporarily disqualifying would be the presence of an upper respiratory tract infection, unresolved epistaxis, and nasal obstruction or dental disease which precludes comfortable maxillary-mandibular occlusion on a mouthpiece. Further, until corrected, severe nasal mechanical obstruction by polyps or deviated septum, intranasal use of illegal drugs, or frequent bouts of nasal discharge must all be evaluated and treated and be disqualifying until resolved. A rule of thumb is that if the diver needs decongestants for clearing his nasal and sinus spaces before diving he should be evaluated by an ENT specialist.
- E. Laryngopharynx--Absolute contraindications to recreational compressed air diving in an aquatic environment would be upper airway obstruction or severe persistent hoarseness with laryngospasm as well as tracheostomies, partial laryngectomy with decannulation and healing of tracheostomy. Laryngocele would be contraindicating. Chronic motion sickness would be an absolute contraindication. The proviso is that drugs known to alleviate motion sickness are not without possible deleterious side effects which would cause problems for the recreational diver diving on compressed air in an aquatic environment.

Screening audiometry which discerns a loss of 25 Db ISO of speech frequencies between 500 and 2,000 Hz should be sent for evaluation to an otolaryngologist. Further speech discrimination audiometry that shows a deficiency of 90% predicted requires otolaryngologic evaluation. Asymmetric loss above 2 kHz should again be referred to a specialist as well as symmetric loss above 2 kHz.

#### IV. Cardiovascular

##### A. Arteriosclerotic heart disease:

Diving is contraindicated in those persons with exertional angina, postmyocardial infarction with left ventricular dysfunction, congestive heart failure, or who require medication to control dysrhythmias. Postcoronary bypass surgery with violation of pleural spaces is also contraindicated. All other cases involving arteriosclerotic heart disease require decisions made between diving medicine specialists and cardiologists and based on exercise testing.

##### B. Hypertensive vascular disease:

Diving is permitted in those cases easily controlled by medication of a nonsedative or ganglionic blocking type. In all cases, the final decision should be performance-criteria oriented. For definition purposes hypertension is defined as blood pressures in excess of 150 mmHg systolic and 90 mmHg diastolic.

##### C. Valvular heart disease:

Performance criteria applies with the exception of aortic and subaortic stenosis in which there is an increased risk of sudden death. There is also the possibility, in stenotic and regurgitant murmurs, of turbulence causing gas nucleation and increased risk of decompression sickness. The decision to allow an individual with such a lesion to dive requires consultations between a cardiologist and a diving medicine specialist.

##### D. Other cardiac conditions:

The reader is referred to Dr. Bove's discussion on cardiovascular diseases in this publication. In general, however, the ability to perform exercise up to 13 METS is considered a reasonable minimum for engaging in diving activities from a performance-criteria point of view. The methodology for performance testing is at the discretion of the physician.

V. Pulmonary:

A. Asthma:

Because of the logical catastrophic consequences of "reactive airway disease" in a diver, breathing decompressed gas underwater, regardless of etiology, makes asthma a contraindication to beginning or continuing diving. When there is doubt as to whether asthma exists or whether an individual has "outgrown" the condition, a standardized methacholine or histamine challenge should be required and *passed* before diving can be recommended.

B. Spontaneous pneumothorax:

A history of spontaneous pneumothorax is considered a contraindication to diving. Traumatic pneumothorax is also considered disqualifying with the possible exception of "deserved" pulmonary over-pressurization diving accidents, which require individual considerations, and special testing in conjunction with a diving medicine specialist and pulmonologist.

C. Obstructive and restrictive pulmonary diseases:

Decisions on candidates with these diseases should be based on performance criteria, including pulmonary function testing and exercise tolerance testing, and obviously needs consultation between diving medicine specialists and pulmonologists.

D. Chest x-ray:

The group strongly recommended a baseline 14" x 17" PA chest x-ray for all diving candidates.

VI. Metabolic disorders and miscellaneous medical conditions:

A. Diabetes: Absolutely disqualifying will be an insulin-dependent or an oral-hypoglycemic-agent-controlled candidate. Diabetics controlled by diet alone can be cleared for diving in a compressed air aquatic recreational environment.

B. Any other endocrine dysfunction needing medication for control should be referred to an endocrinologist familiar with diving medicine before clearance can be given.

- C. Any hematologic disorder which alters rheology, gas exchange or coagulability of blood should be cleared by a hematologist familiar with diving medicine. A history of anemia with a hematocrit lower than 33 and a hemoglobin of 11 in the male would be referred to a hematologist for further evaluation before clearance for diving. Patients with hemoglobinopathies (especially Hgb SS) shall be disqualified. Patients with polycythemia greater than a hematocrit of 55 will be disqualified. Thrombocytopenic patients will be referred to a hematologist for further evaluation before clearance for diving.
- D. Obese patients will not be disqualified per se but should be evaluated further by performance testing on a case-by-case basis.
- E. Musculoskeletal disability:
1. Metabolic bone diseases that compromise circulation will be disqualifying.
  2. Any infectious disease of bone that compromises circulation will be disqualifying.
  3. Any fracture or bony injury that compromises circulation will be disqualifying.
- F. Renal disability:
- End stage renal patients will be disqualified. Considered at risk for increased injury from decompression sickness or from gas embolism or trauma would be candidates with a renal transplant.
- G. Ophthalmologic considerations:
1. Candidates with recent keratotomy or ocular surgery would be disqualified.
  2. Candidates with abnormal visual acuity which can not be corrected or who would be disabled should they lose their corrections while diving should not be cleared for diving.
- H. Hernias that could strangulate must be repaired before clearance for diving can be given. Anyone with an acute gastro-intestinal problem should not be cleared for diving until it is resolved. Colostomy and ileostomy patients may dive.

VII. Psychiatric Disorders:

A. Disqualifying:

1. Alcohol and drug abuse or dependency.
2. Thought disorder--schizophrenia.
3. Affective disorder--severe depression.
4. Personality disorders (for example, sociopathy).
5. Psychoneurosis to include phobias and anxiety attacks.
6. Any candidate under treatment with psychotropic medication.

B. Possibly disqualifying because of increased risk:

1. Affective disorders--mild depression.
2. Uncertain motivation.

ANY CANDIDATE WITH A HISTORY OF CHRONIC ILLNESS SHOULD BE REFERRED TO A DIVING PHYSICIAN FOR EVALUATION AND CLEARANCE FOR COMPRESSED AIR AQUATIC RECREATIONAL DIVING. LIKEWISE, ANY CANDIDATE ON MEDICATION ON A LONG-TERM BASIS SHOULD BE REFERRED TO A DIVING PHYSICIAN FOR CLEARANCE. CANDIDATES SHOULD BE ENJOINED TO LIST ALL OF THE MEDICATIONS THEY HAVE TAKEN IN THE LAST SIX MONTHS BEFORE THEY ARE DECLARED MEDICALLY FIT TO DIVE.

It is realized that the above list is far from complete, but for further opinions regarding specific conditions, discussion of relative vs. absolute contraindications of diving the reader is referred to the specific chapters that follow.

## CHAIRMAN'S REMARKS

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It is with a great deal of anticipation that as chairman, I call to order this workshop on the Fitness to Dive sponsored by NOAA. When first contacted at the Long Beach meeting in 1985 by Dr. Shilling, the workshop was supposed to pertain to the sport diving and research diving communities only and was scheduled to follow a workshop on the disadvantaged entitled, "Scuba for the Disabled," an interesting temporal arrangement that caused me some anxiety.

In the funding request document, it was stated that all forms of diving would be considered, specifically including, Commercial, University Research, Sport, Habitat Saturation and Tunnel Work. Encompassing in one symposium essentially all forms of diving activity, to be covered in two working days made me even more uncomfortable. Then, happily, I recalled the old Navy expression, "rank hath its privilege," and as chairman I decided to modify the scope of this workshop to defining fitness to dive in "generic terms", i.e., general fitness criteria for exposure to increased ambient pressure and/or the underwater environment, to be based on best medical fact and judgment.

Further, I used the chairman's prerogative in recommending for participation on the workshop panel only physicians, and only those who were experienced and have been actively working with divers on a day-to-day, hands-on basis, or those experienced in evaluating disability or establishing fitness to return to diving, or medical-legal experiences with diving. Due to fiscal constraints, many of the suggested participants were not able to be formally invited.

I hope to complete this workshop with a basic set of consensus standards that the Undersea and Hyperbaric Medical Society can offer to inquiring individuals or groups and on which special interest groups can expand, or can retract from, as the case may be, to satisfy their own special requirements. To accomplish this they may need to sponsor other workshops. I hope that research directions and needs will also be identified during this workshop.

I have been subjected to unusual pressures from special interest groups to include persons on this panel to speak to their own needs and aims. As you can see from the assembled, I did not change my criteria for participation. I think it is essential if we are to properly fulfill our mission, that we not be influenced by social, economic, insurance, or political factors but that we derive our consensus recommendations based on sound, Preventive Medicine principles and judgment. It should be clearly recognized that we will not be preparing any "regulations" or establishing



"policy" for the Undersea and Hyperbaric Medical Society or anyone else, but will simply be expressing our best opinions, including provisions for minority dissenting opinions from those who have strong feelings regarding any proposed standard.

In selecting the topics and speakers, pro and con positions were considered and anticipated. Those of you who are not "picked" for a specific topic should not feel slighted because you will have ample opportunity to speak to your own ideas and opinions in the discussion following each presentation, particularly in the plenary sessions tomorrow.

It is hoped that we can develop a reasonable set of practical medical criteria for advising people whether it is safe for them to engage in underwater activities or be exposed to increased ambient pressure environments. We must consider whether a medical evaluation versus a simple questionnaire fulfills a fitness assessment. Last, tomorrow we must decide whether to produce a document duplicating, modifying, or updating published guidelines or whether to restrict ourselves to controversial or new areas.

I want to thank each of you for giving of your valuable time to this workshop.

GENERAL PHILOSOPHY REGARDING THE NEED FOR  
FITNESS TO DIVE STANDARDS

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Dr. Paul Linaweaver asked me to discuss philosophically the fitness to dive standards, the examination of divers, and the qualifications of the examiners. The fitness-to-dive question is a most difficult occupational health problem and properly should encompass preplacement examinations, medical surveillance examinations, termination examinations, and, for research programs, delayed effects examinations or surveys. The efforts to make diving work safe from acute and late health hazards is the objective of the whole world of experimental diving research. The occupational health aspects are a measure of accomplishments to make diving safe.

The purpose of examinations is to help prevent workplace injury and to choose the most able divers by setting preplacement standards. Also, I see our workshop is tasked to develop medical surveillance examinations which will determine maintenance of fitness standards with aging and will detect workplace injury.

The grizzled veterans at this workshop have wrestled with diving for most of their careers. They have experimented at great depth, ached during compression, experienced performance decrement, chilled rectal thermistors, drawn painful breaths, feared drowning; and then pale and limping, readied for the next dive.

No workplace so alters normal physiology as does diving. We should reflect that all our diving procedures are near man's predicted physiological limit; the oxygen levels just below convulsion or damaged lung, the inert gases at tolerable narcosis, the cold protection suitable only for the predicted dive duration, and the decompression procedures only usually safe. And when the work is deeper than 1200 feet or so, we hope the divers really recover. What other industrial workplace may cause a ten percent loss of body weight and crease the fingernails with a deep indentation? At about the time the groove wears off the end of the nails, the recollection of the dive begins to mellow.

Standards should match the anticipated work. The need to sustain severe exertion would be exemplified by the recreational diver who surfaces well astern of the dive boat in open water. The activity perhaps most likely to cause back injury would be handling doubles on a rolling and pitching dive platform. Personality problems are unveiled in the diver by accidentally securing his breathing gas. Entrapment would serve, as well. Engineering support determines the difficulty of most working dives.

The hardest civilian diving I have watched was at the Norfolk Naval Station in less than 45 feet of ice-covered water, handling the giant-hydraulically powered brushes to clean fouling from Navy ship bottoms. The warmest, least exertion, no-narcosis, no-hyperoxia, no-hypercarbia, no-

missed-meals, no-danger dive suitable for anyone's grandmother is a saturation dive to a moderate depth in a well-engineered system. The need for perfect health or great strength can be engineered out of the working dive. Or, consider the opposite question: How often has imperfect health been the cause of dive injury or death?

There are two general approaches to industry standards and perhaps the concept can be applied to the human machine. There is a design standard that describes the details of material and mechanics. For the human that might include age, height, weight, percentage body fat, blood pressure, forced expiratory volume, chest x-ray, blood count, and similar laboratory measurements. Perhaps a history of never having smoked is a design standard.

Another approach is the performance standard. The performance test should match the job. A suggested list for divers' preplacement performance standards might include:

1. Demonstrate a ventilatory response to increased inhaled carbon dioxide concentrations.
2. Sustain for five-minutes a work rate equivalent to 3 liters of oxygen consumption on an underwater pedal ergometer.
3. Climb a 10-foot, vertical, swaying ladder in complete MK-5 heavy gear.
4. Swim on the surface in wetsuit and scuba doubles one-half mile at one-half knot.
5. Swim a similar distance and speed underwater in double tanks and full wetsuit.
6. Recall 8-number series or mentally multiply large numbers in the dark with hands and feet immersed in icewater.

More adventurous but always employment-matched performance tests could be developed. The U.S. Navy uses performance tests. They put everybody in a chamber and take them to 112 feet. The chamber gets hot and they have to learn to clear their ears and if they don't do this, they don't go to diving school. Then they're decompressed and the chamber fogs and it's noisy and cold and then they put on a mask and breath oxygen for 30 minutes at 60 feet. It's a good psychological test that has some bearing probably on oxygen tolerance. But that's a performance test.

Periodic examinations for divers' working lives are often presented as repeats of the preplacement examination, rather than medical surveillance examinations. Once trained and once employed, the periodic examinations should be medical surveillance and should detect injury caused by diving. The laboratory studies that we do during that period should be pruned as much as possible by looking at specificity and sensitivity or at least the economics of this situation, so we're not getting studies that have no bearing on diving injury.

In the Navy, diving examinations for candidate divers or periodic examinations are not done by Diving Medical Officers. Instead, requirements are detailed and all examinations are reviewed at the Naval Medical Command. If physicians are provided with a standard format and detailed standards, replacement diving examinations are not difficult. Periodic examinations of the aging and deteriorating diver are causes of endless problems and seem insoluble because no one wants to end the career of a productive diver.

I think also that we must use some caution at this workshop in thinking about the list of performance standards or design standards that I've mentioned. Divers are not paid to blow bubbles. They're paid for their other skills and there's always a question of whether it's easier to take a skilled welder and make a diver out of him. The other aspect is, that with proper engineering support, a diver need have none of the physical performance qualities that I have discussed. Anyone's grandmother would be safe in a good saturation system. Worldwide, mostly untrained, physically deconditioned tourists scuba on shallow reefs without incident.

#### DISCUSSION FOLLOWING PRESENTATION BY DR. SPAUR

LINAWEAVER: You've mentioned a fair amount on the occupational aspect. Do you have any feelings about the sport diver who's not going to be doing any work? Any ideas about whether they should be required to have a physical examination?

SPAUR: I'd have to answer that no.

LINAWEAVER: Just as part of your philosophy.

SPAUR: Sure, if I were responsible for them I'd want to know what they were like.

LINAWEAVER: I think that was a well-chosen phrase. "If you were responsible for them," you would want them to have an examination. That was a well-chosen phrase because I think particularly with sport divers, it's fairly germane. With commercial divers and with military divers, with the hyperbaric technician, obviously somebody is ultimately responsible because they're either directing programs or directing activities. But the sport diver is a little different, and I think the key lies in that choice of the word responsibility, in particular whose?

## WHY RESTRICT PEOPLE WHO WANT TO DIVE?

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Most articles (1-3) or chapters (3-6) currently written in diving medicine texts present to the readers what might be called "conventional wisdom" concerning physical standards for diving. Although in many of these there are some areas with which one might disagree, I would generally put those areas into the category of minor disagreements rather than major controversy. On the other hand, one of the responsibilities of this workshop should be to carefully reassess "conventional wisdom" in light of available data. To this end, I would like to construct a "straw man" and attack almost all of the precepts we currently consider inviolate. I am doing this only as a method to provoke thought and discussion, and these arguments are not under any circumstances meant to be recommendations for divers.

With all that in mind, let me begin by saying that even at my most iconoclastic, I believe that military and professional divers must for many reasons be held to a different set of physical standards. Although certain stresses of the pressure environment are the same regardless of location or profession, other aspects of underwater activity are clearly performed under different circumstances and therefore demand different physical standards. Military diving, for example, could be performed under combat conditions or under rescue circumstances requiring extremely urgent action. Additionally, the military diver, unlike the commercial diver, cannot quit if he doesn't like his job assignment nor can he simply decide not to dive if he doesn't feel up to it, as the recreational diver can. The commercial diver, like the military diver, can also find himself working under extremely difficult and arduous circumstances, but there is an additional factor in commercial diving which demands extremely strict physical standards and that is the litigious nature of our society. Due to the ever increasing financial burden of work-related litigation, most commercial diving companies follow the philosophy of "zero defect." Whether this is justified from a medical point of view is questionable, yet from a practical point of view the adoption of such a philosophy is inescapable. As a result, the physician examining potential commercial diving candidates must be aware of these realities. Finally, commercial and military divers have a "job" that must be done. This "job" may be for military or economic reasons; however, delay due to a diver's physical limitations may not be tolerable.

With these differences in mind, the following remarks pertain to divers in general and their risk in the water, without considering the additional requirements that military or commercial diving may have.

Before I go on to deal with more specific physical disabilities or limitations, I would like to make some general comments more of a philosophical nature about disabilities to set the tone of subsequent remarks.

Dr. Linaweaver, on his chapter in the "Physician's Guide to Diving Medicine," (6) comments "... any condition present that could hurt the diver or result in injury to his buddy should be considered disqualifying" is in summary form the current opinion of many diving physicians. It is this philosophy that this workshop could re-examine. Any activity entails some degree of risk, and the limitation as quoted may be too restrictive. Some physical disabilities pose more risk than others. Many physical disabilities, although they theoretically could increase risk to a buddy, are within the "risk envelope" that the diver is willing to take. Thus, one philosophical point I feel the symposium should examine is "How much additional risk can a diver accept on his own?" In normal day-to-day practice, physicians have little right to tell a person what he can or can't do based on their opinions of what is best. Certainly no surgeon can operate on a patient because that surgeon feels it is in the patient's "best interest." Yet diving is different because there is a buddy involved and that buddy might require assistance or be drawn into a multicasualty accident because of his companion's "condition." Every year there are multicasualty accidents (7), and if the precipitating factor could be identified by a prediving physical examination several lives could be saved. The diving physician has a responsibility not only to the person he examines but to that person's buddy as well.

On the other hand, it can be argued that as long as the buddy is made aware of his partner's condition, he can either choose or not choose to accept the additional risk or, alternatively, the buddy system can be enlarged to include a third person. The latter is the recommendation of the Handicapped Scuba Association and, to the best of my knowledge, this group of divers does not have an unacceptable accident rate nor even an increased accident rate. Indeed, accident statistics overall do not support the notion that lesser physical standards are the prime cause of accidents (7-10). Therefore, the workshop should address as an additional philosophical point that our opinions about increased risk are indeed just "opinions." There are very few data that really suggest that morbidity and mortality in scuba incidents are related to known pre-existent conditions. Somewhere in the report of this workshop there should be a very large disclaimer that the recommendations being made are based only on extrapolations from our understanding of the physiology involved, and that the data do not actually support prohibiting diving with many pre-existent medical conditions. [One exception may be coronary artery disease, for there seems to be a number of deaths each year due to myocardial infarctions in the water (7)]. Once again, though, we must ask what degree of risk an individual should be allowed to take upon himself. One might argue that the data on accidents are skewed because current physical standards proscribe diving for people with significant disabilities. Personally, I am sure that this is not the case, as it seems that on almost every recreational dive trip I've been on there have been individuals diving who by "conventional wisdom" should not. From a historical point of view, perhaps the medical community's strong opinions concerning physical standards and abilities stems from the military and commercial background from which most of us come. In many respects, most of us are, as Dr. Glen Egstrom has sometimes put it, "dinosaurs." We originally learned to dive when equipment was more difficult to use, when training was not as good and when diving really did require a "tougher" individual.

Additionally, with the military background most of us have, any other divers, to a large extent, have been looked down upon as poorly trained and somehow not really "fit" enough to dive.

With the above comments in mind, rather than dealing with specific disease processes, it might be easier to examine the problems that are thought to occur because of underlying medical conditions. Once again, "conventional wisdom" may not be supported by the available data.

#### INCREASED SUSCEPTIBILITY TO DECOMPRESSION SICKNESS (DCS)

A wide variety of conditions have been suggested to increase the likelihood of DCS; however, analysis of almost all diving statistics does not support the contention that large numbers of the cases are found in the overweight, older, female, or diabetic population (8-11). The only analysis currently available of overall DCS incidence in the recreational dive community suggests that the incidence of DCS is actually lower compared to presumably more fit U.S. Navy divers (12). Analysis of series of DCS suggests that the main causes of DCS are attributable to not following standard decompression tables (including repetitive diving) or not monitoring depth or time of dive (11, 13). Conversations with other diving physicians who see reasonable numbers of accidents also suggest that they do not see a large number of cases of DCS that can be related to the "classic" predisposing factors but rather to the violation of basic diving rules (13-15). Even if such conditions actually did predispose to DCS, such divers could be counseled to dive according to more conservative profiles. Indeed, more conservative tables are now being advocated by a number of individuals and organizations. These changes are even being recommended without significant evidence that they are needed. Finally, conditions that were thought to be easily confused with DCS (previous nerve injury, chronic orthopedic problems, etc.) have not, in clinical practice, been difficult to differentiate from DCS and/or treat.

FARMER: Tom, may I interrupt?

NEUMAN: Yes.

FARMER: Do the data support the notion that the converse is not true?

NEUMAN: I think that the data suggest, and if you go to hyperbaric chamber operators and people who take care of decompression sickness, that the overwhelming number of decompression sickness accidents are caused by people not following the tables, whatever those tables are going to be. By not diving within the rules as they are outlined.

FARMER: But do you have enough data to say that obesity and lack of physical fitness does not contribute to decompression sickness?

NEUMAN: I don't think you'll ever have enough data for that. But on the other hand, if you talk to people who see a large number of accidents, the overwhelming majority of them are caused by not following the rules. It's not as if no fat people dive. It's not as if no women dive. It's not as if diabetics, epileptics, asthmatics do not dive. They're there, they're in the

population and they do not seem to be over represented as far as decompression sickness is concerned. But that's going to be a recurrent problem in everything we deal with.

Also, even if such conditions did predispose to decompression sickness, you could advise those people to dive to more conservative rules and in fact right now a large number of people are advocating more conservative diving tables without there being any real data that they're necessary. The other argument is that people who are predisposed to decompression sickness for one reason or another who have some sort of underlying disability might be more difficult to sort out whether they've got decompression sickness or not. At least in my own situation, we've had no difficulty telling whether somebody is bent or not bent and what to do about it or what not to do about it.

In summary then, a strong argument can be made that the "predisposing factors" for DCS have not really caused significant numbers of cases of DCS and, therefore, this reason should not be used to disqualify large numbers of scuba divers.

#### UNCONSCIOUSNESS UNDERWATER

Clearly, losing consciousness under water represents a grave risk to a sport diver, yet it can be argued that, in the past, physicians have been far too restrictive about divers with such medical conditions. This is not meant to imply that all individuals who have illnesses that can result in a decreased level of consciousness should be permitted to dive; however, there may be a population that, due to increased understanding, maturity, etc., who, if carefully selected, might be able to dive safely even with such conditions. Diabetes mellitus (either Type I or II) can cause unconsciousness rather suddenly via hypoglycemic reactions (ketoacidosis and non-ketotic hyperosmolar coma both develop far too slowly to be a true risk to a diver), yet such reactions can be prevented in Type I diabetics merely by reducing normal insulin dosage prior to activity and/or eating a reasonable meal approximately one hour beforehand. Such a therapeutic alteration will cause a modest hyperglycemia, making the risk of sudden unconsciousness almost nil. Similarly, the Type II diabetic can either reduce the normal dose of oral agent or omit it entirely. Under such circumstances, some diabetics should be able to dive safely. The argument that the diabetic is more susceptible to DCS is not statistically tenable, but even if it were, the arguments in the previous section would pertain.

A well-controlled seizure disorder is a similar example. Admittedly, a seizure underwater could be fatal, however, if the seizure disorder is *totally controlled* and the diver and buddy (buddies) understand and accept the risks to which they are exposing themselves, it could be argued that such a person be permitted to dive.

On the other hand, any individual who suffers from unexplained lapses of consciousness or who might *reasonably* be felt likely to lose consciousness underwater should be advised not to dive. Most states have laws prohibiting people who have sudden lapses of consciousness from operating motor vehicles. Uncontrolled seizure disorders, as well as other conditions, generally result in the loss of a driver's license. If a person is permitted to operate a



motor vehicle (which certainly is a greater risk to the public than a diver), what rationale can a physician use to prevent such a person from diving if he and his buddy are willing to assume the risk?

Without being facetious, perhaps the most important neurologic conditions to be diagnosed prior to diving are the behavioral ones. Although it is rare (almost to the point of being unheard of) for diabetes or epilepsy to cause a scuba fatality, it is quite common for fatal air embolisms to be precipitated by the diver running out of air while underwater. I'm not sure how to screen for such behavior, but perhaps we would be doing diving safety a great service if we would suggest that people do not dive if they answer "yes" to the question: "While driving, have you ever unexpectedly run out of gas on the highway?"

#### INCREASED SUSCEPTIBILITY TO PULMONARY OVERINFLATION SYNDROMES

In this area, I am at my least iconoclastic. If one discounts the people who die because they run out of air or get lost in a cave (no amount of physical screening can protect a diver from his own stupidity), air embolism is the second leading cause of death in divers. [Depending on how the statistics are interpreted, one might argue that it is the leading cause of death (7)]. Unfortunately, this is where the reliable and useful statistics end. According to my own experience and that of several others who treat or have treated reasonable numbers of diving accidents, the vast majority of individuals who get air embolisms clearly "deserve" them (13-15). In other words, in our personal experience, almost all of these accidents are associated with panic or out-of-air situations with uncontrolled ascents. Yet there remain a small percentage of pulmonary overinflation syndromes that occur in divers who, to the best of our ability to detect, neither ascend too quickly nor hold their breath. The disquieting point is that, invariably, those individuals have no prior history of lung disease and therefore they would be impossible to screen out by any reasonable pre-dive physical examination.

On the other hand, data from the DAN reports suggest that approximately 50% of air embolisms occur without identifiable cause (8). Does this mean that almost one-half of the individuals getting AGE have underlying conditions that caused the accident, or is this a reporting error due to the inability to collect data? In any event, I believe that as a result of these disparate data, one must be extremely cautious about allowing an individual to dive who has lung disease that is associated with air trapping. Asthma, however, must be treated as a separate case. First of all, it is an extremely common affliction affecting approximately 4% of the population (16). As a result, if asthma were the cause of unexplained pulmonary overinflation syndromes, I would expect that experienced diving physicians would have seen such syndromes with some frequency occurring in asthmatics on normal dives without breath-holding. Yet as I mentioned before, I have polled Drs. K. Kizer, A. Pilmanis, and A. Newman, and none of us has seen an "undeserved" arterial gas embolism in an asthmatic. Once again, I believe that the argument that accidents are not seen in asthmatics because asthmatics are not allowed to dive is untenable. Although there may be small geographic areas where doctors are knowledgeable about diving, in general,

most divers have no physical screening at all. Additionally, I suspect all of us have been on dive trips where there were asthmatics along.

If, then, asthmatics dive and, as it appears, those asthmatics are not involved in significant numbers of otherwise unexplained accidents, is it fair to recommend that all asthmatics not be allowed to dive? This is certainly a difficult question compounded by the fact that the spectrum of the asthmatic diathesis is very large. I believe it is fair to say, however, that the currently available data do not support prohibiting diving in all asthmatics. As far as other lung disease is concerned, the numbers of such individuals who dive are too small to allow us to look at accident data in a meaningful fashion. As a result, we are forced to rely only on extrapolation from our physiologic understanding in these cases, and I would recommend that any pulmonary disorder that results in an abnormal ventilation scan or has physiology that would be reasonably expected to produce such an abnormality be considered a contraindication to diving (i.e., cystic, bullous, chronic obstructive disease, etc.). Pure restrictive disease is probably not a contraindication to diving from a mechanical point of view; however, such people usually have combined obstructive and/or cystic lesions or gas exchange (exercise) limitations and these latter conditions would be a contraindication to diving.

A history of spontaneous pneumothorax should also be considered a contraindication. Not only because it may recur (17), but perhaps more importantly because the cause of most spontaneous pneumothoraces are blebs and other poorly communicating spaces. Traumatic pneumothoraces should be evaluated on a case-by-case basis. If the underlying lung injury was minor, I do not understand the rationale prohibiting diving. In the case of a thoracotomy, one must also examine the underlying pulmonary pathology that was the indication for the thoracotomy.

#### INABILITY TO EQUALIZE PRESSURE (Increased Risk of Pressure Equalization)

Most diving physicians recommend that patients with severe allergic rhinitis, nasal polyps, or difficulty equalizing be advised not to dive. But for the individual who is not in circumstances where he must dive if asked, there appears to be little or no risk to at least attempting to dive. If the diver is unsuccessful at descending and as long as the individual does not try to equalize excessively, the major risk has been only to the time and money to arrive at the dive site.

Clearly, such a person should be advised of the risks of over strenuous clearing maneuvers (round window rupture, etc.), but once again, it can be argued that an individual may choose to accept those risks.

Similarly, a person with previous middle or inner ear surgery should be permitted to decide whether or not the increased risk of permanent hearing loss is one he is willing to accept.

Once again, diving accident statistics do not support the assertion that predisposing factors of this sort are the cause of significant numbers of accidents.

Finally, although it is not technically a "physical standard," I would suggest this workshop address what is the apparent cause of most diving accidents. It is clear that the large majority of diving accidents are caused by poor judgment or inattention to the basic rules of diving safety, and as a result, perhaps we would be well advised to pay more than just lip service to the level of a candidate's maturity and responsibility.

#### REFERENCES

1. Becker GD, Parell GJ. Medical examination of the sport scuba diver. *Otolaryngol Head Neck Surg.* 91:246, 1983.
2. Neblett LM. Otolaryngology and sport scuba diving: Update and guidelines. *Ann Otology, Rhinology and Laryngology* 94(1):2, Supplement 115, 1985.
3. Medical Standards. In: Edmonds C, Lowry C, Pennefeather J. Eds. *Diving and Subaquatic Medicine (Second Edition)*. Diving Medicine Center: Mosman, New South Wales, Australia, 1981.
4. Kindwall EP. Medical examination of the diver. In: Strauss R. Ed. *Diving Medicine*. New York: Grune and Stratton, 1976.
5. Elliott DH, Davis JC. The causes of underwater accidents. In: Bennett P, Elliott D. Eds. *The Physiology and Medicine of Diving (Third Edition)*. San Pedro, CA. Best Publishing Co., 1982.
6. *Physician's Guide to Diving Medicine*. The Undersea Medical Society, Bethesda, MD, 1984.
7. McAniff JJ. United States underwater diving fatality statistics, 1970-1982. National Underwater Accident Data Center, University of Rhode Island, Providence, RI.
8. Dick AP, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. *Neurology* 35:667, 1985.
9. Walker D. Provisional report on diving-related fatalities in Australian waters, 1984. *SPUMS Journal* 15(3):17, 1985.
10. Walker D. New Zealand diving-related fatalities, 1981-82. *SPUMS Journal* 14(2):12, 1984.
11. Kizer K. Epidemiologic and clinical aspects of decompression sickness in Hawaii. Sixth Annual Conference on the Clinical Application of Hyperbaric Oxygen; Memorial Hospital Medical Center, Long Beach, CA, June 13-15, 1981.
12. Bassett B. Diving safety. Presented at *Diving Medicine in Depth*. Bonaire, Netherlands, Antilles, 1985.

13. Pilmanis A. Personal communication.
14. Kizer K. Personal communication.
15. Newman A. Personal communication.
16. Terr AI. Bronchial asthma. In: Baum GL. Ed. Textbook of Pulmonary Disease (Second Edition). Boston, Little, Brown & Co., 1974.
17. Clausen J. Pneumothorax. In: Bordow RA, Moser KM. Eds. Manual of Clinical Problems in Pulmonary Medicine (Second Edition). Boston, Little, Brown & Co., 1985.

#### DISCUSSION FOLLOWING PRESENTATION BY DR. NEUMAN

LINAWEAVER: There are people who enter the diving situation or want to continue in the diving situation in which a medical condition exists and the physician may be asked whether he can dive. The training agencies themselves really have no control over the operational environment in which an individual finds himself after training. Have we, in the past, been conservative and somewhat restrictive by saying, "I'm going to see this individual, I'm going to do an examination and say he is fit to dive knowing that he may go to remote areas for sport diving where there is no medical help if he gets into trouble. Is he going to do cave diving? Is he going to set a world record off the Caymen wall? Are we overly conservative as a result of that?

Also, do we make a distinction between somebody entering into the training situation versus the experienced diver who, 10 years after he's become a diver and dives regularly, is found to be a Type I diabetic?

Does anybody have any comments on the two previous presentations, the questions that I've raised, or maybe you have questions yourselves?

BOVE: One brief comment and then a question or two. I think we have been driven in the past by concern for propagating increased incidence of accidents and introducing legislation. That kind of thing has tended to threaten the sport diving community in the past, and I think one of the underlying modalities that hangs in the background all the time is what happens if a large number of a certain population of individuals gets cleared for diving and then suddenly the accident rate goes up and now we have legislation. I guess that's something that we at least ought to keep in the back of our minds as we go through this next couple of days, because we don't have a major accident problem right now. It's not clear, whether that is because we're conservative or because there aren't any accidents. But that's a consideration that has to be brought up.

I wanted to ask a question, working back up the list. It was said that it doesn't harm somebody with an ear problem to come and spend some money and not be able to dive. I want to ask Joe Farmer, because my experience of

those people is they always get squeezed and they always have an ear injury that turns them off for the rest of the week. Joe, is that going to generate long-term hearing loss problems in these people when they're 45, 50, 60 years old? Is that like recurrent industrial injuries?

FARMER: We don't know the long-term implications of repeated middle ear barotrauma. There's been evidence to indicate that late dysfunction may result. The problem with a lot of the ear, nose and throat considerations is that this area has been long relegated to secondary considerations in any discussion like this or in any diving-related medical consideration. That's understandable, because you have pulmonary problems that are more immediately life threatening. But, we just don't know and I'll have to be the devil's advocate to Tom as well and say that the fact that we don't have data to say that it's wrong does not mean it is safe. To emphasize the importance of ear and sinus problems in diving, I will show you a couple of slides. Temporal bone specimens are very hard to get in a fresh state where you can really make some judgment about what was going on at the time of death. Canadians who reported in the UBR a year ago have collected two temporal bone specimens, one of which clearly shows a labyrinthine window rupture in an experienced diver who drowned while diving in shallow water. He apparently had vertigo, vomited into the regulator, and drowned. Also, there have been a few case reports of pneumocephalus and facial subcutaneous emphysema supposedly related to paranasal barotrauma. As you all know, just because you can get down and clear your sinuses doesn't mean you're going to be able to come back up again. The ethmoid plate is one of the thinnest bony partitions in the body. I'm surprised it hasn't happened more often. But to answer your specific question, long term otological effects may occur.

BOVE: I'm specifically addressing the fact that, if you go on a diving trip, I would say between seven and ten percent of the people on that dive trip have clinical evidence of ear squeeze some time during the week.

LINAWEAVER: I'd say it's a lot higher than that.

BOVE: Well, it may be higher, but let's use that as a number. I'm just saying, my own experiences may be 10 percent or so if you look at the symptomatic ones. If that kind of thing goes on over a multiple of years, it seems to me that its model as an industrial hearing loss is not far off the mark. Somebody in that category is going to have to need a hearing aid or have some problems with hearing when they're 60 years old.

FARMER: There is evidence that recurrent bouts of middle ear inflammatory disease will increase the chance of nerve deafness as time passes.

YOUNGBLOOD: I would like to try to get us back into focus: the thing that we should be addressing here--as mentioned in the introductory remarks--is the entire hyperbaric environment. We keep getting off into esoteric aspects of military and commercial diving problems when we have never clearly identified the specific medical conditions that are hazardous in a hyperbaric environment. We should specifically identify those conditions that a medical examination might detect and which, if present, should preclude hyperbaric exposure. That is a much shorter list than the book we're talking about

writing. Each physician responsible for a particular recreational or occupational group can add specific contraindications that he finds are appropriate. It is unrealistic, however, to assume that a busy general practitioner asked to examine a prospective scuba diver will be able to take the time to draw diagrams of the inner ear or give lectures on several different aspects of diving physiology. It's just not going to happen.

LINAWEAVER: I think that's a good point. That's why I, after looking at the myriad of requirements from the sport diver all the way through to probably the hardest working group, the tunnel workers, came up with the concept of the generic approach to fitness as exposure to pressure and the aquatic environment. Has our tendency in the past been, by medical judgment, to influence the selection of people that should be handled operationally from a training aspect or from an equipment aspect? Have we been kind of the protector, the knight with shining armor on the white stallion, protecting the mass of people, when there is no medical basis to do so?

YOUNGBLOOD: I think we have been and I've been as guilty as anybody.

GREER: I was surprised, Tom, when you said that you didn't know anybody who'd been injured from air embolism who had asthma. I can think of three cases.

NEUMAN: Undeserved?

GREER: Undeserved. Three cases right now. One of them I treated, one of them Dr. Van Meter treated, and one of them was an invited participant at the workshop we had on the rehabilitation of paralyzed divers. All of them, one a military commercial diver, one a longstanding sport diver and a veterinarian, and one a scientific diver had a history of asthma. All of them had undeserved arterial embolism. Two of them were permanently paralyzed.

NEUMAN: I'm not aware of them. I put the list down of the people that I have contacted and none of them had seen it either.

MEBANE: I had one last summer.

NEUMAN: I've seen undeserved air embolisms and undeserved pulmonary overinflation syndromes. In the ones I've seen, they just haven't had underlying lung disease.

The next question that we have to ask is "Do those undeserved air embolisms show up in numbers greater than would be expected as far as all undeserved air embolisms?" That depends on whether you look at the DAN data, which say 50 percent of air embolisms are undeserved, or whether you look at your experience. In my experience, it is a much smaller percentage of people who have undeserved air embolisms, maybe 5 or 10 percent.

FARMER: Let's call it something besides undeserved. Call it idiopathic. I think that is a good term.

ARMER: I have a number of problems with what we've been saying. I think one issue is "Is there a way to predict from the physical examination whether certain people are, in fact (or whether they can be proven to be) at a greater risk in terms of accidents?" But, the other problem is that when someone comes to you for a diving medical clearance, you sign your name to it. As the physician related to a scientific diving program, the question I am asked is "From your examination, do the following people have a minimum risk of diving accidents due to their physical status?" Because there are matters of liability involved, the institution has to make a decision as to whether it's going to take the risk. As far as I can tell, we have a very poor correlation between anything that you or I can determine in examining these people and the actual accidents. There are other things that seem to correlate much better. For example, I think the scientific diving community has an excellent record compared to certain types of commercial divers and certain types of sports divers, although the diving may be quite similar. Why is that? Well, they are a special group of divers. They have diving safety people pushing them all the time in terms of training. They are highly educated and use diving as a tool for research. They have to get their dives approved by diving officers and so on. I find little, if any, correlation between anything I do for them medically, other than teaching, with whether or not they get into trouble in the water. In fact, in our own program, a typical accident is the one that happened last week in the China Sea when two of our scientists were diving at 30 feet and a fisherman came by and dropped TNT in the water to kill fish, and instead almost killed our divers. That has very little to do with our physical status.

The diving physical is a problem. I feel very frustrated because the divers say "Why are you making me do all these things, why do we have to spend all this money"

On the other hand, it seems to me that it is still useful, in an initial diving exam, (done before someone goes into the water to be trained) even though we've not yet seen trapped air in our chest x-rays, still it should show in occasional exploratory chest x-rays, to do expiratory views even if it shows only once in 10,000, in a liability situation, it's worth doing for that one. What the worth of repetitive (annual) chest x-rays is, I can't tell.

NEUMAN: If it is one in 10,000, just the number that was picked, we have to ask the question whether it's worth doing at all. And that brings us to another question and a medical-legal one. On the one hand, the diving company may say, "Yes, it's worth it for me to pay for 10,000 x-rays to detect who may get an air embolism because the amount of money that that's going to cost me is so great that it's worth it." But, that's not a medical argument. That's an economic argument that doesn't pertain to the guy who has that x-ray and now comes to you and says "Is it safe for me to dive?" And you have to shrug your shoulders and say, "I don't know."

GREER: I think there was a contradiction there. You said that it's not a medical argument for the diving company to say it's worth it. But, you say it is a medical argument to say that if it's only one in 10,000, it isn't worth it. Those are both economic considerations.

ALEXANDER: In the Los Angeles area the diving instructors and the divers themselves are often much more cognizant of problems than the physicians. Usually they are screened twice. Once from the physical aspect, if they have problems and they can't hack it, and again by their diving instructor. If there is good physiologic evidence that things may predispose one to accidents, they are told they shouldn't dive.

MEBANE: I have a couple of comments about sport diving. The recreational diving industry has been dissatisfied with physical exams for years largely because 90 percent are done by physicians without the slightest concept of what happens in diving whose advice is "It's okay to dive just so you stay shallow." The certifying agencies had a conference in January and their recommendations eliminated the physical examination for most divers. Divers entering into training are to complete a medical history form prior to the SCUBA water skills portion of the course. It is strongly recommended that individuals with unusual medical histories receive a physical examination and certification by a physician before engaging in SCUBA water skills. That means that the instructor will have the responsibility of screening prospective candidates and refer them to a physician when a physical exam appears necessary. There is currently no requirement for a physical examination before beginning any of the SCUBA courses. I think it is important to understand in the discussion we may not be seeing many sports divers for physical exams unless they're screened by knowledgeable instructors.



## CARDIOVASCULAR DISEASES AND DIVING

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### INTRODUCTION

As the diving and undersea environment has become available to the general population because of equipment development, standard medical requirements for diving which were initially developed for commercial, military, and scientific divers have been challenged. Early diving involved surface-supplied heavy equipment in deep operations usually related to ship salvage or underwater construction. Medical standards developed for such diving within the United States Navy and other navies were stringent and designed to eliminate completely any possible medical contingencies during a diving operation (1). With the development of scuba equipment and the availability of this equipment to the public at large, as well as the less rigorous requirements for diver training in sport diving as compared to commercial and military diving, the population of sport divers increased rapidly until now it is estimated that over two million individuals in the United States have been trained and at some time have been exposed to sport diving (2).

With minimal medical requirements and the likelihood in two million or more divers of finding individuals with significant handicaps and chronic medical illnesses, the issue of medical qualifications for sport diving needs to be addressed. Clearly, requirements are different from those involving commercial and military divers. In order to evaluate cardiovascular disease and its interactions with diving, it is necessary to elucidate those environmental factors that might affect various aspects of cardiac disease, and then to relate the diseases of the heart and circulation to these environmental stresses in a way that will provide a rational basis for understanding interaction of the diving environment with various cardiovascular disorders.

### ENVIRONMENTAL STRESSES IN SPORT DIVING

Although sport diving is a relatively low-stress sport for the well-trained individual, several aspects of sport diving can produce cardiovascular stresses that will interact with chronic cardiac or circulatory conditions to produce adverse reactions. Such reactions in the cardiovascular system are unpredictable and result in disability or even sudden death. The physician involved in examination of sport divers must assure the diver and himself that no unexpected cardiovascular contingencies will arise from exposure to the diving environment.

Table 1 describes environmental factors in sport diving that can interact with cardiovascular diseases. The first component of this environment that is important in understanding cardiac interactions is exercise. Although sport diving generally does not require significant amounts of exercise, certain

TABLE 1

Environmental and Cardiac Factors  
Requiring Consideration in Diving with Heart Disease

<u>Environmental Factors</u>	<u>Cardiac Factors</u>
Exercise	Coronary
Anxiety	Valvular
Cold	Congenital
Pressure	Hypertension
Gases	Heart Failure
Decompression	Pacemakers
Trauma	

situations such as long walks with full equipment and wetsuit, long swims against currents or winds, the need to occasionally tow a disabled partner, lifting of tanks and other heavy equipment all require physical activity that raises cardiac output, heart rate, oxygen consumption, and blood pressure above basal levels. The need for increased physical activity is immediately sensed by the cardiovascular system, which responds by providing increased blood flow to skeletal muscle to fuel the muscles for increased work. Emotional stress or anxiety in diving is common, especially in the novice diver. Many divers who might be comfortable in warm, clear water under no physical stress can become panic stricken and irrational with severe anxiety reactions which result in hypertension, tachycardia, increase blood catecholamine, and excess stimulation of the heart and circulation. Reactions of this type directly affect the cardiovascular system and must be taken into consideration when examining and qualifying an individual for sport scuba diving.

The direct effects of pressure on the body are generally of small concern with regard to the cardiovascular system. Since pressure is distributed equally throughout the tissue and the blood there are no additional pressure differentials against which the heart must work in a diving environment. Several factors, however, can contribute to excess load on the cardiovascular system. One of these is the loss of gravitational pooling of blood in the lower extremities when immersed. This effect causes a shift of blood from the legs into the central circulation (3) and, in individuals with compromised cardiac function, may precipitate congestive heart failure or pulmonary edema. Fluid shifts of this kind are well known to produce diuresis because of distension of the central circulation, and must be considered when evaluating patients with cardiac disease. Another effect of pressure is the direct compression effects on implanted cardiac pacemakers. Although few people with pacemakers are healthy enough to undertake diving, in some instances younger individuals with only conduction system abnormalities of the heart and who are otherwise in excellent health with implanted pacemakers, do undertake sport scuba diving. Of concern here is the direct effect of pressure on the pacemaker case, and one must consider this effect when providing advice or certification for a sport scuba diver who has an implanted pacemaker. (See section on pacemakers.)

Thermal stress, especially cold, is a common problem in sport diving. In temperate or northern waters, significant heat loss occurs if a diver is exposed without thermal protection for even brief periods of time. Water temperatures must be in the mid 80°F range to achieve true thermal balance. Temperatures below this level will cause heat loss and hypothermia. Cold stress produces vasoconstriction which elevates blood pressure, induces tachycardia, and increased cardiac output (4). There are suggestions in the cardiovascular literature that cold stress can induce coronary artery spasm and angina (5). This mechanism is postulated to be one of the means by which angina is induced by exposure to cold air. Divers with coronary artery disease for example can have angina aggravated or ischemia produced by exposure to cold air, and cold water exposure is even more of a stimulus for this type of ischemic response.

Trauma in sport diving is not generally considered to be an important issue, however, when one examines sport diving experience, it is common for minor bruises and injuries to occur in the process of any sport diving operation. These scratches, bruises, and other minor injuries are never considered to be important by a sport diver; however, they can be important in an individual treated with anticoagulants, for example, for deep vein thrombophlebitis or for a prosthetic cardiac valve. Of course, more severe trauma would be complicated by anticoagulants and serious hemorrhage can result, including subdural hematoma in individuals sustaining what otherwise might be considered minor trauma.

Decompression problems in diving are generally uncommon in the sport diving community. However, it is well known that in many sport diving exposures silent venous bubbles are formed which flow into the pulmonary circulation and are dissipated in the lung (6). In certain cardiac disorders where a transient right-to-left shunt may occur (patent foramen ovale or atrial septal defect), these bubbles which otherwise would be totally asymptomatic and benign can transit the heart to become emboli in the arterial circulation. Such embolization, because of a right-to-left communication, would result in cerebral or cardiac manifestations which produce serious morbidity or mortality.

Finally, one must consider exposure to toxic gases during diving including carbon dioxide and carbon monoxide. In general, small amounts of carbon monoxide that might be inhaled in contaminated breathing air would be of little consequence to a healthy individual unless prolonged exposures occurred. On the other hand, in individuals with coronary artery disease or congestive heart failure, the compromise of oxygen transport by hemoglobin bound with carbon monoxide would precipitate myocardial ischemia or aggravated congestive heart failure.

These seven environmental factors must be considered when examining and qualifying a sport diver. Specific interactions with each of these environmental conditions and certain aspects of cardiac disease can be readily deduced, and are well known in other aspects of sports and exercise besides the diving environment. By considering these seven environmental factors, one can

predict whether problems would arise in specific cardiac diseases even though no previous well-established experience is available.

#### INTERACTION OF CARDIAC DISEASE WITH THE DIVING ENVIRONMENT

Although it is impossible to describe precisely how each cardiac disorder would interact with sport diving, the approach to dealing with individuals with cardiac disease involves a thorough understanding of the diving environment, and the physiologic responses of the cardiovascular system to these various environmental stresses. With this understanding one can rationally predict how certain cardiac disorders will respond to sport scuba diving. At the same time diving candidates with mild cardiovascular disease can be instructed on how to avoid certain aspects of the diving environment that might interact adversely with their otherwise asymptomatic cardiac disorders.

#### Coronary Artery Disease

This is the most common serious disease in our population at the present time. It is estimated that over 250,000 people a year die from coronary disease and half of this population dies suddenly without access to medical care (7). The occult nature of coronary disease is best described by Figure 1, which demonstrates the relationship between blood flow in a coronary artery and the luminal diameter. If one plots the blood flow through an artery under normal blood pressure, as the artery is narrowed by an atherosclerotic plaque, one finds no changes in resting blood flow until at least 60% of the lumen has been occluded by atherosclerotic plaque. During high flow states, for example induced by exercise, flow can be compromised when the lumen is 40-50% narrowed, however, in every case significant progression of this disease can occur prior to any symptoms being evident at rest or even with mild exercise. On the other hand, if an individual has an atherosclerotic plaque which results in 60-70% narrowing of the coronary artery and does not exercise adequately to produce cardiac ischemia or symptoms of coronary disease, then this individual is at high risk for sudden cardiac events during the stress of exercise, including the exercise induced by sport diving. It is, therefore, imperative for physicians involved in the assessment of sport divers to rule out coronary artery disease prior to approving such an individual for a diver training program. The increased cardiac activity which results in increased myocardial oxygen demand can result from the exercise involved in diving, from anxiety which raises blood pressure and heart rate, and from cold stress producing increases in blood pressure (8). In the presence of significant coronary artery narrowing myocardial ischemia can result. In addition to a fixed stenosis limiting coronary blood flow, it is also quite possible that coronary spasm can occur under anxiety or cold stress and result dynamic narrowing of the coronary artery which can produce the sudden onset of ischemia with attendant arrhythmias and sudden death (9). Coronary spasm is generally found in patients with already existing coronary artery disease; it is rare for a subject totally free of coronary disease to be subjected to coronary spasm.

The most important considerations in dealing with the diver candidate suspected of having coronary disease is to first diagnose the presence of this condition and, second, to determine whether the disease is extensive enough to produce a risk of ischemia during the exposure to sport diving.

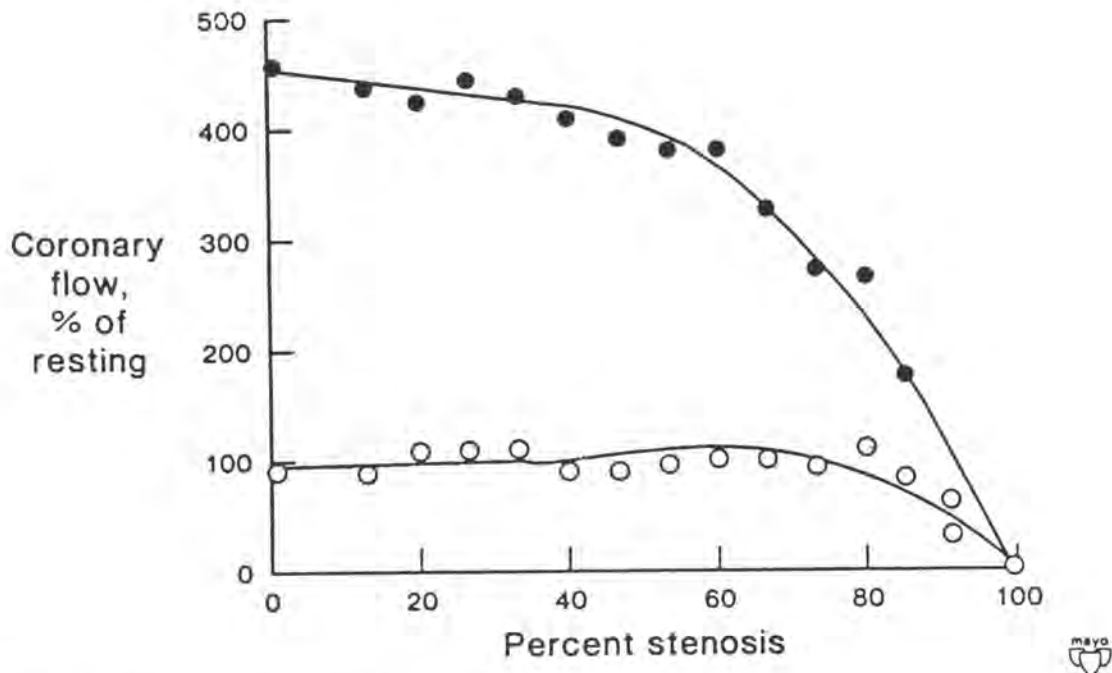


Fig. 1. Coronary blood flow changes in response to narrowing of a coronary artery. Significant narrowing can occur before resting flow is compromised. o resting blood flow; o exercise blood flow. Adapted from Gould KL, et al.,: *Am J Cardiol* 33:87, 1974.

Diagnosis of coronary artery disease can be made in several ways. Often subjects wishing to participate in sport diving have already diagnosed coronary disease manifest either by myocardial infarction or by coronary artery bypass surgery. In these instances exercise-based criteria must be established to ascertain the individual's capacity to exercise (see below). In these individuals the diagnosis is certain and it is necessary to determine their capacity. On the other hand, apparently healthy individuals with occult coronary disease present a more complex diagnostic problem. The incidence of coronary artery disease increases with age (Table 2) and with certain risk factors (Table 3). These include cigarette smoking, diabetes mellitus, a strong family history of coronary disease, hypertension, hyperlipidemia or hypercholesterolemia, and significant deconditioning (10). Although age is the most important factor used for predicting coronary artery disease, a risk factor profile can be established for each individual so that the physician can develop an approach to diagnosis in diving candidates of various ages based on these risk factors. For example, a 20-year old athlete who exercises, is in good physical condition, and has no significant past medical history is highly unlikely to have significant coronary disease that would interfere with the diving environment. At the other end of the spectrum, a 70-year old male, with mild diabetes, apparently healthy, but with hypertension and hypercholesterolemia, is highly likely to have coronary artery disease even if

asymptomatic at the time of the evaluation. Between these extremes lie an infinite number of probabilities for coronary disease and each person must be assessed in relation to risk factors and age so that occult coronary artery disease is detected. This author recommends that all individuals over the age of 40 who have any significant risk factors and who have not been previously tested, be approved for sport scuba diving only after successfully completing an exercise test with electrocardiogram and blood pressure monitoring. This test should be conducted to 13 METS (metabolic equivalents) and can be done using a stationary bicycle or a treadmill. The Bruce protocol is a standard protocol used in many laboratories for treadmill testing, and 13 METS would be equivalent to 12 minutes on this treadmill protocol (Fig. 2). A sport diving candidate should successfully complete the 13 MET exposure without evidence of ischemia, without angina or serious arrhythmias, and with a normal blood pressure response (160-170 systolic, 70-80 diastolic mmHg, see Fig. 3). These criteria can be applied to individuals who have sustained a myocardial infarction, who have normal cardiac function, and who are otherwise asymptomatic. A typical example is a 45-year old otherwise healthy male who sustained a small inferior myocardial infarction, has been demonstrated to have single vessel coronary disease by angiography, with no other serious disease and who can exercise successfully on a treadmill following the above-noted protocol. Such an individual who is free from other significant coronary disease and who does not demonstrate any ischemic changes with exercise is able to undertake sport diving safely. Similarly, an individual who developed cardiac symptoms, has one or two stenosed vessels bypassed surgically or dilated with a balloon catheter, Percutaneous transluminal coronary angioplasty (PTCA), and who demonstrated the treadmill response noted above, can be assumed to have adequate myocardial revascularization and can undertake sport diving without concerns for coronary disease being a limiting factor.

Patients who have sustained significant myocardial damage from coronary disease and who have evidence of left ventricular failure, should not be approved for diving since the fluid shift from the aqueous environment, vasoconstriction due to cold, exercise, and anxiety produce work loads added to the already compromised ventricle, which can precipitate acute left ventricular failure and pulmonary edema. Such individuals are not qualified for diving.

#### **Bypass Surgery and Angioplasty**

Patients who have undergone either coronary artery bypass graft (CABG) or PTCA must be considered in the same fashion as those with unoperated coronary disease. The degree of revascularization must be reviewed, the amount of damaged myocardium documented, and the subject's exercise tolerance documented as described above. A number of patients have returned successfully to sport diving after thoracic surgery. This experience indicates that the surgery itself is not a limiting factor; rather, the cardiac factors considered in anyone with coronary disease must control the decisions about diving. Post CABG or PTCA patients should undergo cardiac rehabilitation and develop good exercise tolerance to 13 METS without ischemia or serious arrhythmias.

TABLE 2

*Incidence of Coronary Disease by Age*

<u>Age</u>	<u>Relative Risk*</u>	
	<u>Male</u>	<u>Female</u>
35-44	0.18	0.09
45-54	0.79	0.47
55-64	2.19	2.24
65-74	6.29	5.92

\*Multiple of average age adjusted incidence: Males - 337/100,000  
 Females- 119/100,000

TABLE 3

*Coronary Risk Factors*

Smoking  
 Cholesterol  
 Hypertension  
 Diabetes  
 Poor Fitness  
 Family History  
 Stress

**Valvular Heart Disease**

Valvular heart disease presents several unique problems which are somewhat different from those that occur with coronary disease. Because the heart has significant functional reserve, small abnormalities of the cardiac valves can be tolerated exceedingly well and no significant evidence for cardiac decompensation can be found at rest or at exercise. Such is the case with mild mitral regurgitation that might be present in association with mitral valve prolapse. This small or trace amount of mitral regurgitation is inconsequential to the function of the heart (11), and would not compromise function during the stresses which might be imposed during diving. On the other hand, a large volume overload induced by severe mitral regurgitation or severe aortic valvular regurgitation stresses the heart beyond its normal function and induces a state of hypertrophy with some loss of functional reserve. If the regurgitation is moderate, the myocardium will hypertrophy

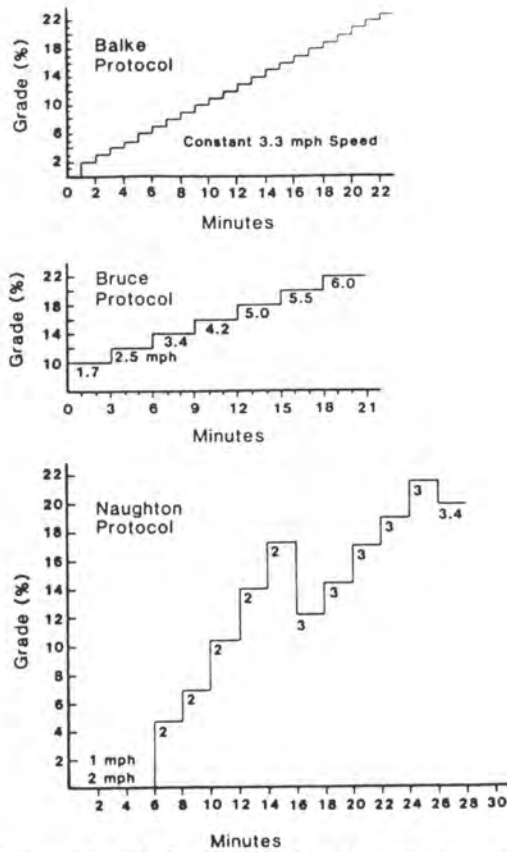


Fig. 2. Standard protocol for treadmill exercise tests. From Squires RW, Bove AA: Cardiovascular profiling. Clinics in Sports Medicine 3:11, 1984.

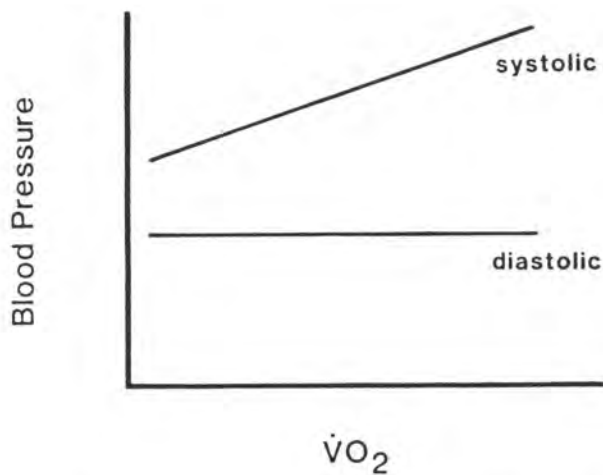


Fig. 3. Normal blood pressure response to increasing exercise intensity.  $\dot{V}O_2$  = oxygen consumption. From Squires RW, Bove AA: Cardiovascular profiling. Clinics in Sports Medicine 3:11, 1984.



adequately and the heart will function properly at rest compensating this additional volume overload. This compensated state, however, has occurred in the presence of a loss in total myocardial functional reserve; and if additional stress is added in some instance, the myocardium may decompensate and the state of congestive heart failure will develop. The present state of knowledge on cardiac hypertrophy and its relationship to valve-induced overload states is not complete. It is not readily obvious which degree of valvular lesion will produce a decompensated state of ventricular function versus a well-compensated state although several clinical studies provide some guidelines (12,13). Studies examining functional reserve using radionuclide techniques suggest that the observation of deterioration of ventricular performance during graded exercise testing with a nuclear scan to measure left ventricular end diastolic volume and ejection fraction can be used to predict the response of the ventricle to exercise states (14). In this type of examination a reduction in ejection fraction, and increase in end diastolic volume at moderate exercise loads suggest that the heart cannot handle the valvular overload in addition to the exercise load and develops a state of heart failure. On the other hand, a well-compensated ventricle may show normal performance during exercise in the presence of valvular regurgitation, and such information would indicate that the individual is likely to be able to handle moderate levels of exercise associated with diving. The same criterion noted in the coronary artery disease section relative to workload (13 METS) can be applied to determine whether an individual with valvular heart disease can tolerate the exercise loads required in diving.

Ventricular responses to the volume overload lesions of mitral regurgitation and aortic regurgitation can be applied equally to both of these lesions when judging functional capacity. Both produce excess volume load on the heart, require the heart to enlarge to handle the additional volume, and both appear to be well tolerated at mild to moderate degrees; only when the overload lesion becomes severe does the ventricle seem to decompensate (15). Since exercise imposes an additional volume load by the requirement of increased cardiac output, one can readily understand why exercise in the presence of a severe volume overload lesion could quickly move the heart from a state of compensation to a state of heart failure. Immersion in water with central fluid shifts can also augment the volume load on the heart, and a marginally compensated heart with volume overload in this instance may demonstrate a heart failure response during the diving activity.

Clinical manifestations of cardiac decompensation from the volume overload lesions include dyspnea and acute pulmonary edema. Such individuals usually have low exercise tolerance and cannot function adequately at moderate-to-high workloads. This symptom complex can be examined by carefully done graded exercise testing to determine, as noted above, whether the individual can tolerate the exercise required for a diving program.

#### **Stenotic Valvular Lesions**

The lesions which produce stenosis of the mitral and aortic valve cause a different response under exercise stress than do the regurgitant lesions. Both aortic and mitral stenosis produce an obstruction to flow through the left heart chambers and ultimately can cause very high pressure gradients across the valve, which ultimately can affect the lungs in the case of mitral stenosis or

the left ventricle directly in the case of aortic stenosis. Of the stenotic lesions the most important to avoid in the diving environment is aortic stenosis. In this lesion, the first manifestation of the disease is often sudden death during exercise. Thus it is essential that aortic stenosis be examined for and ruled out prior to approving anyone for diving. The incidence of rheumatic aortic stenosis is extremely low in the United States, but the incidence of congenital bicuspid aortic valve appears to be constant in the population at about 2% (16). Of these 2% of the population about 1/3 develop a calcific stenotic aortic valve as age progresses and by the age of 40 it is evident that the patient has aortic valvular stenosis. Usually the valvular lesion progresses with time so that by the time the individual with this type of aortic valve reaches the age of 60 the stenotic lesion is significantly narrowed, symptoms may be evident, and the valve must be replaced.

Mitral stenosis likewise inhibits the forward flow of blood through the heart during exercise, causing increase in pressure in the left atrium, pulmonary venous hypertension, and congestion associated often with hemoptysis. The patient with mitral stenosis may not be aware of the presence of this lesion, and it is a commonly missed lesion on physical examination. Most mitral stenosis is due to rheumatic heart disease, but often the patient with mitral stenosis has no history of rheumatic heart disease. Streptococcal infection with a subsequent mild illness may be the only history. When the patient with mitral stenosis attempts to exercise, increased blood flow through the mitral valve raises the pressure in the left atrium and results in high pulmonary venous pressure with the symptoms noted above. A significant confusing factor may arise if a patient during diving has central fluid shifts and exercise that cause lung congestion from mitral stenosis. If individuals develop hemoptysis from this illness and severe dyspnea, they could be readily diagnosed as pulmonary embolism and treated inappropriately.

Left ventricular performance and the status of all cardiac valves is readily determined noninvasively using echocardiography. The combination of Doppler sonographic examination and echo sonographic examination of the heart can provide clear evidence of the status of the aorta, mitral, tricuspid, and pulmonic valves, the degree of regurgitation of any of these valves and the degree of stenosis. In addition, the size and performance characteristics of the left ventricle can also be determined using ultrasonic techniques, and this diagnostic modality should be used if any doubts arise concerning the status of the cardiac valves or the response of the myocardium to the valvular heart disease. Unlike occult coronary artery disease, most valvular lesions can be detected by careful physical examination. Their severity can be estimated by examination; however, more precise measure of the status of the valvular lesion is obtained by echocardiography. Once the status of the cardiac valves is known and judged to be either mild or moderate, an exercise test will provide information on the physiologic response of the candidate to exercise in the presence of mild or moderate valvular heart disease. Patients with severe aortic stenosis are generally recommended not to exercise because of the risk for sudden unexpected death. One exception to this concept is the presence of congenital aortic stenosis in adolescents and young adults. Normally, congenital aortic stenosis progresses in severity at an early age, and is manifest earlier than the aortic stenosis which develops from a congenital bicuspid valve. It is not uncommon to find young individuals with aortic

stenosis in the teens or early 20s who are exercising. These individuals should be exercise tested to determine the safety of unsupervised exercise. In any case these individuals are generally advised not to participate in competitive athletics. In the diving environment any excess exercise stress could result in a lethal outcome. Stenosis of the pulmonic or tricuspid valves is usually of less significance or consequence than stenosis of valves of the left heart. Subjects with significant pulmonic stenosis may be asymptomatic even though a loud pulmonic murmur can be detected on examination. Individuals with tricuspid stenosis may have elevated venous pressures and be prone to edema. Normally isolated tricuspid stenosis is uncommon. Usually this lesion is associated with lesions of the left heart, and the left heart lesions dominate the clinical picture. Congenital pulmonic stenosis is not an infrequently encountered finding and in general requires no specific treatment.

Regurgitant lesions of pulmonic and tricuspid valves are uncommon. Generally mild or moderately regurgitation of these two valves is of no consequence and the patients survive with minimal symptoms. Patients with tricuspid regurgitation may have this disease subsequent to endocarditis. In individuals with tricuspid endocarditis one should test carefully to determine if this candidate has used illegal intravenous drugs. Tricuspid valve bacterial endocarditis is a common accompaniment of the injection of contaminated drugs and is one of the common cardiac disorders found in chronic drug addicts. The presence of tricuspid or pulmonic regurgitation if moderate or mild and occasionally even if severe in an otherwise healthy individual who has no symptoms would not be a contraindication to diving.

#### **Intracardiac Shunts**

Of the disorders of the heart that can directly interact with the diving environment, the presence of a right-to-left communication in the heart is an important consideration when one realizes that many sport divers who appear to be asymptomatic following a dive within no decompression limits may have intravenous bubbles formed in the peripheral tissues. These normally would travel to the lungs and be excreted via the normal respiration. In the presence of a communication between the right and left heart, intermittent flows from right to left may carry gas bubbles from the right to the left heart and produce arterial gas embolism. Patients with atrial septal defects thus have an absolute contraindication to diving because of the high risk of paradoxical gas embolization and cerebral air embolism even when diving well within what apparently would be safe limits. Shunts at the ventricular level, ventricular septal defect (VSD) when small and asymptomatic generally flow early from left to right because of the pressure differential in the two cardiac chambers. However, in some portions of diastole a shunt may transiently reverse, producing a small jet of blood moving from right to left prior to the increase in pressure during early systole (17). During this brief reversal of shunt flow, bubbles may cross the ventricular septum and appear in the arterial circulation. Although the net shunt in ventricular septal defect is commonly from left to right, this intermittent reversal of the shunt in diastole, although physiologically inconsequential, may carry gas emboli into the arterial circulation. Thus patients with ventricular septal defects also should be advised not to dive.

A number of cases of cerebral embolism appear to have occurred in the absence of any significant pulmonary overpressure. It has been suggested by some individuals that the transient rise in intrathoracic pressure, such as during a Valsalva maneuver, can cause a patent foramen ovale to open for brief periods to allow small amounts of blood to move from right to left of the heart. In some studies it has been estimated that a patent foramen ovale may exist in up to 18-20% of a normal population (18), thus individuals with a patent foramen ovale are common among the population and one could predict that about 1/5 of all sport divers have a patent foramen ovale. This situation can lead to brief right-to-left flow of blood across the foramen ovale when pressure changes in the thorax are favorable. Echocardiographic data using microbubble contrast have demonstrated a brief right-to-left shunt across a patent foramen ovale during the Valsalva maneuver (18). This response indicates that divers who had bubbles appearing in the venous circulation can force bubbles into the arterial circulation by a Valsalva maneuver if a patent foramen ovale is present. At the present time there appears to be no need to test for this physiologic entity. However, in individuals who have unexplained air embolism from what appears to be normal diving operations, it would be useful to determine first whether the foramen ovale is patent and second whether flow across the foramen ovale can be induced by changes in intrathoracic pressure.

Arteriovenous shunts in the peripheral circulation are unlikely to produce embolism from the venous to the arterial circulation. These shunts are truly unidirectional, flowing from left to right throughout systole and diastole, and such shunts would not carry bubbles in a retrograde fashion into the arterial system. Patent ductus arteriosus falls in this category. In a small patent ductus which might not be repaired because of inconsequential hemodynamics, the flow is continuous throughout systole and diastole from the aorta to the pulmonary artery, and bubbles are unlikely to transit from pulmonary artery to aorta. On the other hand, if a patient has a long-standing patent ductus with a marked rise in pulmonary resistance and therefore pulmonary artery pressure, pulmonary pressures begin to reach systemic pressures and a right-to-left shunt across the patent ductus will occur. In this case bubbles can embolize paradoxically. On the other hand, individuals with such severe pulmonary disease secondary to a patent ductus are generally in poor physical condition, appear cyanotic, and usually are too dyspneic to consider participation in a diving activity.

### **Hypertension**

High blood pressure is estimated to be present in 12 million or more American adults (19). This high incidence of hypertension is a serious public health problem and detection of hypertension is an important role that the physician must fulfill. Values of blood pressure above the usual norm (120/80 mmHg to 130/88 mmHg) should be pursued by subsequent blood pressure measurements to determine if the recurrent pattern of blood pressure is elevated above normal. Risks for coronary artery disease and stroke increase even with moderate elevations in blood pressure, and it is imperative for the physician to detect even mild-to-moderate levels of blood pressure and attempt to achieve normal blood pressure levels. The central fluid shifts attendant with total water immersion, the cold stress, emotional stress, and exercise associated with diving will all contribute to a further rise in blood pressure

in individuals prone to hypertension. Individuals who demonstrate severe elevations of blood pressure during these stresses should not be approved for diving but should be provided with some therapy to bring blood pressure within normal range including responses of blood pressure to exercise stress. If this can be achieved with moderate levels of medication, with exercise, and with salt restriction, then the individual can resume diving or begin diving. On the other hand, if blood pressure elevation is severe and drugs which produce significant autonomic paralysis are used for therapy, then the subject will have difficulty with exercise tolerance and usually cannot fulfill the treadmill exercise criteria mentioned above without either severe dyspnea, evidence of myocardial ischemia, or arrhythmias. Patients taking beta blockers alone for mild hypertension are not limited in their diving. Normally, moderate amounts of beta blockers, although reducing the heart rate response to exercise generally do not alter exercise tolerance (20). Larger amounts of beta blockers, however, can limit maximum exercise output, and may inhibit performance when extremely high workloads are required. Patients using other vasodilator agents such as prazosin, hydralazine, and captopril may demonstrate orthostasis or autonomic insufficiency during exercise, and should not dive if these responses are known to occur. The use of a diuretic in general will not produce a problem in diving, however, the associated potassium loss noted from diuretics as well as mild chronic hypovolemia may make the diver less capable of handling the stresses of diving. Individual judgment should be exerted in these cases. A reasonable approach would be to expect the diver candidate to meet the treadmill exercise requirements in the presence of medication prior to clearance for diving.

One should note also that presence of chronic hypertension is a significant risk factor for coronary artery disease, and if individuals with hypertension are to be evaluated for diving, a careful search for occult coronary disease should be undertaken.

#### **Congestive Heart Failure**

Although the most common cause of congestive heart failure in the American population is coronary artery disease, other causes may be less obvious and may result in significant cardiovascular decompensation during diving. Coronary artery disease produces congestive heart failure by damage or infarction to significant portions of the left ventricular myocardium and lack of adequate remaining myocardium to handle a normal cardiac workload. Usually, patients with this degree of coronary disease are severely limited because of myocardial damage and often by angina pectoris. The history is rarely benign. Usually a history of several myocardial infarctions ranging over many years is evident. An exception to this is the small recurrent asymptomatic myocardial infarctions known to occur in patients with diabetes who can appear initially with congestive heart failure prior to any clear manifestations of coronary artery disease. These individuals are usually in the 50-60 year-old population and have severe clinical diabetes. In most cases these individuals would be excluded from diving because of the presence of insulin dependency and should not raise a significant issue because of the cardiovascular aspects.

Other types of congestive heart failure, however, can be occult in response to various causes of cardiomyopathy. Mild myocarditis with subsequent myocardial damage is known to produce decompensation of left ventricular performance on a chronic basis. Excess long-term ethanol ingestion also produces myocardial damage, as do a number of other disease processes. In most cases, however, the damage to the myocardium is slow and progressive and is not often detected until severe. The most common initial manifestation of cardiac decompensation due to cardiomyopathy is a loss of exercise tolerance. This usually occurs first with moderate levels of exercise and then in exercise activity required for daily living such as climbing stairs, walking rapidly, lifting, or carrying moderately heavy objects. Another manifestation of early cardiomyopathy is cardiac arrhythmia, and many younger individuals who appear with intractable or serious cardiac arrhythmias have underlying cardiomyopathic processes. In all cases of cardiomyopathy the function of the heart is diminished. The severity of the functional degradation varies and in many individuals, may be mild enough that it is undetected in normal activity. The evidence for cardiac decompensation, therefore, may only appear during severe or moderately severe exercise or in the event of central fluid shifts plus exercise that is common in the diving environment. Individuals suspected of having cardiomyopathy can be examined successfully using echocardiography. The cardiac size and contractile performance can be determined by echocardiography. Performance of the heart during stress or exercise can be tested using radionuclide techniques during exercise and, as noted previously in relation to valvular heart disease, cardiac decompensation during exercise is further evidence that myocardial damage is significant enough that the individual should not undertake diving.

#### **Cardiac Pacemakers**

Two important issues arise when considering patients with cardiac pacemakers. First is the type of pacemaker and its response to exercise, and second is the ability of the pacemaker to withstand increased ambient pressure. With regard to the latter, most pacemaker companies now have published information on the pressure tolerances of their devices and this can be found in the information provided with the instrument. For sport divers, pacemakers should be able to withstand pressures to approximately 130 fsw. This appears to be a design parameter of most pacemakers so that the presence of the pacemaker alone would not be a contraindication to diving.

Of more importance is the reason for the presence of the pacemaker and its type and function. Patients with severe heart disease with associated conduction system abnormalities should be evaluated for the underlying heart disease and considered on that basis, rather than on the presence of the pacemaker. Patients with fixed-rate pacemakers may not have adequate exercise tolerance in the diving environment because of the inability of the pacemaker to increase the heart rate above a preset level. Many new pacemakers are designed to sense the sinus node signal from the atrium and stimulate both the atrium and ventricles in a physiologic manner. These pacemakers (often called AV sequential pacemakers) can cause the heart to respond normally to exercise and will often produce a physiologic response to various types of stress or exercise. Individuals with otherwise normal cardiovascular systems who have pacemakers of this type are likely to dive successfully without significant problems. However little experience at present is available in this area and

advice should be provided with caution and with careful evaluation of the cardiovascular status of the individual.

#### CONCLUSIONS

Diving with cardiovascular disorders must be considered on an individual basis and on the basis of a good examination and evaluation of the heart and circulation. The wide variety of cardiac disorders precludes a general statement concerning sport diving in association with cardiovascular disease. Many cardiovascular diseases are benign and of no consequence throughout a lifetime, whereas others are serious and can result in sudden death as the first manifestation. It is imperative that the physician evaluating a diver for cardiac disease determine the type of disease and the severity as well as the individual's response during exercise in the presence of this disease. Often consultation with a cardiologist will be required, but it is unreasonable to reject out of hand all individuals who have cardiac disease without understanding the extent of the disease and the likelihood for limitations in the sport diving environment.

#### REFERENCES

1. Manual of the Medical Department--U.S. Navy. Article 15-36. Washington, DC: Naval Medical Command, Aug. 1984.
2. United States Underwater Fatality Statistics. Rep #URI-SSR-77-11. University of Rhode Island, 1977.
3. Sheldahl LM, Tristani FE, Clifford PS, Kalbfleisch JH, Smits G, Hughes CV. Effect of head-out immersion on responses to exercise training. *J Appl Physiol* 1986; 60:1878-1881.
4. Bove AA, Hardenberg E, Miles JA. Effect of heat and cold stress on inert gas (<sup>133</sup>xenon) exchange in the rabbit. *Undersea Biomed Res* 1978; 5:149-158.
5. Raizner AE, Chahine RE, Ishimori T, et al. Provocation of coronary artery spasm by the cold pressor test: Hemodynamic arteriographic and quantitative angiographic observations. *Circulation* 1980; 62:925.
6. Spencer MP, Campbell SD, Sealey JL, Henry FC, Lindberg J. Experiments on decompression bubbles in the circulation using ultrasonic and electromagnetic flowmeters. *J Occup Med* 1969; 11:238-244.
7. Lown B. Cardiovascular collapse and sudden death. In: Braunwald E, ed. *Heart disease*. Philadelphia: W.B. Saunders, Co., 1980: 778-817.
8. Bove AA, Squires RW. Exercise conditioning alter the circulatory response to the cold pressor test. *J Amer Coll Cardiol* 1985; 5:518.
9. Maseri A, Mimmo R, Chierchia S, et al. Coronary artery spasm as a cause of acute myocardial ischemia in man. *Chest* 1975; 68:625.

10. Levy RF, Feinleib M. Risk factors for coronary artery disease and their management. In: Braunwald E. ed. Heart disease. Philadelphia: W.B. Saunders, Co., 1980: 1246-1278.
11. Alpert JS. Mitral regurgitation: Mitral valve prolapse. In: Dalen JE, Alpert JS, eds. Valvular heart disease. Boston: Little Brown and Co., 1981: 153-170.
12. Spann JF, Bove AA, Natarajan G, Kreulen TH. Ventricular pump performance, pump function, and compensatory mechanisms in patients with aortic stenosis. Circulation 1980; 62:576-582.
13. Osbakken MD, Bove AA, Spann JF. Left ventricular function in chronic aortic regurgitation with reference to end-systolic pressure, volume, and stress relations. Am J Cardiol 1981; 47:193-198.
14. Borer JS, Bacharach SL, Green MV, et al.. Exercise-induced left ventricular dysfunction in symptomatic and asymptomatic patients with aortic regurgitation: Assessment with radionuclide angiography. Am J Cardiol 1978; 42:351-357.
15. Osbakken MD, Bove AA, Spann JF. Left ventricular regional wall motion and velocity of shortening in chronic mitral and aortic regurgitation in man. Am J Cardiol 1981; 47:1005-1009.
16. Levinson GE. Aortic stenosis. In: Dalen JE, Alpert JS. eds. Valvular heart disease. Boston: Little Brown and Co., 1981: 171-230.
17. Miyazawa K, Smith HC, Wood EH, Bove AA. Roentgen videodensitometric determination of left to right shunts in experimental ventricular septal defect. Am J Cardiol 1973; 31:627-634.
18. Lynch JJ, Schuchard GH, Gross CM, Wann SL. Prevalence of right to left shunting in a healthy population: Detection by valsalva maneuver contrast echocardiography. Am J Cardiol 1984; 53:1478-1479.
19. Hypertension Detection and Follow-up Cooperative Group: Five year findings of the hypertension follow-up program. JAMA 1979; 242:2562-2571.
20. Tesch PA. Exercise performance and  $\beta$ -blockade. Sports Med 1985; 2:389-412.
21. Lawlor MR, Thomas DP, Michele JJ, Carey RA, Paolone AM, Bove AA. Effect of chronic  $\beta$ -adrenergic blockade on hemodynamic and metabolic responses to endurance training. Med Sci Sports Exerc 1985; 17:393-400.



#### DISCUSSION FOLLOWING PRESENTATION BY DR. BOVE

NEUMAN: How long after someone has had a coronary artery bypass would you wait before evaluating them for diving?

BOVE: First of all, I insist that the coronary bypass patient get into a cardiac rehab program. By the mere fact that the patient had bypass surgery you know what the angiogram looks like. Coronary bypass surgery is never done without an arteriogram. Fortunately we know what the arteriogram shows and we know what the surgeon did. In one example, you may have a person that has severe multivessel disease and the surgeon bypassed the 4 worst lesions. That patient will still have coronary symptoms. Another example is the 40-year-old male with a single anterior descending lesion. It is symptomatic and the internal mammary artery is anastomosed. Subsequently, no occlusive regions remain. The scar heals and is nearly invisible. A chest x-ray will show the sutures, otherwise you might not know the subject had bypass surgery. There is a spectrum of disease. To deal with the spectrum I insist that the individual who wants to go back to diving follow a rehab program for 6 months post-op. We recommend for any patient who is returning to activity a six month rehab program, with the first 8 weeks supervised, and then subsequently on their own. A diagnostic treadmill at 8 weeks, and then a treadmill at 6 months. The treadmill at 8 weeks is limited and the treadmill at 6 months tests the 13 MET goal. At the end of 6 months, if the subject has been working out faithfully, with swimming or jogging or whatever else (if the patient is interested in aquatic sports, put them back in the water as part of their rehab program) the treadmill test must be normal. That is, no ischemia, normal blood pressure response, no severe dyspnea or angina. Then, I would let that person return to diving. Now, the patient with every vessel diseased with 4 bypasses is not going to pass that test. The diagnostic treadmill test will demonstrate to him that diving will be dangerous.

ARMER: It's a practical question. An individual has mitral prolapse, has been diving and has a normal resting cardiogram. A stress cardiogram shows runs of ventricular arrhythmia after a short run. The question is then, first, if he was just getting ready to start diving, would you let this man dive? And second, if he was an experienced scientific diver in the midst of a long-term project and the diving activities were very specific that he needed to do, would you permit him to dive?

BOVE: It's very straightforward. I consider mitral valve prolapse to be several diseases. It is a disease of arrhythmias, a disease of a click that the valve makes, and a disease of mitral regurgitation. In fact, a recent study using echocardiography demonstrated that half of a group with mitral prolapse had no physical signs or symptoms at all, which means that a reported 15 percent incidence is really 30 percent. With this incidence prolapse can't be a disease if one-third of the population has some evidence of mitral valve prolapse. We often treat the symptomatic person with beta blockers. A beta blocker usually is adequate for the ventricular arrhythmia that occurs with mitral valve prolapse. In this patient the beta blockers are usually efficacious, and my approach would be to give a beta blocker, then test the patient on the treadmill to 13 METS, and then put them back to full activity as long as no significant arrhythmias were noted with the beta blocker therapy. A beta blocker generally won't limit a

younger, healthy individual from doing 13 METS. It will limit his maximum exercise. His 10 K time will go from 32 to 38 minutes, if he's a good runner. But his 13 MET time is unlikely to change at all.

NEUMAN: You talked about the person who had an abnormal electrocardiogram. You did not discuss the person who has no symptoms or signs of coronary artery disease but, for a flight physical or some other reason, gets a stress ECG and now has an abnormal electrocardiographic response. What is your feeling in that area?

BOVE: I think they have two choices. Either stop diving or have a coronary angiogram to find out what's going on. I think the general approach ought to be first: Exercise stress is the key issue with cardiovascular disease and the diving environment. I think diving should be considered as a high exercise environment or at least potentially so, because when a person is committed to a dive there is work needed for carrying the gear, climbing up and down ladders, and the possibility of swimming against winds, tides, and currents. Even though diving might be easy and a diver with limitations could dive safely, the circumstances that arise unexpectedly must be considered. I recommend the treadmill test. Now, if you take a population below 30 years of age who are otherwise healthy, who have no significant medical history, you will find that the incidence of coronary disease is extremely low. Other diseases like valvular heart disease should be detected by history because most patients with significant cardiac abnormalities have a past history of being in the medical care system. With a totally benign history, you are unlikely to have somebody in their 20s with coronary disease. The one exception to that is cocaine. Apparently cocaine damages the coronary arteries, and produces a very early lesion in the coronaries that looks like coronary disease. It's a little different but it produces occlusion of the vessels in young patients.

Aside from that, there isn't any need to do a treadmill test on a 22-year-old who is otherwise healthy and is in reasonable physical condition. When you get into the 30s, there is a gray zone. The way I deal with people in their 30s is to examine their risk factors, look at their level of conditioning and what exercise they do. If they have a high number of risk factors such as being overweight or not exercising, I do a treadmill test. Beyond 40, unless it is a 42 year old marathon runner that proves himself every day by his exercise training--and even here I question--I recommend a treadmill test. What I am looking for here are signs of ischemia, especially asymptomatic ischemia. If they have ischemia, they are referred for further evaluation. If they are normal they can go ahead with their diving program.

LINAWEAVER: The interesting thing is how to get a person in for that treadmill. There has got to be some sort of a flag. Your slide shows asymptomatic people that you had to get up to a significant exercise rate to achieve that, yet the people that we see dying and are labeled sport diving deaths are mostly cardiovascular deaths except those that clearly have either decompression or air embolism accidents--but run-of-the-mill diving death, at least in our experience in California, is cardiovascular. The question is how to get them in? Because if they fill out a questionnaire, most of them are asymptomatic.

BOVE: Inform the diving training organizations. The physical questionnaire is fine for 20-year-olds, but beyond a certain age, whatever it is, 38 perhaps, it is imperative that these individuals have physical exams because the incidence of disease increases in that population. Beyond the age of 40, before you accept a diver into a scuba program, he ought to see a physician. Below that, there is a gray zone and in the 20s, I don't think it is necessary unless there is a flag on the questionnaire.

GREER: Doesn't that mean that if he's over 40 or over some arbitrary age that he should see a physician and have his exercise tolerance studied or established. Or is it good enough just to have him see a physician and the family doc sign off and say it's okay for him to dive?

BOVE: There is a population of 40+-year-olds that are vigorous and healthy and exercise. They are usually safe for a scuba program without an exercise test if I know they go out and run 5 or 6 miles a day. There are a few of those people. The deconditioned subject needs a stress test before the stress of diving.

GREER: Once you have established in a middle-aged man that his exercise tolerance is adequate, when do you repeat it? A fifty-year-old diver should have a treadmill how often?

BOVE: Every two to three years is adequate if a person is otherwise healthy.

ARMER: Just a simple straightforward question regarding scientific divers. Should they have a plain electrocardiogram initially before they start diving or should they also have the treadmill? Also, should they have them repetitively, annually, or should it change with age?

BOVE: If you have a group of people coming into the system at 22 years old, I think a physical exam with a chest x-ray is adequate. A resting electrocardiogram is worthless except as a baseline to put on record for 10 years later. You could justify an ECG, but it is not necessary. If a subject enters the diving community at age 42 I would then do a treadmill test, absolutely. And I would probably repeat it every 3 or 4 years in the 40s and maybe every 3 years in the 50s.

DUEKER: I'm amazed that nobody is playing the "devil's advocate" on stress testing, because if you read the literature there's a lot of controversy whether it is really valuable.

BOVE: I think it's a very straightforward issue. The question often raised is: Can you use a stress test to predict the involvement of the coronary arteries with atherosclerosis? In the right hands it is about 80 percent accurate; some people will say 50 or 60 percent accurate, in predicting coronary disease. The issue for diving is in predicting an adverse incident with exercise. Here the test is valuable. Those who do a lot of stress testing can be about 80 to 85 percent correct. We have verified this with angiography. But that isn't the issue. Patients may have many minor (20 percent) lesions in their coronary arteries. You cannot detect this level of disease with a treadmill test. What I want to know is if that person becomes ischemic during exercise. If the test is normal the coronary arteries are normal. It is a statement that there are no

critical lesions which produce ischemia. In spite of the fact that detection of a lesion in a coronary artery is less than perfect with a treadmill test, the detection of ischemia is reliable with a treadmill test. If a subject can exercise at 13 to 15 METS without ischemia or arrhythmias, I am quite comfortable about him tolerating that level of exercise in a different environment.

YOUNGBLOOD: Just to reiterate, let's remember, at least in my perception, that we're looking at the treadmill test for two things. One is to rule out ischemia and the other one is to rule on physical capacity. And to me, that is extremely important. I've tried for years and failed to get the commercial diving industry to do that, because unfortunately, contrary to popular impression, the average commercial diver is not a terrifically fit person.

LINAWEAVER: It is interesting that you've had trouble with commercial industry. In the work that I do for the commercial industry, I require a stress test for divers going into the business for a company or transferring to a new company, then at age 30 and every three years after until they reach age 40. After 40, I require an annual physical for those two reasons that you discussed. But I've never had any trouble with the commercial diving companies doing that. We found that we had to ground a few divers who weren't in shape until they got in shape. So it does work.

ALEXANDER: Two comments. One, when we're defining cardiovascular death, how are we defining that? Among the Korean War dead, significant coronary artery disease was found, obviously not the cause of death, in young individuals who had no symptoms. Second, with regard to the exercise capacity of divers, I think commercial, scientific, and other professional divers know that what they're going to be required to do exercise wise. We should stress that people have the idea that sport divers don't need to do heavy exercise. It's nice, you look at a fish and come up. But they are equally at risk because they have unpredictable requirements of very high stress and they're probably at much more risk because they're not screened as well for exercise ability as is the commercial diver.

LINAWEAVER: To answer your first question, it depends on the jurisdiction that the death occurred in. In Santa Barbara we had five diving-related deaths last year. Four were clearly cardiovascular. The criteria was the presence of significant atherosclerosis. One had a silver dollar-sized, healed infarct and a subintimal hemorrhage, but the others were based on a very careful history of the accident. We have a good sheriff's coroner's department which goes into great detail on circumstances of such events. For example, a diver was swimming through the kelp with gear, maximum effort, sudden cessation of activity, and then subsequent immersion. Immersion effects were there, but the history was clearly that some catastrophic event had occurred and most likely a dysrhythmia.

BOVE: Data from Korean War casualties demonstrated early atherosclerosis. It showed plaquing in the vessels. It did not show severe obstructive lesions in these 20-year-olds. This observation is the basis of the statement that early atherosclerosis goes back to early life. The post-mortem data that I've seen in sudden deaths in the water have severe, three-vessel coronary disease. This severity of disease, if known, would result in a consultation with a surgeon for bypass surgery. This is severe three-vessel disease which predictably would have caused arrhythmias or sudden death with heavy loading of the heart. These people

were unaware or didn't admit to it. These lesions become critical before being detected. In the absence of any significant exercise they will go undetected. In fact, the literature now indicates that of all episodes of ischemia detected by monitoring, 85 percent are silent. Thus, subjects with coronary disease who are having ischemic episodes don't detect 85 percent of them. There is a large amount of undetected asymptomatic ischemia in subjects with coronary disease. That is what makes a person prone to sudden death with a heavy sudden exercise load. It's the snow-shoveling syndrome. You can get into the same environment trying to swim through kelp in a current with heavy gear on.

The second point I want to make is that if we recommend exercise testing, we must be prepared to deal with the "false positive" problem. The Air Force experience is a good one. They stress test all their high performance pilots. They pick up a 17 percent incidence of positive stress tests every year. This is in a population between 25 and 40 years old. Those with a positive stress test get angiograms. To maintain pilot status, they must demonstrate normal coronaries. There is less than a half percent incidence of coronary disease in those angiograms.

ARMER: You mean of the 17 percent that have angiograms, only a half percent of those have coronary disease?

FARMER: So it's 0.5 percent times 17 percent?

BOVE: It's quite low. The procedure is benign and complications of the angiograms are minimal.

LINAWEAVER: Which table is the table of truth?

BOVE: For the present, the angiogram; the pilots go back to flying status based on the angiographic data. Even though they get stress-tested again they don't come back for another angiogram. Now, this is an expensive program. But, that is the approach right now. There are developments going on in the cardiovascular community, based in part on this Air Force information, on what to do with the false-positives.

I think the idea is that those who have positive stress tests, be pulled out of the diving environment and evaluated thoroughly to be sure that the information is properly addressed. It may be that some of those people will go to angiography, and other studies can be done; thallium studies improve reliability.

YOUNGBLOOD: Has the Air Force compared what the thallium does?

BOVE: Yes, but it doesn't help enough. They go to the angiogram. Thallium helps, but because these pilots fly expensive airplanes, they want to know what their coronaries look like. They haven't had any complications from the angiographic procedure itself. Remember these are basically healthy people, so the risk for the angiogram is extremely low. If there is a positive stress test, you are obligated to respond and the way to respond is to get a knowledgeable cardiologist to help evaluate the patient and add an appropriate series of studies to understand what is going on.

GREER: Does that mean that there's a 17 percent expected false-positive in angiograms?

BOVE: In young males in stress tests, and it approaches 50 percent in females.

LINAWEAVER: Given our generic approach to this conference, is there any interrelationship between the cardiovascular realm and exposure to pressure per se.

BOVE: No, unless you're diving to 2000 feet.

LINAWEAVER: How about the patent foramen ovale.

BOVE: The problem of silent bubbles crossing a patent foramen ovale is real and anybody with a documented right-to-left communication at the atrial or ventricular level should not be allowed to dive because they run the risk of paradoxical embolization to the brain.

LINAWEAVER: The aquatic aspect is based, therefore, on effort related to propelling through the water.

BOVE: Effort, cold stress, emotional stress, anxiety, fear, all of those things.

LINAWEAVER: I think those are very important factors. Cold, emotion, and effort.

BOVE: The hyperbaric problems are the pacemaker pressure effects, which I think are within reason and acceptable down to about 100 feet for a sport diver. I think they're testing the pacer cases to 130 feet.

LINAWEAVER: Doesn't the reason that the guy has a pacemaker enter into it?

BOVE: That is the key issue. There are people with pacemakers that were put in for transient diseases where a pacemaker does not often function. As a single entity, the presence of an implanted pacemaker for a sport diver, if that is the only thing, is tolerable, but you must look for underlying heart disease that may or may not be present.

YOUNGBLOOD: Don't you have to know which pacemaker and how old it is? Some of the older ones, I believe, will not tolerate pressure.

BOVE: I think the first time that I asked about this was in 1978 or 1979, and by now almost all the pacemakers from that era have been replaced. Yes, I think you should ask when it was put in. You'll probably never get an answer that it was eight years ago. Almost all of them have been changed in three to four year cycles so I think by now almost everybody will have a pacemaker that has been tested to 130 feet.

ALEXANDER: Since we're saying cardiovascular deaths are number one, we should make very positive statements on how to evaluate this, especially in people who

have no symptoms, because of the high stress level. Maybe we should drop them a little bit below where we normally put people who are nondivers.

LINAWEAVER: The standards that exist now, gloss over the history of angina, the history of myocardial infarction, etc., but they make no reference at all to the age group and the possibility of significant asymptomatic arteriosclerotic heart disease.

BOVE: I have addressed that in the document submitted. I agree. Anyone with clinically documented coronary disease shouldn't dive. I gave some exceptions: young individuals that had a single infarction who can pass the 13-MET stress test or the person that had essentially total revascularization after a bypass procedure who is a sport diver, who had been diving, who wants to get back, and who can pass the 13 METS on a stress test. I think they can go back to diving.

## PULMONARY CONSIDERATIONS I

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The problem of advising individuals with pulmonary disorders on the risk of diving is a particularly vexing one. Whereas most physicians can understand the risk of not being fit enough if caught in a strong current, or not being able to equalize if they have trouble clearing their ears, it is a relatively unusual physician, let alone diver or diving candidate, who can understand the concept of increased risk of air embolism because of abnormal distribution of ventilation.

### ASTHMA

Let us begin with asthma, because this is the most common pulmonary disorder we are asked to evaluate in a diver or candidate. Fortunately, most of us have a basic understanding of what asthma is because the participants of the CIBA Foundation study group on the "Identification of Asthma" concluded, "Asthma could not be defined on the information at present available." (1). (In that respect, asthma seems to be somewhat like pornography; we can't define it, but we all know it when we see it.) For our purposes, it might be more useful to define asthma as a disorder(s) "Characterized by increased responsiveness of the airways (i.e., they are hyperreactive) to various stimuli and by resultant smooth muscle contraction and obstruction." (2). Yet asthma can still be subclassified and most current classifications attempt to define specific syndromes based on specific precipitating factors and specific patterns of response. These classifications are important because the natural history of these different syndromes appears to be different, and therefore the recommendations of diving physicians might well be different.

One of the more common asthma syndromes is atopic asthma. This is characterized by onset in childhood and association with allergic rhinitis or allergic dermatitis. Attacks may be precipitated by various sensitizing agents. Children who develop this asthma syndrome in the first few years of life do not necessarily have persistent asthma into adult life. In one study only 15% of children ten years old whose asthma began before the age of two had persistent wheezing (3). If the precipitating cause of attack is infection, it was found that 50% of children were considered to be "cured" when re-examined twenty years later (4). Adults do not seem to fare as well with asthma having a much lower percentage of individuals who become free of disease with time (5-7), and the proportion of severe-to-mild cases seems to rise steeply with age (8).

In many patients, however, attacks cannot be ascribed to a specific precipitating agent or event. This syndrome is called "intrinsic" asthma; however, there is a great deal of overlap between groups and many individuals present a "mixed" picture (9-10).



Another important syndrome is exercise-induced asthma. The cause of this syndrome, most frequently seen in the young, was for many years unclear. Evidence now suggests that the underlying cause is not hyperventilation, hypocapnia, etc., but rather cold stimulus to the tracheobronchial tree in susceptible individuals (11,12).

Other less common syndromes are the aspirin sensitivity triad (nasal polyps, urticaria, and asthma following aspirin ingestion) and occupational asthma.

Thus, asthma is not a single disease and there is a tremendous degree of heterogeneity among asthmatic patients. As mentioned previously, the factors that precipitate attacks vary tremendously. But equally important, the actual location of obstruction seems to vary with different patients, depending on what you believe various pulmonary function tests measure.

Most importantly, there is considerable difference in the degree of reversibility of airway obstruction that asthmatics demonstrate. Some asthmatics demonstrate completely normal pulmonary function tests "including measurements of pulmonary mechanics and of regional ventilation distribution" between attacks (13). Whereas others, although asymptomatic, continue to show evidence of airway obstruction even after vigorous therapy. Under such circumstances, the distinction between chronic obstructive lung disease (COPD) and reactive airway disease (RAD), i.e., asthma, becomes muddled.

As a result of these marked differences in prognosis, in pulmonary function studies between acute episodes, and the baseline function, recommendations concerning diving for the individual patient should take into consideration the patient's asthma syndrome and history.

As everyone here is aware, the major concern in permitting the asthmatic to dive is that such an individual might be dangerously susceptible to cerebral air embolism even during a normal ascent. This concern is based on our understanding of pulmonary physiology and extrapolations based on that understanding. Liebow has demonstrated that under certain conditions, partial pulmonary obstruction in large airways can lead to arterial gas embolism (AGE)(14). Similarly, Schaefer has demonstrated that overpressurization of the lung can lead to AGE (15), and Colbatch has shown that decreased compliance is associated with AGE (16). All of these factors are operative in asthmatics. Coupled with this, some asthmatics have significant noncommunicating air spaces as demonstrated by differences in measured lung volume compared by helium dilution techniques and whole body plethysmography (17). Additionally, the acute asthmatic is usually hyperinflated and, as has been demonstrated, not only is overpressurization required to produce AGE, but overinflation is as well (15). More sophisticated studies on asthmatics reveal that even in asymptomatic individuals [although they all had abnormal pulmonary function tests (PETs) or arterial blood gases], marked abnormalities of ventilation perfusion ratios can exist. This study demonstrated that as many as half of the lung units were perfused by completely closed airways that were ventilated only by collaterals (18). Finally, it is clinically well known that patients with severe bronchospasm when given IPPB develop barotrauma (pneumothorax,

pneumomediastinum), and at times such individuals will develop pneumomediastinum spontaneously.

Thus, there is a wealth of physiologic data that suggest it may be unsafe for the active asthmatic to dive. This concern, however, has been transformed into dogma that asthmatics are more susceptible to air embolisms and it is unsafe for them to dive.

It is, therefore, worthwhile to examine the diving accident statistics on air embolism that are available, and to attempt to assess realistically the underlying factors in AGE and to see if asthmatics make up an excessively large percentage of victims. Since asthma is found in approximately 4% of the population (19), such an examination should be meaningful if asthmatics dive. (I assume now that there are significant numbers of asthmatics who dive. As I mentioned in my previous discussion, it seems that almost every diving trip I go on has several asthmatics in the group. Similarly, I believe Dr. Linaweaver will attest that even in his local area he has been unable to screen out asthmatics from some diving programs.)

Perhaps the largest series of diving fatality reports currently available come from the National Underwater Accident Center in Rhode Island. Examining these reports dating from 1972 to 1982 reveals that air embolism is a major factor in diving fatalities (20-29). Unfortunately, no data are available that describe the precipitating factors that produced the air embolisms, other than general comments about buddy breathing, out-of-air situations, and panic ascents. At no point in these reports is there mention that significant numbers of these fatal air embolisms may have been precipitated by underlying lung disease. I could find only one death in all of those reports that was ascribed to previous lung disease and that was "pneumonia."

Reviewing the air embolism reports from Hawaii from 1976-1979 revealed that there were 42 cases during this four-year period (3). Two of these 42 cases were asthmatics; however, in both cases the AGE was associated with an out-of-air-at-depth, uncontrolled ascent situation (31).

Of all the New Zealand fatalities in 1981-1982, one of 11 divers who died was asthmatic. The conclusions in that case were "No evidence of pulmonary barotrauma. Pool situation. Untrained. Inexperienced. Out of air. Borrowed/hired equipment, using another's authority. Asthma bronchial changes. Inaccurate gauge. Alone. Buddy ignored warning about loaning." (32). Considering the egregious breeches of diving safety, it is not clear to me that this fatality can be ascribed to the underlying lung disease either. New Zealand statistics from 1982-1985 listed one of 21 fatalities associated with asthma; however no specifics of that case were described (33).

Australian fatalities in 1984 reveal that one victim out of 13 was asthmatic. This individual was described as a "mild smoker" but likely on occasion to smoke heavily. A visit to his unit revealed four empty "Ventolin" inhalers, but his sister and mother vigorously denied he suffered "asthma" although admitted "he did until he was eight years old." This case too, was an uncontrolled ascent with the regulator out of the victim's mouth, thus also an apparently "deserved AGE (34)."

In 1985 DAN reported the results of reports they received in 1981-1982 (35). Of 39 AGE, only one was ascribed to medical predisposition, but neither the exact medical condition nor the details of the incident are described. Perhaps more interestingly, they state that there was no identifiable cause in 50% of the incidents.

A personal communication from A. Pilmanis at the Catalina Marine Science Center provides similar information. Although he does not have exact statistics, he remembers "a few" air embolism in asthmatic divers, but in all cases the AGEs were "deserved" (i.e., out-of-air, uncontrolled ascents). My own personal experience in San Diego is similar. Of the approximately half-dozen AGEs per year, I have seen no air embolisms or pulmonary overinflation syndromes in asthmatics in the last ten years.

Thus, it appears that asthmatics are not overrepresented in gas embolism statistics, and that when they get air embolisms they get them in the same way and under the same circumstances as other divers who are not "predisposed." This is not, however, meant to suggest that all air embolisms can be ascribed to panic, out-of-air, or breath-holding situations. It is clear from the experience at Catalina, Hawaii, the DAN report (35), and my own experience that on occasion pulmonary overinflation syndromes and/or air embolism occur in the setting of normal and controlled ascents. Pilmanis (36) and Kizer (31) believe approximately 5-10% of the AGEs they have seen cannot be explained by out-of-air, uncontrolled ascent circumstances, and in my own experience, I have seen four such cases in San Diego. In contradistinction, from these estimates, the DAN report suggests approximately 50% of the AGEs cannot be explained by situational causes. What makes these cases particularly disturbing is that none of the cases Dr. Kizer, Dr. Pilmanis or I have seen have had predisposing medical (pulmonary) conditions identified that could theoretically have been discovered in a pre-dive physical examination.

Thus, on the one hand we have sound theoretical reasons suggesting that diving with asthma predisposes to AGE, but with little hard accident data to support this. Yet it is also clear that a percentage of AGEs do occur in the setting of "normal" ascents to apparently "normal" divers.

What then are reasonable recommendations that can be made in light of available data? Recommendations that are on the one hand safe, yet on the other hand not overly conservative? It would seem that there may exist a subset of individuals with asthmatic diathesis who in all probability are safe to dive (if for no other reason than that they are already diving and do not have increased accident statistics). Yet it is equally clear that at present there are insufficient data to recommend with any certainty which group of asthmatics, if any, can be allowed to dive.

It is apparent, therefore, that any recommendations concerning asthmatics will at best be arbitrary and, therefore, rather than trying to justify recommendations that can't be justified, I will present to you the criteria we have adopted at UCSD Medical Center as a starting point for discussion.

First: Asthma is a relative rather than absolute contraindication to diving.

Second: All individuals who have current active asthma are advised not to dive. This includes all individuals with a history of reactive airway disease who have had clinically significant bronchospasm within the last five years, whether or not they take medications and irrespective of the precipitating event.

Third: Any individual who seems to have "outgrown" his asthma and has not had any "bronchospasm," wheezing," or chest "tightness" and has not used any bronchodilator in five years may be a candidate for diving if a complete battery of PFTs are normal. If any question still exists, more complete testing and special tests can be performed.

It is our feeling that, given the current state of knowledge, we would prefer to adopt a policy of conservatism until more data have been collected. On the other hand, it is realized that in an intelligent, mature individual who understands the nature of the risks being undertaken, more latitude in these criteria can be allowed. Hopefully, future research will be undertaken that will address the true risk of asthma to the diver. This is information that can be obtained because, although diving accidents are uncommon, asthma is extremely common and, therefore, data on asthmatic divers should be obtainable. To this end then, I would recommend that a study along the following lines be funded. A large representative list of active divers should be obtained (just obtaining the mailing list of *Skin Diver* magazine is probably not adequate), and a personal health questionnaire sent to them. This questionnaire should focus on asthma and its interrelationship with diving. It should inquire about the clinical history of the diver's asthma syndrome, the severity of the syndrome, medications, etc., as well as the individual's diving history. Such a study, if properly conducted, would hopefully then suggest the numbers of active divers who are asthmatic, which then in turn might help to identify the actual risk faced by asthmatic divers. If there actually are 2-3 million active divers in the United States, and if 3-4% of them are asthmatics, then there are between 60,000-120,000 asthmatic divers. If those estimates are accurate, then we are clearly overconcerned about asthma and diving. On the other hand, if only 10% of asthmatics have the physical capability to dive, and therefore asthmatics make up 0.3% of divers rather than an order of magnitude more, there then may be as few as 6,000 asthmatic divers in the U.S. and "conventional wisdom" may be correct.

#### CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD), CHRONIC BRONCHITIS, AND EMPHYSEMA

The question of diving with COPD is in some ways a much easier problem to deal with than advising the individual who has RAD. The same theoretical arguments that apply to the asthmatic apply to the individual with COPD concerning increased risk for AGE, except that the person with COPD never has airway function return to normal. Thus, the diver or diver candidate with COPD is not just at an increased theoretical risk when an "attack" takes place; rather that individual is at increased theoretical risk at all times.

Fortunately though, by the time individuals with chronic lung disease become symptomatic they are usually incapable of sustaining the exercise necessary to dive, and as a result, it is extremely rare to see a diver who has significant COPD. Additionally, COPD is generally a disease that takes decades of smoking to develop, so it is a disease of older individuals, once again

making these individuals rare in diving medicine. Finally, by the time COPD can be detected clinically, such major physiologic alterations have occurred that I believe there would be little argument that such individuals be advised not to dive. Thus, the question of advising someone with COPD is really the question of advising someone who is asymptomatic but who has abnormal PFTs. In many ways this individual is like the asthmatic we have previously discussed. They are also similar because people with COPD of any severity also may have a component of RAD, their pulmonary obstruction varies with external stimuli, and the obstruction is treated with similar bronchodilating drugs. If this workshop is to be consistent in dealing with these two entities (which I recommend), then individuals with abnormalities of PFTs more than two standard deviations from "normal" should be advised not to dive (see Table 1). Yet the exact definition of "normal" is still unclear, and as a result, individuals with mild disease will overlap many predicted normal values between two standard deviations and the mean (17). As a result, individuals whose isolated values may be "low normal" should undergo more extensive testing if their values may be "low normal" should undergo more extensive testing if their clinical history is suggestive of chronic lung disease.

Reviewing diving accident and fatality statistics, we once again find that it is difficult to ascribe any significant numbers of deaths or accidents to chronic lung disease that could have been detected by a screening physical exam. Thus, as in the case of RAD, there are no data that practically demonstrate the increased theoretical risk of diving with COPD. However, in marked contradistinction to asthma, finding significant numbers of individuals with COPD in divers (group whose major activity requires significant exercise tolerance) is unlikely. This problem is further compounded because, although the asthmatic can identify himself by symptoms, it requires PFTs to identify the individual with early COPD. (As stated previously, once an individual with COPD has symptoms, they are usually not physically capable of diving.) Thus, unlike asthma where data may one day become available, it is unlikely that it will be practically possible to determine what percentage of divers have "mild" or early COPD without an expensive prospective longitudinal study. Furthermore, considering the number of individual's involved, such an expense is probably not warranted.

TABLE 1

*Approximate Lower Limits of Normal at Fifth Percentile Level*

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<u>Parameter</u>	<u>Percent of Predicted</u>
VCBelow	75
FRCBelow	70 or above 130
RVBelow	65 or above 135
TLCBelow	80 or above 120
FEV <sub>1</sub> Below	80
FEV <sub>1</sub> /FVC%Below	85
FEF <sub>25-75</sub> Below	65

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#### SPECIAL TESTING

Large amounts of material could be presented concerning special tests of pulmonary function, however I have elected to be extremely brief in this section as I believe most of these test to be for truly unusual circumstances and most will be used by the pulmonary specialist investigating problem cases. Furthermore, there is considerable disagreement over the utility and validity of some of these procedures.

#### Methacholine or Histamine Challenge

This test has considerable utility evaluating a patient with a questionable history of asthma. Notwithstanding, a limited number of unusual asthmatics unresponsive to methacholine (17), essentially all asthmatics, "consistently show hyperreactivity to nonspecific agents." (2). Thus, in a patient with a clearcut history of asthma, there is no point in bronchial provocation since that individual will have a positive response. Indeed, in such a setting, bronchial provocation testing might even be dangerous. On the other hand, if the history is questionable, if "episodes" are not clearly bronchospastic, then methacholine challenge can be useful in identifying patients with asthma. Methacholine challenge is probably not, however, indicated to make the diagnosis of exercise-induced asthma. In that setting, pre- and postexercise PFTs are more specific and certainly less dangerous. Unfortunately though, methacholine, although extremely sensitive is not specific for asthma. Some patients with a so-called "cough asthma" have normal PFTs, no history of episodic wheezing, but positive methacholine challenge testing. Their sole manifestation of "asthma" is a relatively prolonged period of coughing following URIs (30). Many other individuals with so-called "twitchy airways" have abnormalities of PFT following URIs and can have a positive response to methacholine for as long as two months after the acute infection (39). Thus,

although useful, a positive bronchial provocation challenge is not equivalent to the diagnosis of asthma.

#### Specialized Pulmonary Function Testing

Since there is considerable overlap between "mild disease" and "low normal" functioning in routine pulmonary function testing, on occasion more sophisticated tests may be indicated. In circumstances where divers or candidates have PFTs below the 5% probability of normalcy, such testing is probably not warranted since the diagnosis of COPD is secure: however, if an individual has "low normal" PFTs and has a heavy smoking history or another reason to clinically suspect early COPD, a more complete evaluation of static lung volumes can reveal a pattern of obstructive lung disease. Such evaluation may require helium dilution and body plethysmography and can at times determine whether or not significant noncommunicating air space is present. A discussion of the utility and limitations of these techniques is beyond the scope of this paper.

In summary, I have tried to make recommendations concerning two of the more common pulmonary disorders. These recommendations are basically conservative and in keeping with "conventional wisdom." They are being made not because I necessarily feel they are absolutely correct, but rather because I feel, with very little expenditure of effort and money, important information can be gathered that will allow better recommendations to be made. These recommendations are, I believe, appropriate for the interim. If it was felt that such data would not be forthcoming, I think a more liberal set of recommendations for the asthmatic could be realistically made.

#### REFERENCES

1. Report of the Working Group on Definition of Asthma. In: Porter R, Birch J, eds. Identification of asthma. CIBA Foundation Study Group No. 38, January 13-14, 1971. Edinburgh: Churchill-Livingstone, 1971.
2. Ramsdell J. Asthma: Clinical presentation and diagnosis. In: Bordow RA, Moser KM, eds. Manual of Clinical Problems in Pulmonary Medicine (Second Edition). Boston: Little, Brown & Co., 1985.
3. William H, McNickol KN. Prevalence, natural history and relationship of wheezy bronchitis and asthma in children: An epidemiologic study. Br Med J 1969; 4:321.
4. Rackeman FN, Edwards MC. Asthma in children: A follow-up study of 688 patients after an interval of 20 years. N Engl J Med 1952; 246:815.
5. Derrick ED. The significance of the age of onset of asthma. Med J Aust 1971; 1:1317.
6. Ogilvie AG. Asthma: A study in prognosis of 1000 patients. Thorax 1962; 17:183.

7. Smith J. Survey of rural children with asthma and hay fever. *J Allergy Clin Immun* 1971; 47:28.
8. Pearson RSB. Natural history of asthma. *Acta Allergol* 1958; 12:277.
9. Bernstein IL, Siegal SC, Brandon ML, et al. A controlled study of cromolyn sodium, sponsored by the Drug Committee of the American Academy of Allergy. *J Allergy Clin Immun* 1972; 50:235.
10. Linblad JR, Farr RS. The incidence of positive intradermal reactions and the demonstration of skin sensitizing antibody to extracts of ragweed and dust in humans without history of rhinitis or asthma. *J Allergy Clin Immun* 1961; 32: 392.
11. Deal EC, McFadden ER, Ingram RH, Strauss RH. Heat loss vaporization of water and exercise-induced asthma. *Am Rev Respir Dis* 1978; 117(4)-Part 2:328.
12. Zebellos RJ, Shturman-Ellstein R, McNally JF, Hirsch JE, Souhrada JF. The role of hyperventilation in exercise-induced bronchoconstriction. *Am Rev Respir Dis* 1978; 118:277.
13. Bates D, Macklem P, Christie R, eds. Spasmodic asthma. In: *Respiratory function in disease (Second Edition)*. Philadelphia: W.B. Saunders, 1971.
14. Liebow AA, Stark JE, Vogel J, Schaeffer KE. Intrapulmonary air-trapping in submarine escape training casualties. 1959; *U.S. Armed Forces Med J* 10:265.
15. Schaefer KE, McNulty WP, Carey C, Liebow AA. Mechanisms in development of interstitial emphysema and air embolism on decompression from depth. *J Appl Physiol* 1958; 13:15.
16. Colebatch HJH, Smith MM, Ng CKY. Increased elastic recoil as a determinant of pulmonary barotrauma in divers. *Respir Physiol* 1976; 26:55.
17. Farr RS, Spector SL. Management of the difficult asthmatic. In: Stein M, ed. *New directions in asthma*. Park Ridge, IL American College of Chest Physicians, 1975.
18. Wagner PD, Dantzker DR, Jacoboni VE, Tolmin WC, West JB. Ventilation perfusion inequality in asymptomatic asthma. *Am Rev Respir Dis* 1978; 118:511.
19. Terr AI. Bronchial asthma. In: Baum GL, ed. *Textbook of pulmonary disease (Second Edition)*. Boston: Little, Brown & Co., 1974.
20. Schench HV, McAniff JJ. United States underwater fatality statistics, 1972. U.S. Department of Commerce, NOAA. Washington, D.C. U.S. Government Printing Office Rep No URI-73-8, December 1873.



21. Schench HV, McAniff JJ. United States underwater fatality statistics, 1973. U.S. Department of Commerce, NOAA. Washington, D.C. U.S. Government Printing Office Rep No URI-SSC-75-9, May 1975.
22. Schench HV, McAniff JJ. United States underwater fatality statistics, 1974. U.S. Department of Commerce, NOAA. Washington, D.C. U.S. Government Printing Office Rep No URI-SSR-75-10, April 1976.
23. Schench HV, McAniff JJ. United States underwater fatality statistics, 1975. U.S. Department of Commerce, NOAA. Washington, D.C. U.S. Government Printing Office Rep No URI-SSR-77-11, March 1977.
24. Schench HV, McAniff JJ. United States underwater fatality statistics, 1976. U.S. Department of Commerce, NOAA. Washington, D.C. U.S. Government Printing Office Rep No URI-SSR-78-12, December 1978.
25. McAniff JJ. United States underwater fatality statistics, 1970-78. National Underwater Accident Data Center, University of Rhode Island. Rep No URI-SSR-80-13, September 1980.
26. McAniff JJ. United States underwater fatality statistics, 1970-79. National Underwater Accident Data Center, University of Rhode Island. Rep No URI-SSR-80-14, August 1981.
27. McAniff JJ. United States underwater fatality statistics, 1970-80, including a preliminary assessment of 1981 fatalities. National Underwater Accident Data Center, University of Rhode Island. Rep No URI-SSR-82-15, December 1982.
28. McAniff JJ. United States underwater fatality statistics, 1970-81, including a preliminary assessment of 1982 fatalities. National Underwater Accident Data Center, University of Rhode Island. Rep No URI-SSR-83-16.
29. McAniff JJ. United States underwater fatality statistics, 1970-82, including a preliminary assessment of 1983 fatalities. National Underwater Accident Data Center, University of Rhode Island. Rep No URI-SSR-84-17.
30. Kizer KW. Dysbaric air embolism in Hawaii. Seventh Annual Conference on the Clinical Application of Hyperbaric Oxygen; Memorial Hospital Medical Center, Long Beach, CA, June 9-11, 1982.
31. Kizer KW. Personal communication.
32. Walker D. New Zealand diving-related fatalities, 1981-82. SPUMS 1984; 14(2):12.
33. Fraundorfer RM. Diving fatalities in New Zealand, 1982-85. (Abstract) SPUMS Journal 1985; 15(4):21.

34. Walker D. Provisional report on diving-related fatalities in Australian waters, 1984. SPUMS 1985; 15(3):17.
35. Dick AP, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. Neurology 1985; 35:667.
36. Pilmanis A. Personal communication.
37. Clausen J. Pulmonary function testing. In: Bordow RA, Moser KM, eds. Manual of clinical problems in pulmonary medicine (Second Edition). Boston: Little, Brown & Co., 1985.
38. Corrao WM, Braman SS, Irwin RS. Chronic cough as the sole presenting manifestation of bronchial asthma. N Engl J Med 1979; 300:633.
39. Fedullo P. Chronic cough. In: Bordow RA, Moser KM, eds. Manual of clinical problems in pulmonary medicine (Second Edition). Boston: Little, Brown & Co., 1985.

## PULMONARY CONSIDERATIONS II.

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Pneumothorax  
Trauma  
    Blunt  
    Penetrating  
Sepsis  
Parenchymal Surgery  
Intrathoracic non-lung surgery

Primary spontaneous pneumothorax, because of its unheralded onset, predilection for otherwise fit, young men, high incidence of recurrence, and catastrophic implications at depth, is of major interest to the diving community.

### DEFINITIONS:

*Pneumothorax* (PNX). Air in the pleural space.

*Spontaneous pneumothorax* (SPNX). Air in the pleural space as a result of an intrinsic defect in the lung. It occurs without antecedent trauma or other obvious cause.

*Traumatic pneumothorax* (TRNX). Air in the pleural space as a result of direct or indirect trauma leading to pleural injury.

*Iatrogenic pneumothorax*. Air in the pleural space as a result of an intended or inadvertent consequence of a diagnostic or therapeutic maneuver.

*Primary spontaneous pneumothorax*. A pneumothorax that occurs in an otherwise healthy patient most often the result of a ruptured congenital subpleural bleb. This is the classical pneumothorax involving young, thin, active males and is the most common form of spontaneous pneumothorax. It is usually thought of as a self-limited, benign illness with a low degree of morbidity and mortality but with a high incidence of recurrence. Most of the complications are iatrogenic and related to the methods of treatment.

*Secondary spontaneous pneumothorax*. A pneumothorax as a result of the progression of an underlying lung defect. At one time tuberculosis was the most common cause. Many diseases are associated with pneumothorax; asthma, cystic fibrosis, scleroderma, histiocytosis, tuberous sclerosis, interstitial pneumonitis, xanthomatosis, biliary cirrhosis, Marfan's syndrome, sarcoidosis, idiopathic pulmonary hemosiderosis, pulmonary alveolar proteinosis, pulmonary infarction, rheumatoid disease, hydatid disease, silicosis, berylliosis, and metastatic and primary tumors of the lung. At the present time the most common underlying cause is chronic obstructive pulmonary disease. These

secondary pneumothoraces most often involve a compromised patient and represent a life-threatening episode associated with prolonged hospitalization. Serious septic complications are the rule rather than the exception.

#### INCIDENCE OF RECURRENCE OF SPONTANEOUS PNEUMOTHORAX

Once an individual has had one spontaneous pneumothorax, he tends to have recurrent spontaneous pneumothoraces. Seremetis followed 155 patients for several years and found that 40 percent had a recurrent pneumothorax after tube thoracostomy (1). Larrieu and coworkers followed 63 patients treated with tube thoracostomy for their initial pneumothorax and found that 23 percent had a recurrence within the first year (2). Gobbel et. al. followed 110 patients treated with tube thoracostomy for primary spontaneous pneumothorax and found that 52 percent had an ipsilateral recurrence (3). The average interval between the first and second pneumothorax was 2.3 years. Once a patient had a second pneumothorax, 62 percent had a third pneumothorax; and once a patient had a third pneumothorax, 83 percent had a fourth pneumothorax unless decortication was performed.

Bilateral pneumothorax is seen in 5-7% and synchronous bilateral involvement occurs in about 10% of cases (4).

The rate of recurrence with secondary spontaneous pneumothorax has not been as thoroughly studied but also appears to be relatively high. Dines et. al. reported on 57 patients with secondary spontaneous pneumothorax seen at the Mayo Clinic between 1959 and 1969 (5). When these 57 patients were first evaluated, 22 (39 percent) had already had one pneumothorax over the observation period. The seriousness of the pneumothorax in these patients with underlying lung disease is attested to by the fact that 10 out of 57 (18 percent) died from their pneumothorax. In another series of secondary spontaneous pneumothorax reported by Shields and Oilschlager, eight (16.3 percent) of the patients died with their pneumothorax and in six, the deaths were attributed directly to the pneumothorax (6). (These were VA patients over 40 years of age.) In addition, the pneumothoraces in patients with secondary spontaneous pneumothorax are more difficult to manage because the air leaks tend to be prolonged and multiple tube thoracectomies are frequently required.

The author's interest in spontaneous pneumothorax dates back to medical school and over the years he has been able to collect a series of 164 patients representing 203 episodes (7). The author was the primary physician in only a minority of these cases. The cases were a mix of primary and secondary representing the type of practice. Those coming from a University Hospital or private practice were younger than the VA patients.

#### Seasonal

There was no significant seasonal incidence though there were more during the winter months.

#### Age

The bulk of the patients were under age 40, but there is a second peak in the sixth decade. The range was from 16 to 79.

#### Sex

This is primarily a disease of men. Only 5% of the patients were female. This was somewhat biased by the VA population, as most authors report a range around 10 to 15% of patients being female.

#### Smoking

There were only 7 non-smokers in the series!

#### Extent

Right and left sides were equally involved. Eight patients had severe tension pneumothoraces and four had bilateral collapse.

#### Delay

While most patients were clearly able to time the onset of their collapse because of pain, only 15% were seen within six hours and nearly half waited 48 hours or longer!

#### Activity at Onset

Only 12 episodes were related to vigorous activity and most cases occurred at rest or during mild activity.

#### Past History

10% gave a history of pneumonia

11% had emphysema

3% gave a history of or were found to have tuberculosis.

The most significant pulmonary history by far was that of a previous pneumothorax. Twenty-five percent had a documented previous episode and upon questioning 11 more patients gave a convincing history of a previous episode for a recurrence rate of 30% prior to entering the series. Another 15 patients had a second episode in the series for a total overall recurrence rate of 46%.

#### Clinical Presentation

While most of the patients had little or only moderate distress, eight patients were in severe respiratory distress. Deaths from pneumothorax would often not be detected unless the pathologist is alert to this possibility.

#### Treatment

The treatment was fairly standard. Small collapses, less than 20% in the non-compromised patient, were simply observed. Most significant collapses were treated with tube thoracotomy. Twenty-seven patients came to open thoracotomy, most for multiple previous episodes or failure to expand on initial therapy.

### Complications

The most interesting complications were two cases where thoracotomy had to be done to locate lost, misused, small intracath type catheters that were used in the place of chest tubes. One patient suffered a lacerated lung during placement of a chest tube.

### Deaths

There were nine deaths, four from progression of chronic disease; one from pneumonia; one from cardiac disease; one from cancer; the patient with the lung laceration, and one unfortunate young man who died of staph pneumonia following a bilateral sternal pleurectomy.

Contrary to most situations where multiple procedures are really an admission that there is no good procedure, most of the operations done to prevent recurrence appear to work well. Presently, the most favored approach is that of excision of any blebs and pleural abrasion with dry gauze. Some authors advocate parietal pleurectomy and others are satisfied with simple excision of the blebs. All these procedures require general anesthesia and a thoracotomy. Recently there has been a renewal of interest in obtaining pleurolysis by instilling a chemical or other irritant into the thoracic cavity.

The Veterans Administration sees about 900 spontaneous pneumothoraces each year and is presently carrying out a controlled cooperative study involving thirteen hospitals to evaluate the current popular use of tetracycline as a sclerosing agent. Briefly, only patients requiring tube thoracostomy for spontaneous pneumothorax are considered. In this treatment, 1500 mg of tetracycline are mixed to a volume of 50 cc with saline. This is given only after lung expansion, and is immediately followed by 50 cc of saline. The tube is left clamped for one hour while the patient is maneuvered to facilitate pleural coverage.

The study will have two years of accrual and at least three years of follow-up. Since it is a controlled study, I can make no comment about outcome. So far we have 180 patients entered, the majority have been older men with underlying lung disease. The procedure can be quite painful and careful attention to pain control at the time of instillation is mandatory.

Until the results of the controlled study are completed, I could not consider any form of chemical pleurolysis reason to lift the proscription against diving after a spontaneous pneumothorax.

### Summary - Spontaneous Pneumothorax

Spontaneous pneumothorax is an absolute contraindication to diving. The fact that no recurrences have occurred within 2 to 3 years does not imply that we are dealing with a normal lung.

Surgical correction should greatly reduce the incidence of recurrence but not to a completely normal level, and the contralateral lung should be considered at some, though lesser risk.

Death from spontaneous pneumothorax may not be diagnosed unless the examiner is alert to the possibility.

#### CYSTIC LUNG LESIONS

Bullae, sequestrations, tubercular or fungal cavities, or just about any process that goes on to form a discernible cavity or cyst must be considered a contraindication to diving. While some of the infective process cavities may occur on a background of normal lung tissue, sequestration and emphysematous bullae really are an end stage of an abnormal lung and certainly would be considered to be a contraindication even after surgical removal. Any large cyst, no matter what the etiology, by virtue of its mass effect will lead to distortion of the surrounding pulmonary architecture and would place that lung at risk for air trapping.

#### TRAUMA

Penetrating injuries, i.e., knife or gunshot wounds of the pulmonary parenchyma, are going to heal by scar and as such well may set up areas of potential air trapping. If the injury was significant enough to lead to hospitalization and certainly if there are residual x-ray changes, even a veteran diver should not be cleared without extensive diagnostic and chamber testing and then with the admonition that he will be at greater risk. The neophyte should be rejected.

#### PULMONARY SURGERY

While there are benign lesions that require resections the bulk of surgical resections of pulmonary tissue are done for carcinoma. The development of the stapling device for use in pulmonary surgery has been a great boon. It allows for a leak-free bronchus closure, and now one can go across larger areas of parenchyma without even one small air leak. The previous method of clamping and oversewing a leaking emphysematous lung was as frustrating as it was ineffective. One of the theoretical problems with stapling from our point of view is that it is so easy to go across lung tissue, that anatomical planes may be ignored, and more architectural distortion leading to air trapping may take place. This is an area where further study might be indicated. In any case, anyone undergoing pulmonary resection must be suspect and should only resume diving after the physician has expended our current diagnostic techniques to detect air trapping. The presence of all negative ventilation studies does not remove all risk and that patient should be made aware that he has a theoretical higher risk. The non-diver should be encouraged to remain in that status.

#### NON-PULMONARY SURGICAL PROCEDURES

It is not uncommon that a patient will have a pneumothorax secondary to central line placement, or mediastinal or cardiac surgery where the pleura is violated. Under these circumstances there should be no contraindications on the basis of the pneumothorax. The patient who has a pneumothorax on only positive pressure breathing is another matter, and must be considered to have abnormal lungs. If he is a non-diver he should be discouraged from getting into diving. If he is already an active diver, then he should be warned of the possible increased risk and be made aware of the symptoms and signs of a pneumothorax and the consequence of it occurring at depth. He should have

flow studies for air trapping and good quality chest x-rays looking for potential air trapping areas.

#### HIATUS HERNIA REPAIR

Most repairs of hiatus hernias now are based on reestablishing the physiologic cardio-esophageal sphincter. This is typically done by wrapping a portion of the cardia around the esophagus, effectively making a one-way valve. Patients often complain they can no longer vomit or belch. Theoretically, this could lead to gastric rupture on rapid ascent.

#### SUMMARY

The violated chest will disqualify the beginner, and where exceptions are made for the experienced diver the burden of proof lies with the examining physician.

#### REFERENCES

1. Seremetis MG. The management of spontaneous pneumothorax. Chest 1970; 57:65-68.
2. Larrieu AJ, Tyers FO, Williams EH, et. al. Intrapleural instillation of quinacrine for treatment of recurrent spontaneous pneumothorax. Ann Thorac Surg 1979; 28:146-150.
3. Gobbel WG Jr, Rhea WG Jr, Nelson IA, Daniel RA Jr. Spontaneous pneumothorax. J Thorac Cardiovasc Surg 1963; 46:331-345.
4. De Vries WC, Wolfe WG. Spontaneous pneumothorax. Surg Clin NA 1980; 60(4):851.
5. Dines DE, Clagett OT, Payne WS. Spontaneous pneumothorax in emphysema. Mayo Clin Proc 1970; 45:481-487.
6. Shields TW, Oilschlager GA. Spontaneous pneumothorax in patients 40 years of age and older. Ann Thorac Surg 1968; 5:474-477.
7. O'Hara VS. Spontaneous pneumothorax. Mil Med 1978; 143:32-35.

#### DISCUSSION FOLLOWING PRESENTATION BY O'HARA

LINAWEAVER: Okay. I'd like just to make one comment. In conditions of the pulmonary system, many of the comments pertain to people on dry land. Data from the studies that I did at UCSD, and Dr. Lundgren has done at New York, as well as others, clearly demonstrated that there is a tremendous difference in the lung structure immersed, particularly immersed upright, which is the position that a diver assumes when making an ascent. The gradient between the apices and the bases are grossly magnified and there is a shift of lung volumes from the bases to the apices, and we all know the relationship of



airway diameter with lung volume and therefore conductance. So, when we're talking about passing people or considering people for diving and obstructive airway disease, I think we have to bear in mind that we're talking about their being in an aquatic environment with airway closure, airway attenuation, and therefore, conductance changes. Now, on to the pulmonary aspects. These are extremely important because the presence of extra alveolar air in the arterial circulation is, to me, the greatest nightmare that could happen.

FARMER: The bottom line is it's absolute abject disaster.

LINAWEAVER: That's right. The potential is there. Do we err on the side of conservatism or do we let the guy go and give it the "smoke test?" I think that wheezing while running around the track or playing tennis is one thing. Wheezing at 10 feet, I use that level because they always say "I'm only going to dive shallow," is another.

ARMER: I have to ask this question because the biggest problem I have is my scientific divers objecting to getting any radiation at all. These are scientists knowing nothing about medicine as a rule, generally ill-informed, but who do read what NIH says about x-rays when it comes out in the *Washington Post*.

FARMER: Know just enough to be dangerous.

ARMER: That's right. These people say, "Why should I have any x-rays and if I have x-rays, why should I have two or three?" So the question really is, Do you really find trapped air on full expiratory films? I'm assuming that inspiratory films are done more for soft tissue masses than anything else. Should you do an expiratory film on every diver when he starts? Should you ever repeat it unless there are indications that for some reason the film was post-op or something like that? These are questions that are really important to answer because I get them day after day, Diving Board meeting after Diving Board meeting. You have to justify these things.

LINAWEAVER: Can I give a little perspective on the chest x-ray? The chest x-ray really came to be considered mandatory in the 1950s. As a matter of fact, it was 1955 or 1956 to be exact within a year. It was as a result of the famous Hadley case in the Navy in which a submarine-escape candidate made a buoyant ascent, did everything technically perfect, hit the surface, had a massive cerebral embolism, and died. He was autopsied by A. Liebow, who was Professor of Pathology at Yale, and he found a primary Gohn complex that was very small, probably less than a centimeter in diameter, in the peripheral lung. It happened to be in a bronchiole and acted like a one-way valve. From that time on the Navy insisted that Navy divers and submariners who are going to submarine escape have a normal chest x-ray.

So that's one of the historical things. The other is to detect the presence of bullae, cysts, and blebs. I really defy any of us who are pulmonary physicians on a PA or an expired PA to find the usual kind of small blebs that Vince finds on a young guy who's had a spontaneous pneumothorax that may come to surgery. Some of them are big, but many of them are very

small and are impossible to see. So again here we've taken something that is historical. However, I won't see anybody for a diving physical unless he has a normal chest x-ray. But, I'm one of the "dinosaurs" that Tom alluded to. Very low yield. In the past 10 years I've been out of the Navy working at the clinic, I have turned down one diver on the basis of his chest x-ray. Incidentally, that was a commercial diver who had been diving for 15 years and had a history of asthma. He came in for his annual physical and he didn't have lung tissue above the hilum, just two great big bullae. That's the only diver I turned down on the basis of a chest x-ray.

NEUMAN: And he was diving for 15 years? Safely?

LINAWEAVER: Right.

FARMER: Paul, can I try to defend the dinosaur philosophy? I get very concerned about all this. I mean, here we are being asked as physicians to take responsibility for telling a person that, from a health standpoint, he should dive. If he is a scientific diver, he can do something else in science, that's not the only science he can do. A recreational diver can also do something else to have fun in this world. We're being asked to take that responsibility and yet, we are frequently asked to justify going against basic scientific knowledge and what evidence we do have that says well, it's not worthwhile. But we don't have any evidence that we can get away with not doing certain tests. None whatsoever. We've got zero evidence that I can safely tell a young Navy diver who wants to fight in shallow water that he is not going to get a significant increase in his chance of total deafness or other injuries; vertigo, nausea, vomiting, and drowning, if he dives. And you're asking me to consider giving up being an old dinosaur, so to speak, in this medical-legal climate. I would ask the question "Why should I expose myself to radiation when one of the most eminent diving medicine physicians in the country says "Well, I haven't found but one chest x-ray in how many years that did me any good." To me, the logical, clear answer to this is that if you get air in your arterial system, you'll either die or end up not being able to do the things you want to do in this world. We have no other way to try to prevent that. That is a proven way, even though it may not be useful very often. We don't have the other side of the essential evidence we must have, at least before I'm going to take the responsibility for it.

BOVE: I want to argue about this radiation business with the chest x-ray. It doesn't sound to me as though you have scientific divers. Your divers sound to me like data collectors, because if they're really scientists, they would look at the real issues about what a chest x-ray does. There is no datum that says there's any problem with a chest x-ray. So I don't think that argument is valid.

ARMER: That's not true, because, they know that a chest x-ray gives you approximately 20 millirads and millirads are cumulative over a life time and they know they've got a background exposure of X number per year.

BOVE: We don't have any data on what that does.

ARMER: But, you do have the National Institutes of Health saying "We don't think anybody should get an x-ray unless there's a very good reason to get one."

FARMER: What's a very good reason?

ARMER: My argument is, as a baseline, I want the best chance I have to be able to say "I don't think you have trapped air." That's my argument. I tell them, getting 45 or 50 millirads of radiation is infinitesimal. You fly over Denver, Colorado, at 20,000 feet and you will get more exposure.

BOVE: That's what I'm saying. I don't think the radiation argument about a chest x-ray is valid because by flying in airplanes at 30,000 feet, we get that much a couple of times a year.

ARMER: I can convince them about getting three x-rays as a baseline quite well. They have a hard time arguing with that. It's a lot harder to convince them that they need a single chest x-ray every year.

BOVE: I agree with that. I think, though, it depends on your age population. If you've got 20- and 30-year old people who have essentially a zero chance of developing some specific disease unless they show it clinically, I don't think there's a need to repeat a chest x-ray every three years. I think if somebody has pneumonia and gets better, then you'd better repeat a chest x-ray to make sure things are clear.

ARMER: One of the reasons why I keep asking these questions is that you now have a situation in the scientific diving community where an organization of scientists, the American Academy of Underwater Scientists, publishes a book of standards, safety and medical, for scientific divers. The AAUS then says "if your diving program does this, then we're going to certify your diving program to OSHA as being an exempt program under OSHA's regulations." What we'd really like to say, if you're one of those programs let's say at Scripps or the Smithsonian, is that our divers are now interchangeable because they've all had the same physical exam and the same safety standards. And that's a very good point, because now that is not the case, and divers have to incur large expenses because my exam is tougher than that basic exam.

FARMER: Who are their physicians?

ARMER: That's a very good question. The physicians that have had input are not necessarily diving physicians. One of the things I'd like to see is an organization such as UHMS say "We think that your standards should be of this level," and give physicians the input that they're asking for about specific things, such as how often to take an x-ray, what x-ray, how often to give a cardiogram, what kind of cardiogram? That's what they want to know. It's very important to have someone with knowledge and authority advise them.

BOVE: It's even more important for them not to make rules that they don't have the knowledge and authority to make.

ARMER: Absolutely. But rules are being made out of desperation because they're pressured by OSHA. We really have never responded very well. Maybe we've never been asked, or maybe the right people haven't been asked. When I went to their annual meeting last year, two physicians were there. I was one and a fellow from Hawaii was the other. He is a diving physician and took care of the scientific divers in the University of Hawaii program. These two are the only physician members. They've had nobody to ask. Many of these programs don't have a physician who's really attached to them and also knows about diving. The job is farmed out or the student health doctor does it.

BOVE: The literature on yields for chest x-rays, I think, is starting to become public. The *New England Journal of Medicine* last week, I think, had an article. I don't think it's just completely confined to the diving community. If you want to know yields on chest x-rays, you can look at the general medical literature. I think that kind of information ought to be applied rather than the diving doc saying, "I've never seen anything like that." There's a lot of literature on yields for chest x-rays which I think ought to be applied.

VOROSMARTI: The other problem similar to the chest x-ray is that all the NIH studies deal with the general public, all age groups, everybody else. And their basic study was done on identifying people with lung cancer. It's lousy for identifying people with lung cancer but you see a lot of things on x-rays. And maybe the way to sell the x-ray is to explain that divers are a special group. They are not the general public. You might use the argument, too, that they are going into a special environment that 99.9 percent of the public doesn't involve themselves with.

## ENT CONSIDERATIONS: OTOLARYNGOLOGIC STANDARDS FOR DIVING

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Otolaryngologic standards for diving have often in the past been relegated to secondary considerations with the major emphasis being placed on cardiovascular and pulmonary standards. This is understandable since such emphasis may prevent injuries which are greater threats to survival. In the past three to four decades, with the increasing frequency of sports diving as well as commercial and military diving, injuries to the organs of hearing and balance and the paranasal sinuses have been more frequently encountered. Indeed, it is generally recognized that middle ear barotrauma is currently the most common diving medical problem, and that permanent inner ear injuries with potential life threatening vertigo and vomiting while underwater plus sensorineural deafness are possible in all types of diving. These injuries are largely preventable by the adoption of improved standards for diving fitness as well as the institution of safer diving practices. However, adequate data on which to base the exclusion of any one individual to dive are lacking in many areas. Thus, generally conservative otolaryngologic standards are proposed with the understanding that, as future well-documented diving health and accident data are developed, an adequate basis may be found for relaxation of the standards in some areas; conversely, a strengthening to more conservative and restrictive standards may also be indicated.

Otolaryngologic standards will be considered by discussing the major points in the related medical history and physical examination which may reveal factors that increase the possibility of dysfunction or injuries during diving to the organs of hearing and balance, and the nose and paranasal sinuses. Anomalies and diseases of the larynx and the oral, dental, and maxillofacial structures which may compromise a diver will also be listed. It is assumed in this discussion that the presence of acute disease in any one of these areas at the time of an examination would disqualify a person for diving until the problem has cleared and possibly permanently if any of the contraindications discussed below are present.

### THE ORGANS OF HEARING AND BALANCE

Recommendations of otologic standards will be considered as those designed to prevent injuries to the pinna and external auditory canal, the tympanic membrane and middle ear, and the inner ear.

#### Pinna and External Auditory Canal Standards

A history of pinna frostbite or chondritis may indicate an increased likelihood of pinna injury during cold water exposures and be a contraindication to diving. A history of frequent bouts of otitis externa, seborrhea, and/or eczema of the external auditory canal should be considered in any diving medical history. Such occurrences may indicate a relative to an absolute contraindication for diving. An individual who has rare bouts of otitis externa is likely to be suitable for diving provided proper

precautions and prophylactic use of acidifying agents during swimming and diving exposures are undertaken. Conversely, an individual who has frequent episodes of otitis externa should be advised to seek a specialist's advice and care before undertaking diving. A history or physical finding of significant external auditory canal narrowing (less than one-half of the normal cross-sectional area) due to congenital maldevelopment, osteomas, or other growths would be considered an absolute contraindication for diving unless the problem is corrected. Individuals with narrowing of the external ear canal are more likely to develop otitis externa, external auditory canal barotrauma with possible tympanic membrane rupture during pressure changes or, in the case of unilateral narrowing, caloric vertigo due to unequal entry of cold water into the ears. Cerumen accumulation in the external auditory canal increases the likelihood of otitis externa or canal obstruction and thus may be a relative contraindication for diving; however, cerumen can usually be easily removed and, in some cases prophylactic measures instituted to prevent such accumulation such as avoidance of self-cleansing of the ears with cotton tip applicators or other objects.

The use of ventilated or so-called pressure equalizing earplugs or other devices claiming to prevent external auditory canal and middle ear injuries during diving is not recommended since such devices frequently malfunction and may increase the possibility of barotrauma and canal skin irritation. Also, such devices do not result in the avoidance of moisture exposure which is the leading precipitator of otitis externa.

#### **Tympanic Membrane, Middle Ear, and Facial Nerve Standards**

In general, a previous history of otitis media, ear drainage, tympanic membrane perforation, previous middle ear surgery such as mastoidectomy, tympanoplasty, or tube myringotomies, indicate inadequate Eustachian tubal function in the absence of exposures to significant pressure changes. Such individuals should undergo more detailed scrutiny since they are unlikely to have adequate middle ear pressure equilibration when exposed to the altered pressure changes encountered in diving, and middle ear plus possible inner ear barotrauma are more likely.

#### ***History of Otitis Media***

A history of otitis media and ear drainage within the previous three years should be considered absolute contraindications to diving. Those who suffered these entities greater than three years previously, but not since, may be considered for diving provided no other contraindicating otologic history or current problems are present, and there is a normal otological examination including an intact eardrum with an air containing, clear middle ear space, normal hearing, easy autoinflation of the middle ears on the surface, and the candidate can observe slow descents with middle ear clearing every one to two feet.

#### ***History of Altitude Barotrauma***

Individuals who have difficulty with middle ear pressure equilibration during descent from altitude are also likely to suffer middle ear and possible inner ear barotrauma during diving, and should not be cleared for diving until evaluation by a specialist. They should not be allowed to dive unless they have no other contraindications and have a current normal

otological examination, can easily autoinflate the middle ears on the surface, and observe slow descents.

#### ***History of Previous Ear Surgery***

Individuals who have had a stapedectomy should not dive, since the pressure changes and related displacement of the eardrum, ossicular chain, and stapes prosthesis during diving may result in a perforation of the oval window seal and inner ear injury with probable vertigo, in addition to nerve deafness. Individuals who have had a simple myringoplasty (eardrum repair) may be considered for diving provided there is an intact eardrum, no history of infection or drainage in the past three years plus being able to demonstrate the ability to displace the eardrum with gentle modified Valsalva maneuver. Those who have undergone previous tympanoplasty with ossicular chain reconstruction are at risk for undoing the reconstruction with middle ear barotrauma and should not return to diving. Individuals who have had a radical or modified radical mastoidectomy in which the mastoid complex and external auditory canal are converted into one large cavity should be considered as unsuitable for diving since patients are more likely to have a caloric effect from water entering the ear and usually have a significant conductive hearing loss which cannot be improved by further surgery. A history of previous ear surgery for inner ear disease is an absolute contraindication for diving, as discussed under Inner Ear Standards.

#### ***Current Otologic Findings***

A current finding of a tympanic membrane perforation, a retracted tympanic membrane with or without current middle ear effusion, or a thin, monomeric tympanic membrane all indicate continuing poor Eustachian tubal function in the absence of significant atmospheric pressure changes and should be considered absolute contraindications. The finding of an attic or posterior superior eardrum perforation and/or squamous debris in the middle ear or attic area indicate cholesteatoma, and should be considered a contraindication for the same reason.

#### ***Conductive Hearing Loss***

A conductive hearing loss, in most cases, is also secondary to poor Eustachian tubal function and middle ear inflammatory disease and less often to other causes such as otosclerosis, head trauma, or congenital malformation. Conductive hearing losses due to these latter causes or old, inactive middle ear inflammatory disease are considered by some authorities as suitable for diving provided other aspects of the otologic history and examination are normal. However, I advise that the presence of any significant hearing loss, conductive or sensorineural--as defined below under Audiometric Standards--should preclude diving because of the reasons discussed under Inner Ear Standards. Indeed, such losses frequently are related to continuing inadequate Eustachian tubal function and may not be reversed to normal hearing by surgery.

#### ***Diving Middle Ear Barotrauma***

Divers who suffer middle ear barotrauma during diving without any signs of inner ear injury should not be returned to diving until all otologic symptoms have subsided, the middle ear is cleared, any tympanic membrane perforation has completely healed, and the patient can easily autoinflate at

the surface. This usually requires at least one to two weeks. Symptoms or findings persisting longer than two weeks should be evaluated by a specialist. More cautious descents and appropriate methods of middle ear pressure equilibration, at least every one to two feet of descent, should be recommended. A history of repeated or recurrent episodes of middle ear barotrauma in spite of proper diving practices should alert the physician to the possible causes of chronic inadequate Eustachian tubal function such as chronic nasal and paranasal sinus disease as discussed below, and should be evaluated by a specialist.

#### ***Facial Nerve Paralysis***

A few cases of transient facial nerve paralysis in association with shallow diving have been reported. This phenomenon is felt related to inadequate middle ear pressure equilibration in the presence of a dehiscence of the horizontal portion of the bony facial nerve canal in the middle ear. Other possible causes also exist. A history of facial nerve paralysis related to diving would be an absolute contraindication for future diving. A history of facial nerve damage unrelated to diving may be a relative contraindication depending on the likely cause of the paralysis. For example, a prior history of facial nerve paralysis in association with acute otitis media would indicate a high likelihood of dehiscence of the bony facial canal in the middle ear and an increased possibility of facial nerve paralysis with middle ear barotrauma. This history or the occurrence of facial nerve paralysis due to cholesteatoma, head trauma, or other temporal bone or central nervous system lesions would be a contraindication to diving. On the other hand, a history of one occurrence of true idiopathic facial paralysis (so-called Bell's palsy) would not necessarily preclude diving provided adequate return of facial function to ensure sufficient mouth closure around a mouthpiece is present.

#### **Inner Ear Standards**

##### ***History of Inner Ear Disease***

A history of labyrinthitis, Meniere's disease, nerve deafness, fluctuating hearing, tinnitus, or episodic vertigo indicates inner ear disease and should be considered contraindications for diving. An old, isolated occurrence of such symptoms may be evaluated by a specialist; diving should not be allowed if any abnormalities of hearing or balance function are found.

##### ***History of Inner Ear Related Surgery***

Any person who has undergone surgery for Meniere's disease, such as labyrinthectomy, endolymphatic shunts, vestibular nerve sections, or any ear surgery for dizziness or vertigo, should be disqualified for diving. Such individuals usually have persisting inner ear disease or dysfunction with nerve deafness or a hypofunctioning vestibular endorgan. They may not suffer current vertigo because of central compensation. However, they are at risk for inner ear dysfunction or injury with diving.



### ***Diving Injuries***

A previous history of inner ear injury during diving, such as inner ear barotrauma or labyrinthine window rupture during compression or inner ear decompression sickness, will probably be a contraindication for diving. Return to diving should not be allowed unless normal audiometric and balance function as described below are present as demonstrated by well done audiometry and electronystagmography by a certified specialist. This recommendation, more than any other, has received the greatest number of questions and criticism since it was originally proposed over ten years ago. Thus far, no data has been developed which would indicate that this recommendation should be altered, for available current evidence indicates that an individual with inner ear dysfunction is more likely to suffer inner ear barotrauma, labyrinthine window rupture, or inner ear decompression sickness with diving. The subsequent vertigo, spatial disorientation and/or vomiting while underwater can be life-threatening. Also, individuals with unilateral inner ear dysfunction who injure the healthy inner ear while diving are likely to have significant disequilibrium and hearing disability for non-diving activities and occupations.

### **Audiometric and Electronystagmographic Standards**

The standard physical examination and evaluation of a candidate for sports scuba diving should include a screening air conduction audiogram. Any abnormalities should be further investigated by bone conduction and air conduction plus basic speech audiometry by a certified audiologist using established audiometric equipment and techniques. Confirmed abnormalities should be investigated by a specialist. All candidates for military and commercial diving should undergo complete audiometry by a certified audiologist. Any individual who demonstrates an air and/or bone conduction threshold greater than 20 db ISO in either ear for the speech frequencies, 500 hz to 2000 hz, or a speech discrimination score of less than 90% should be disqualified for diving. Symmetrical hearing loss at frequencies greater than 2000 hz are commonly related to presbycusis and/or previous excessive noise exposure. Individuals exhibiting these losses may be considered for diving; however, they should be advised that such losses represent hair cell loss in the basal cochlear turns and that diving may result in the loss of additional hair cells and hearing impairment in the speech frequencies. Asymmetrical high frequency hearing losses are occasionally seen due to unilateral excessive noise exposure, such as that encountered with rifle or shotgun firing; however, asymmetrical elevation of pure tone thresholds and/or losses of speech discrimination may also indicate other auditory system pathology, such as an acoustic neuroma or unilateral middle or inner ear disease. Such individuals should be evaluated by a specialist before being considered for diving.

Electronystagmography should be obtained in any individual who has a past history of symptoms suggestive either of diving-or non-diving-related inner ear dysfunction. The presence of a significant gaze or positional nystagmus, optokinetic asymmetry, abnormalities of sinusoidal tracking, or unilateral weakness or directional preponderance on Hallpike caloric testing indicate abnormalities of the peripheral or central vestibular systems and are contraindications for diving.

Diving candidates who have no past history indicating inner ear disease, particularly no history of vertigo, either diving-or non-diving-related, and who have no other neurological symptoms or findings that would require further evaluation, do not routinely need to undergo prediving electronystagmography. However, this test is essential with a history of vertigo, since complete destruction of one inner ear in an otherwise healthy individual usually results in severe vertigo and nystagmus which completely disappear within four to six weeks due to central compensation. Thus, the absence of current vertigo and/or nystagmus does not rule out a current or persisting vestibular endorgan abnormality which would definitely be a contraindication for diving.

#### NOSE AND PARANASAL SINUSES

It is important in any diving certification examination to obtain a history of possible nasal or paranasal sinus disease. Frequent episodes of upper respiratory infections, sinusitis, chronic nasal obstruction, discharge, maxillary, dental, or frontal discomfort would indicate a chronic nasal or sinus condition which may predispose to inadequate Eustachian tubal function during diving with subsequent middle and possible inner ear barotrauma and/or paranasal sinus barotrauma. Chronic nasal and/or paranasal sinus disease are usually due to one of several of the primary causes:

1. Allergy--Upper respiratory tract allergies are often associated with lower respiratory tract allergies and possible asthma.
2. Chronic Irritation--usually from smoking or excessive or prolonged use of topical nasal adrenergic agents.
3. Mechanical Obstruction--Commonly from a deformed septum or nasal polyps which are usually due to chronic infection or allergy; less commonly from mass lesions in the nose, sinuses, or nasopharynx.
4. Less commonly encountered primary causes of chronic nasal and sinus disease include vasomotor factors, immune deficiencies, primary infections from syphilis, tuberculosis, rhinoscleroma, mycotic leprosy, biochemical diseases such as mucoviscidosis, microstructural anomalies such as Kartagener's syndrome, other idiopathic causes such as Wegener's granulomatosis, lethal midline granuloma, relapsing polychondritis, lupus, or sarcoidosis.
5. An increasingly common cause of chronic nasal or sinus problems is the intranasal use of illegal drugs such as cocaine.

The presence of any of these underlying factors should be considered relative and possible absolute contraindications for diving depending on the severity and potential for reversal by treatment. Frequent bouts of purulent nasal discharge indicate secondary infection which is usually due to one or several of the above chronic causes and would suggest that the problem is severe. Diving should not be allowed unless the underlying cause is appropriately and successfully treated. Individuals who require systemic or

topical decongestants in order to avoid otologic or paranasal sinus barotrauma during diving should not dive and should be evaluated by a specialist. Diving should not be recommended until the underlying problem is identified and successfully treated.

#### LARYNGOPHARYNX

A history that indicates upper airway obstruction, or laryngeal pathology that results in persistent hoarseness, paroxysms of coughing, laryngospasm, and/or aspiration should be considered a contraindication for diving because of the risk of laryngospasm, upper airway obstruction, or aspiration while under water. An individual who has undergone a partial laryngectomy and has been successfully decannulated with removal of the tracheotomy and healing of the tracheotomy site is at an increased risk for aspiration and should not be recommended for diving. The presence of a tracheostome after a total laryngectomy or an open tracheotomy is an absolute contraindication to diving and swimming. A history or finding on physical examination of a soft, laryngeal-connected upper neck mass which appears with blowing a wind instrument or performing maneuvers which result in increased oropharyngeal and laryngeal pressures would indicate a possible laryngocele. Diving should not be allowed until this is surgically corrected.

#### ORAL, DENTAL, AND MAXILLOFACIAL STANDARDS

Diving candidates who have suffered previous facial fractures involving the mandible, zygomatic-malar area, orbit, and/or the nasal-ethmoid area may be at risk for safe diving even though the fractures have been properly treated and well healed. Such individuals more frequently have nasal or paranasal sinus disease and are more likely to suffer paranasal sinus barotrauma while diving. These fractures frequently heal by fibrous union and may provide routes for gas bubbles to form in the orbit, cranium, and/or facial tissues. Also, the presence of scarring or implanted prosthesis after surgery to correct these injuries or any major oral surgery may increase the risk of local tissue or intravascular gas formation because of altered tissue perfusion. Some authorities may not consider well-healed facial fractures or major oral surgery procedures to be absolute contraindications to diving provided no chronic nasal or paranasal sinus disease is present and appropriate care in mouthpiece and face-mask fitting plus safe diving practices are followed. However, all should realize that the above risks cannot be ignored. Obviously, a patient who is undergoing intermaxillary fixation for the treatment of facial fractures should not undertake diving.

Full or partial dentures or orthodontic appliances may possibly contribute to airway obstruction in a diving emergency. Therefore, dentures and removable orthodontic appliances should not be used during diving. Custom mouthpieces may need to be constructed to allow the diver to properly hold a scuba mouthpiece in the absence of dentures or with fixed orthodontic appliances that cannot be removed.

Diving after dental extractions is contraindicated until complete healing with the absence of soft tissue defects has occurred. The presence of carious or loose or broken teeth is a contraindication to diving until proper dental treatment has taken place. Patients with multiple fillings should undergo twice yearly dental checks to be sure that decay pockets

around the fillings are not occurring which would precipitate dental barotrauma. Persons undergoing root canal treatment should not dive until the treatment is completed and the root canal completely sealed. Individuals with advanced periodontal disease are at risk for difficulties with mouthpiece fitting and gas bubble development under the gum line during ascent.

Persons with temporomandibular joint dysfunction or a tendency toward temporomandibular joint dislocation may suffer a worsening of the problem by using a scuba mouthpiece. Joint dislocation under water could inhibit the individual's ability to properly seal the mouthpiece and create a serious hazard. Diving should not be recommended for individuals with these conditions. Also, individuals with osteomyelitis or osteoradionecrosis of the mandible or patients who are postop major head and neck cancer surgery and/or radiation treatment should not undertake diving since such conditions may increase the chance of tissue emphysema as well as nasal and/or paranasal sinus barotrauma because of the frequent occurrence of poor nasal mucociliary function after radiation therapy.

#### BIBLIOGRAPHY

1. Becker GD, Parell GJ. Medical examination of the sport scuba diver. *Otolaryngol Head Neck Surg* 1983; 91:246-250.
2. Becker GD, Parell GJ. Otolaryngologic aspects of scuba diving. *Otolaryngol Head Neck Surg* 1979; 87:569-572.
3. Becker GD. Recurrent alternobaric facial paralysis resulting from scuba diving. *Laryngoscope* 1983; 93:596.
4. Davis JC. Medical examination of sport scuba divers. Medical Seminars, Inc., San Antonio, TX.
5. Edmonds C, Loury C, Pennfather J. Diving and subaquatic medicine. Diving Medical Centre, NSW Australia.
6. Farmer JC Jr. Diving injuries to the inner ear. *Ann Otol Rhinol Laryngol* 1977; 86:(1).
7. Farmer JC Jr, Thomas WG, Youngblood DG, Bennett PB. Inner ear decompression sickness. *Laryngoscope* 1976; 86:1315-1327.
8. Farmer JC Jr. Otologic and paranasal sinus problems in diving. In: Bennett PB, Elliott DH, eds. *The physiology and medicine of diving and compressed air work*. 3rd Edition. London: Bailliere and Tindall, 1982.
9. Neblett NM. Otolaryngology and sport scuba diving. Updates and guidelines. *Ann Otol Rhinol Laryngol* 1985; 94:(1).

10. Shilling CW, Carlston CB, Matthias RA, eds. The physician's guide to diving medicine. New York: Plenum Publishing Co. 1984.

DISCUSSION FOLLOWING PRESENTATION BY DR. FARMER

ARMER: Question. We, for example, do audiometry every year on all of them. That's not much of a problem since it's a benign test. If you get somebody whose hearing is deteriorating faster than it ought to for his age group and so on, it becomes a real problem what to do. Initially, we've been telling these people we will watch them closely. If it gets worse, I suggest they don't dive. The reason I do so is because of the very rapidly growing cost to the government due to retirement claims for hearing loss from occupational causes. I think the average claim is something like \$40,000 to \$50,000.

FARMER: If you're lucky.

ARMER: If you're lucky. It's talking about a lot of money. In fact, if you look at my senior divers, divers over the age of 40 - 45, with one exception, they all have considerable hearing loss. But, that doesn't seem to bother them until they're ready to retire. So these fellows who were injured in the explosion the other day have already asked their wives to ask me when they can dive again. They both have broken drums. They can't hear anything yet. It's now a week or so after the accident. One of them is president of my diving board.

FARMER: The facts are as we said. You're going to lose hair cells. You start losing hair cells by the age of 18. A normal person doesn't notice it until he's in his 50s or 60s. But you start losing them and if you go to a lot of rock concerts or run a lot of chain saws, shoot a lot of guns and so forth, you're going to lose them faster. Other diseases such as primary ear disease, renal disease, meningitis, encephalitis, etc., may contribute to this. Do these divers have a greater hearing loss than what you would expect for their age group in a nondiving population? If they do, I'd stop them from diving. I don't think they're qualified to dive anymore. Did the hearing loss come from diving? That's when you get into another kettle of fish. Were any of these other diseases present?

ALEXANDER: An examination that is described in a lot of books and in Dr. Linaweaver's chapter, is the visualization of the eardrum moving as some sort of qualification and test. In our experience, mainly with hyperbaric medicine, only in about 10 to 15 percent of people can this be demonstrated and yet most of them, especially older people, have absolutely no problems clearing. In fact, a lot of older people just sleep the whole time on the way down and up and have no problems. Is this a diving test? Should we continue using it as a criterion? What is your feeling on this?

FARMER: Yes, I think you should. I don't think you ought to base absolute disqualification on that one fact alone. It's just like every other test in medicine. It's not perfect and you use it with the results of other tests. There are other ways to try to check the Eustachian tubal function in

addition to the ability to move an eardrum with a Toynbee or modified Valsalva maneuver. You can use a pneumatic otoscope and put a rubber tube around your ear speculum and try to get a decent seal so that you can gently squeeze that bulb and see if you can move the eardrum. This maneuver increases and decreases the external auditory canal pressure. If the drum moves easily, then chances are that 1) there is no abnormal pressure behind it, 2) there is no fluid behind it, and 3) chances are the ossicular chain works pretty well. In fact, one may diagnose ossicular fixation by seeing how well the long process of the malleus moves with pneumatic otoscopy. One can use these tests; if the eardrum moves well, the chances of adequate middle ear pressure equilibration during diving are improved, but such is not assured. If the drum doesn't move, that doesn't necessarily mean the diver should be disqualified, but the chances of middle and possible inner ear barotrauma may be greater.

LINAWEAVER: Of the 50 copies of my chapter that I sent out for comment, I received roughly 16 replies. There were about four who questioned the validity of the specific comment on observing TM movements. If I had worded it some other way, and had established the ability to clear the ears in some objective manner instead of saying "see the tympanic membrane move" I probably would have been all right. But they did jump on that, that there are other ways and so forth. But, now that you mention a nice little pneumotoscope with a good seal and so forth, that is a tool that any physician could use to test for Eustachian tube competence.

## NEUROLOGICAL CONSIDERATIONS I

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### EPILEPSY

Epilepsy affects about one-half of one percent of the general population, perhaps one million people in the United States. Epilepsy most often begins in childhood, and continues indefinitely. A majority of patients with epilepsy are on medication. With good management such patients are able to finish school, compete in the job market, participate in sports, and drive. Some will want to dive.

Patients with uncontrolled seizures are obviously unsuited for diving, driving, or any activity which exposes them to risk should they lose consciousness. A seizure under water carries great hazard for drowning, uncontrolled ascent, and embolism. It exposes other members of the dive party and the rescue party to additional hazard. Should a serious accident not result, the diver's companions will still be faced with a diagnostic dilemma. Conventional doctrine holds that a seizure in water or after surfacing, is presumptive evidence of air embolism. Treatment is obligatory. Parenthetically, this is analogous to the dilemma that occurs when an insulin-dependent diabetic, after diving, has altered consciousness.

The fitness-to-dive decision is less clear-cut in patients with well-controlled epilepsy. Several considerations are important here:

- a. Does diving increase the likelihood of seizures in an epileptic patient?
- b. Should a patient under treatment with anticonvulsant medication dive?
- c. Should a person who has outgrown his epilepsy dive?

Epileptics are said to have a lowered seizure threshold. This means that they are more susceptible to seizures than the general population, and that stimuli which induce seizures in some percentage of the general population will be more likely to do so in those who have epilepsy (1). Examples of such stimuli are prolonged sleep deprivation, alcohol, sedative withdrawal, visual stimulation with rapidly flashing lights, and hyperventilation. The latter is of particular interest to divers. Hyperventilation at atmospheric pressure is routinely used to test for seizure susceptibility (2).

The effect of hyperbaric oxygen on epileptic populations has not been studied. However, it is well established that high partial pressures of oxygen may induce seizures in normal individuals. Navy diving standards require that candidates undergo an oxygen tolerance test in which they breathe pure oxygen for thirty minutes, at a depth equivalent to sixty feet in a chamber. This

test is designed to screen-out those candidates who are susceptible to oxygen toxicity. About one percent of healthy candidates have grand mal seizures under these conditions. The oxygen tolerance test is based on the assumption that all divers will be subject to increased  $PO_2$  in their work, or perhaps in treatment, and that the test will help to eliminate susceptible candidates (3,6). Both assumptions are defensible. It is likely, although not proven, that epileptics have an increased risk of oxygen convulsions.

The well-controlled epileptic leads a fairly normal life. He has no apparent physical handicap, and his illness is not apparent to others (5). In most jurisdictions he can obtain a conditional driver's license if he has been seizure-free for one year, and in almost all jurisdictions, for two years. He cannot get a pilot's license, a commercial driver's license, and will not meet physical requirements for military or commercial diving (4,6).

As a sport diver he may voluntarily put his own life at risk. Should a seizure occur under water, he may drown, or suffer an embolism. Both of these accidents have occurred in non-epileptic patients with oxygen convulsions. These are personal risks. There is yet another. The diver who has trouble under water exposes to risk other members of the diving party, and of the search party. As a student, he imposes an unusual obligation and liability on the instructor. Value judgments must be made here. The law takes notice of the epileptic driver, not because of the risk to the individual, but to the public. The public safety is greatly endangered by an automobile driver who has a seizure. With the diver, the public risk is much less, but not negligible. The law generally accepts that a seizure-free interval of two years, under treatment, constitutes control sufficient to operate a motor vehicle. The risk of recurrent seizures in the controlled subject is nevertheless several times that of the general population. Because the risk of seizures after two years of control is small, public policy recognizes that driving has great social and economic importance, and publicly accepts the risk (7-9).

The controlled epileptic also pays a price in side-effects of medication. Virtually all anti-convulsive medications have some sedative effect. There is great variation among individuals, both with respect to dosage and susceptibility to sedation. The average dose of medication for epilepsy usually produces only mild sedation. It is nevertheless measurable on performance testing. This has a bearing on divers. All sedative medications can be expected to increase the hazard of nitrogen narcosis, in the same manner as does alcohol.

About twenty percent of children with epilepsy outgrow the disorder by age 21. Those who have been seizure-free for five years, without medication, are generally regarded as cured for legal purposes, except for the requirements of flying and special military service. The risk of recurrent seizures in this population is nevertheless considerably greater than random (8,9).



Based on these considerations, I have recommended the following:

- a. The diagnosis of epilepsy, properly established, is disqualifying for military and commercial diving, without exception.
- b. Individuals who have been seizure-free for five years and take no medication have a small statistical risk of recurrent seizures. There is no definite evidence that diving will increase the risk of recurrence. They should be advised to avoid hyperventilation, and cautioned that elevated partial pressures of oxygen may precipitate seizures.
- c. Individuals with controlled epilepsy, taking medication, seizure-free for two years, meeting the requirements of most driving jurisdictions are nevertheless advised not to dive. While driving is important to livelihood, diving is not, and the risk, both to the individual and to his companions, is inconsistent with the pursuit of sport.

#### **CEREBRAL PALSY, PARAPLEGIA, MULTIPLE SCLEROSIS, MUSCULAR DYSTROPHY**

These disparate illnesses are considered here because patients with each diagnosis have learned diving, and certainly many more will do so. It seems to me that the issue here is purely physical stamina, and each case must be considered individually. Cerebral palsy patients, for instance, have an increased incidence of epilepsy. The previous discussion applies here. If they have not had seizures, are adequately conditioned, and can pass the swimming tests, I see no blanket contraindication.

Paraplegic patients require special thought. In traumatic paraplegia from spinal-cord injury, there is at least a theoretically increased risk of spinal-cord decompression sickness. This is the subject of a separate discussion (10).

Muscular dystrophy comes in different sizes and shapes. Those with generalized progressive dystrophy of the Erb-Duchenne type are severely handicapped and surely will not be divers. Some patients with limited disease (limb-girdle, or facio-scapulo-humeral dystrophy) may have sufficient strength to dive effectively. Diving will not make them worse. Patients with myotonic dystrophy are likely to do poorly when chilled.

Multiple sclerosis is a distressingly common disease. Diving and the exposure to an hyperbaric environment probably will not make it worse. Two years ago, there was a brief flurry of interest in the use of hyperbaric oxygen to treat multiple sclerosis. Studies have shown it to have little effect, positive or negative (11). Multiple sclerosis patients, however, have limited stamina, and vigorous physical exercise usually serves them poorly. Most neurologists advise multiple sclerosis patients to avoid exhaustion, and this also means avoidance of chilling.

#### **SPINAL CORD DISEASE**

This deserves special mention. Spinal-cord disease, whether from trauma or demyelination (multiple sclerosis) is evidence of structural abnormality. The individual who undertakes diving with an altered spinal cord

would seem to be a greater risk if he should suffer decompression sickness (10).

#### SPINAL SURGERY

Although the indications for spinal surgery continue to be a matter of dispute, many operations are done. Since the advent of lumbar laminectomy (12) and anterior interbody fusion (13) many thousands of Americans have had one or more of these operations. Many want to dive. There are two considerations, one theoretical and the other practical.

a. An operation which compromises the paravertebral venous plexus might be expected to increase the likelihood of decompression sickness. Such compromise certainly occurs with lumbar laminectomy in which coagulation of bleeders in the venous plexus is routine. Anterior interbody fusion, which approaches the spinal canal without laminectomy, has relatively little bearing on the paravertebral plexus. I think this theoretical consideration may be important, but I know of no instances in which decompression sickness has been attributed to a previous laminectomy (14).

b. Spinal surgery causes spinal disability. Under the best circumstances, a patient recovers from spinal surgery with altered structure. If the operation is entirely successful and he is symptom-free, he nevertheless emerges with a skeleton which is both physiologically and structurally altered. In Workmen's Compensation Appeals Board parlance, in the State of California, a patient who has had such surgery is usually regarded as "disabled for heavy work." This means in the same parlance that he has lost thirty percent of his previous capacity for lifting, pushing, pulling, etc.

Diving is hard work. Such a structural disability certainly is disqualifying for military and commercial divers. Whether a sport diver should be disqualified on this basis depends on how much pulling and hauling he has to do on the surface. He can get someone else to lift his bottles, or dress him out, but he won't necessarily have help to climb the ladder into the boat or to heave himself over the gunwale after surfacing.

#### REFERENCES

1. Schmidt W, Davis. Epilepsy. Philadelphia. p 108-110, 1968.
2. Hill P. EEG. London: McDonald, 1963: 158.
3. Butler FK, Knafele ME. Screening for oxygen tolerance in U.S. Navy divers. Undersea Biomed Res 1986; 13:91-110.
4. Hoeber. Epilepsy: the Law. New York: Harper and Row, Press, 1966: 63.

5. Epilepsy. 8th International Symposium. New York: Raven Press, 1977: 73.
6. USN Diving Manual. Carson, Best Bookbinders 1979: Appendix E-1.
7. Livingston Berman. Participation of epileptic patients in sports. JAMA 1973; 224:236.
8. Emerson D'S. Stopping medication in children with epilepsy. N Eng J Med 1981; 304:1125-9.
9. Thurston JH. Prognosis in childhood epilepsy. N Eng J Med 1982; 306:831-6.
10. Palmer AC. Long-term neuropathological sequelae of decompression. In: Miller JM, ed. Rehabilitation of the paralyzed diver. 30th Undersea Med Soc Workshop. Bethesda, MD: Undersea and Hyperbaric Medical Society 1984: 155.
11. Fischer BH, Marks M, Reich T. Hyperbaric oxygen treatment of multiple sclerosis. N Eng J Med 1983; 308:181-6.
12. Mixter, Barr. Rupture of intervertebral disc. N Eng J Med 1934; 211:210-15.
13. Cloward. The anterior approach for removal of ruptured cervical disc. J Neurosurg 1958; 15:602.
14. Spurling RG. Lesions of the lumbar intervertebral disc. Springfield: Chas C. Thomas, 1953: 148.

#### DISCUSSION FOLLOWING PRESENTATION BY DR. GREER

LINAWEAVER: I'd like to ask a question with regard to the dystrophies. Is the pulmonary apparatus affected significantly in these conditions?

GREER: It may be. In general progressive dystrophy, it is the cause of death. In the limited dystrophies, it may or may not be. It's usually not a limiting factor. I think that decision can be made on the basis of the man's stamina. If he's strong enough to do the work and strong enough to handle the pulmonary load, then I don't think it's necessarily disqualifying. I must say, that issue never crossed my mind until you showed me that letter from Dr. Shilling. This was a fellow who had limb girdle dystrophy and was a scientist who'd been diving for years. He was told he could not dive at a particular site because of his condition.

ALEXANDER: What about scoliosis where one lung is forced to create a much smaller volume than another? It's not pulmonary in the normal sense. Do you think that would increase the risk?

GREER: No, I think it's not pulmonary limiting.

ALEXANDER: I mean, it's not exercise physiology limiting, but it may cause an increased risk with regard to ventilation, you know, over pressure.

NEUMAN: That's a form of restrictive lung disease and I wasn't asked to address the restrictive lung diseases, I think probably because they're not terribly common in divers. I don't think restrictive lung disease of any sort should be disqualifying, to be honest.

BRADLEY: Tom, I don't have problems with that, frankly.

NEUMAN: I'm assuming, of course, that they don't have an obstructive disorder or underlying disease to go with it. I'm just talking about pure, restrictive airway disease.

BOVE: I'd like to see their exercise tolerance. You know, you could put that same person on a treadmill and crank them up to 13 METS and I'll bet you that many times they won't be able to handle that physical workload.

ARMER: What about the epileptic who has had a number of seizures after adolescence who could conceivably be eligible for military duty as a result? His last seizure at age 12 or something like that. I believe they're eligible. You've got somebody say 25 years old in that situation. Would he be ruled out for scientific or commercial diving?

GREER: Yes.

ARMER: He has a normal EEG now.

GREER: Yes, at least I'd rule him out. It's my understanding of the Navy that you are permanently disqualified for epilepsy even if outgrown. The FAA is the same way. I'm talking about Navy diving. It doesn't mean you can't get into the Navy, but you can't be a Navy diver. You can't be a Navy pilot. You can't get an FAA license. In California, you can't get a Class 2 driver's license even if your epilepsy is outgrown. The risk is fairly small if somebody is grown up and five-year seizure free. But it is still higher than random.

MEBANE: What about a history of one or two seizures in a child associated with fever who never has another seizure?

GREER: If they're really febrile seizures in infancy, I think that most people will disregard that. They're still, however, at increased incidence over the whole life.

ARMER: I think you get around that by saying a couple of febrile seizures with a normal EEG would not be epilepsy.

GREER: If they are febrile seizures that occur in infancy. Statistically, they're nevertheless at greater risk. About five percent of infants have febrile seizures and of those five percent, about half will go ahead and will have subsequent seizures and have epilepsy. Whereas the incidence in the general population is 0.5 of one percent.

LINAWEAVER: Practically everyone who has minor and partial seizures, including petit mal and temporal lobe seizures, will from time to time have had a generalized seizure. That's usually what brings it to medical attention. If somebody has temporal lobe seizures and has never lost consciousness, they often will not come to medical attention and most often will not have their activities restricted at all.

NEUMAN: Part of the definition of epilepsy is recurrent seizures. What do you do with the person who's had one seizure earlier in his life?

GREER: Temporize really. I think many neurologists take the position that everybody's entitled to one, that five percent of the population will have a seizure some time during their lives. If they have two, then they have epilepsy. But, if somebody has only had one seizure and if he's been looked at and has no other neurological disease and if his EEG's normal, most people will wait and see.

NEUMAN: So you'd wait for five years in that person?

GREER: No, I don't think I would. When I made my recommendations, I said the diagnosis of epilepsy, properly established. If this man has had one seizure, he can't really be said to have epilepsy. Even in California, where the law is fairly compelling, most people will not report a single seizure to the Department of Motor Vehicles.

LINAWEAVER: I got a lot of comments because of my use of the Air Force criteria of traumatic unconsciousness; that a period of unconsciousness from trauma lasting 24 hours would be disqualifying. That was right out of the Air Force manual that Jeff Davis sent me as a recommendation because they apply it to pilots. Any comments on head trauma and unconsciousness due to trauma?

GREER: Yes, that is a cloudy area. Certainly unconsciousness which is brief and from which people apparently recover all of their faculties within 24 hours is usually regarded as benign. If somebody is unconscious for 24 hours, that is evidence of a pretty severe head injury even without any other neurologic findings, and I can understand the Air Force being concerned about putting those people back to flight duty. I'd be inclined to at least keep them off duty for a long time, perhaps several months. After that period of observation has gone by, however, there probably is no reason to disqualify a person with a head injury with 24 hours of unconsciousness from diving unless he has seizures. If he's going to have seizures as a result of head injury he will almost certainly have had them either at the time of the injury or very shortly thereafter. Short of having had a penetrating head injury or depressed skull fracture, there is not an increased risk of seizures after unconsciousness.

LINAWEAVER: I'd like to get John Hallenbeck now and get Neurology, Part II. Maybe, since Hugh didn't mention it specifically, I could ask you to address migraine in your talk. I disqualified a young girl from becoming a member of the Santa Barbara Sheriff's Dive Team because she had a history of severe, incapacitating migraines with visual disturbance, and was unable to focus and really was unable to function.

## NEUROLOGIC CONSIDERATIONS II

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Dr. Linaweaver asked me to address several issues at this workshop. Three of these issues are neurologic, and they include criteria for returning to diving following decompression sickness that affects the neuraxis, seizures and diving, and finally, diving after a laminectomy.

With respect to the first point, a neurologic deficit which clears completely within 24 hours with or without therapy may be associated with virtually no irreversible nerve cell damage. There is an awareness that in focal brain ischemia, a state of neuronal paralysis can exist for hours during which there is potential for complete recovery if normal blood flow can be restored. This circumstance has been termed the "Ischemic Penumbra" and it is associated with local blood flow rates ranging from about 12-20 ml/100 g/min (normal is about 50-80 ml/100 g/min) (1). This has been described in the gray matter of the brain, but the spinal cord could almost certainly enter a similar state although the relevant blood flows would be lower. Transient ischemic attacks (TIAs) are related to this state, and the defined upper limit for resolution of symptoms and signs in TIAs is 24 hours. On this basis, one might be justified in viewing an individual whose neurologic deficits cleared completely with or without recompression therapy in roughly 24 hours as not very different from normal. A more conservative approach would be to require full recovery during the first therapeutic recompression to consider the patient to be without residual neurologic damage.

The existence of a persistent neurologic deficit raises another issue. There is some evidence that regions of the central nervous system that have suffered some degree of irreversible damage are selectively vulnerable to further damage under circumstances of decreased blood flow. One mechanism for partial recovery in zones of ischemic nerve cell damage involves the reinnervation of synapses on dendrites by collateral sprouting of surviving axons (2). This compensatory mechanism ought to be less effective in subsequent episodes of ischemia as the reserve is depleted. Also, blood flow in zones of ischemic CNS damage may remain at subnormal levels with impairment of the ability of the vascular bed to adjust its resistance to changes in perfusion pressure (autoregulation) and, hence, the zones stand at increased risk during states, which cause further lowering of local blood flow (3). Based on these considerations, it would seem prudent to advise an individual with sequelae from a CNS injury not to resume diving. An individual choosing to dive anyway should be apprised of the increased vulnerability of the affected region to further damage in the course of diving. I think that the information, at least that I have, about this is soft. There are anecdotal accounts. I know David Elliott had an account of somebody who had a nerve that was hit with a lumbar puncture who later had radicular pain in the same nerve distribution and probably most everyone has seen that report. Physicians who

see a lot of cases of diving accidents in the New Orleans area have had the experience that if they allow somebody to go back to diving, it's kind of uncanny how often they'll have a recurrent hit in the same place as their previous decompression sickness involving the neuraxis.

With respect to diving and seizures, it is a generally accepted principle that any neurological condition that materially increases the probability of injury to a diver or to a diver's companion or fellow worker is disqualifying.

It is important to recognize that diving may demand the mental alertness and physical endurance necessary for sustained, heavy exertion without the opportunity to rest. Therefore, good health in a sedentary person is not enough. The prospective diver must be capable of vigorous physical output without becoming weak or exhausted. A seizure disorder is associated with a variable period of impaired or abolished intellectual and/or motor performance that, in the underwater environment, could lead to drowning, pulmonary barotrauma with air embolism, or other forms of injury or death. Although a patient may apply for a driver's license after a period of demonstrated seizure control (varying from state to state). At present there is no formal provision for patients with seizure disorders to qualify for scuba certification on the basis of a seizure-free interval (4). There is, however, controversy on this point and there are some epileptologists who would recommend that patients with well-controlled seizure disorders not be restricted from any activities.

The final issue is that of diving for patients who have had surgical laminectomies. The basis for this concern is that obstruction of the epidural veins within the spinal canal may be an etiologic factor in the development of spinal cord damage due to decompression sickness (5). Following a laminectomy, the anatomy of the epidural veins can be altered and some of the epidural channels may be permanently occluded. This could theoretically predispose to spinal cord damage and decompression sickness. So far as I know, there is insufficient data at this point to make a judgment on this issue. Certainly there are many people diving who have had laminectomies, and I am not aware of a reported series which suggests that there is an unacceptable incidence of decompression-induced spinal cord damage in this group.

#### REFERENCES

1. Astrup J, Siesjo BK, Symon L. Thresholds in cerebral ischemia: the ischemic penumbra. *Stroke* 1981; 12:723-725.
2. Raisman G, Field PM. A quantitative investigation of the development of collateral reinnervation after partial differentiation of the septal nuclei. *Brain Res* 1973; 50:241-264.
3. Fujishima M, Nishimaru K, Omae T. Long-term prognosis for cerebral infarction in relation to brain circulation--a 7 year followup study. *Stroke* 1977; 8:680-683.



4. Hallenbeck JM. Neurological disorders and diving. In: Davis JC, ed. Weekly update. Hyperbaric and undersea medicine. San Antonio, TX: Medical Seminars Inc, 1978.
5. Hallenbeck JM, Andersen JC. Pathogenesis of the decompression disorders. In: Bennett PB, Elliott DH, eds. The physiology and medicine of diving. London: Bailliere Tindall, 1982.

#### DISCUSSION FOLLOWING PRESENTATION BY DR. HALLENBECK

ARMER: You mentioned that there is anecdotal evidence that some divers have had recurrent hits in the same area. Is this true even if they had no residual symptoms or signs?

HALLENBECK: I'm not sure this is written. This is by word of mouth. It would be a very fertile area in which to do a retrospective study. The objective would be to see how often people who have little nicks here and there in their nervous systems have further problems, if they continue to dive.

LINAWEAVER: John, I recall a discussion concerning recurrent pain-only DCS and that in many of these cases the divers were not allowed to go back to diving. I have seen recurrent DCS at sites of previous pain-only DCS, but I don't remember it specifically with neurologic symptoms.

YOUNGBLOOD: Here is an interesting thing along that line which just came up the other day. We're all familiar with A.C. Palmer's work, but I was reading the workshop that came out of the rehabilitation of the injured diver. He elaborated on some of his work there and said something that I don't recall being published in his earlier work. That is that about 40 percent of RNPL goats showed evidence of pain during decompression sickness. On those goats, when they removed the cords later, 25 percent of the X percent who showed the supposed pain-only during decompression sickness had lesions in their cords. That's interesting, bordering upon being disturbing.

ARMER: We recently made a decision about a diver who had a spinal cord hit that disappeared when he went back in the water, just on compressed air. They were in an isolated diving spot. Two years later this fellow, went into a habitat. He didn't tell them about the history of the cord hit, and he spent a week in the habitat without any problem. Clinically, he had no residuals, although at that time he had not been examined by a neurologist except when he first came back from the trip when he had no residuals either. After the habitat tour, he applied to us to dive again and we insisted that he have a fresh neurologic evaluation. He was again normal. I talked to the habitat people and they were horrified when they heard his history. I discussed the case with Mark Bradley and Dr. Shilling and several other people and, if he was normal now and normal about a month after the accident and had appeared to clear completely, could come up with no good medical reason for not letting him dive. I let him dive but restricted him as to depth. But I'm not sure that really would have made much difference. It's a hard decision.

HALLENBECK: I guess the issue would be whether you could set up circumstances or criteria that would help you to clinically determine whether an individual with a prior cord hit had a spinal cord which would be pristine on pathological examination, or would look like the one that Palmer reported.

LINAWEAVER: Is magnetic resonance imaging useful in these cases?

HALLENBECK: I would think that it might be, particularly with a surface coil.

VOROSMARTI: Palmer did have the human spinal cords which showed some deficits. You have to be careful about the goat stories for two reasons. They didn't do neurologicals, some of those goats were 20 years old, were used in decompression experiments, and had multiple decompression sickness over many years. You have to be careful about any of those data. I agree that there is a problem with spinal cords. If you talk about anecdotes where they've had repeated CNS decompression in the same place--I remember a police diver who had decompression sickness three times which caused a weakness of his left leg, and which always cleared up on treatment and never had any residual symptoms; but he'd get back in the water and he'd make a dive and end up with the same lesion again. I don't think you should let those persons dive.

GREER: As far as I know, none of these people have been looked at with MRI up to now. The thing that's been disappointing is that the spinal cord evoked potential studies have not been helpful at all in studying people.

HALLENBECK: I guess the other issue is surgical laminectomies. Hugh talked about that. If you consider epidural venous obstruction to be a major factor in the development of cord-damaging decompression sickness, then individuals with prior laminectomies would theoretically be at increased risk if they had had obliteration of a lot of those venous channels. Once again, if the idea were not to just speculate, but rather to base decisions on data, I think you'd have a hard time. There are a lot of people diving who have had laminectomies. I just haven't seen any studies that really would provide hard information about this. Have other people had experience? I think the one case you mentioned was one that John Miller was very interested in and was going to pursue. I have not seen any series dealing with this.

LINAWEAVER: With the attendant degree of disability possible from a spinal cord infarct, you've got an individual who has had a back injury. He's had a laminectomy and he is 15 percent totally, permanently disabled and that's worth X amount. He's standing there talking to you, walked into your office and the next time you see him, they wheel him in in a wheelchair with a catheter in because he had a laminectomy and it was the one case, like John's case, out of maybe hundreds of people who have done it. That's a pretty significant disability for that individual. I guess you'd take your choice. I know Navy divers who have gone back to dive following a laminectomy, but we all know from Berghage's study that the typical Navy diver is a shallow water diver. Therefore, the statistics are incomplete. Most commercial divers don't go back after a laminectomy because they're precluded from doing heavy work. So we really don't have a data base.

HALLENBECK: That's the problem.

ALEXANDER: Throughout this discussion we get stuck in a double standard. We say that we're going to set certain criteria for people who want to start diving, for those who have done the experiment on themselves and lucked through, and for people who are continuing diving. This is obviously another situation. If somebody comes in with a lot of spinal surgery who's never dived before, I would tell him to jump out of airplanes or something. But, if he's been diving already for 10 years and is scarred up on his back, what are you going to say? He surely has an increased risk. The statistic for the individual is 100 percent. He's not a statistic.

YOUNGBLOOD: This particular thing has already been addressed. As you know, in tunnel work people are eliminated from new hiring because of back problems and a history of back surgery whereas someone who's a senior or a supervisor and one of only 4 or 5 guys in the world have this capacity is readily hired by the companies because both sides are willing to take the risk. But they know the risk.

Can I ask John a couple of neurological questions? One thing that really is of increasing concern to me is whether, in a spinal cord injury, you can have injury, overt clinical signs of spinal cord involvement, an apparent cure or resolution, and then, several months or weeks later a return of symptoms compatible with injuries of the same area? I know that conventional neurological wisdom is that that doesn't happen. But I've seen at least three cases in the last two years and Hugh saw one of them, and I don't think this one was faking. This guy came down two or three months later with painful toxic seizures and several other neurological problems which got worse with time. The most convincing scientific evidence to me that it was real was that it didn't get better after his \$800,000 settlement and continued to get worse.

I was going over some things last week about brain electroactivity in mapping and was very surprised to find that Duffy at Boston Children's had done some studies on people with TIAs that resolved within the time frame that you're talking about. But when you do brain electrical activity mapping afterward, you find focal areas identified of abnormal function where there are no longer any clinical signs or symptoms. This is frightening, but interesting from the standpoint of being able to repeat that kind of study in decompression sickness.

HALLENBECK: With MRI or a good CT, it's quite common to be able to see a persisting lesion in somebody that, even when gone over very carefully, looks clinically normal. So that's a problem in making this decision. You can definitely have somebody who returns to normal and yet has a clear-cut, persistent lesion.

YOUNGBLOOD: There is a famous case that Palmer reported in the *British Medical Journal*, he had the cord of the diver who was killed four years after his decompression incident.

HALLENBECK: He wasn't totally normal.

YOUNGBLOOD: He would have been disqualified by the criteria that we've recommended in New Orleans. But, let's say that he was a sport scuba diver and

had chosen to continue diving. Do you feel that once that level of injury has been established, his injury would stay the same, get better, or as he ages, would he expect further compromise.

HALLENBECK: You're talking about the consequences of further diving?

YOUNGBLOOD: Yes.

HALLENBECK: Not just what happens to the focus over time if he doesn't dive?

YOUNGBLOOD: Yes, well I wonder both.

HALLENBECK: There aren't enough longitudinally followed cases, at least that I know about, that are secondary to decompression sickness to be able to talk about a series. In the more common situation of stroke, the tendency is for improvement which comes up to some plateau. Then, unless there is a recurrence of the stroke, which can be in the same general area and cause an exacerbation of the old problem, the patients don't seem to just slowly deteriorate. But the idea for contraindicating diving is that the lesion would be a region where impaired perfusion might not be adequately compensated for by collateral circulation. The capacity to maintain a consistent perfusion, when there are problems with the head pressure delivering flow to the area, is often not preserved in these cases. Autoregulation is gone and maybe the capacity for reorganization, the plasticity of the CNS parenchyma is reduced the second time around. When an area that has been injured is subjected to a second injury, it is usually much more devastating than the initial injury.

YOUNGBLOOD: One of the things that's emerging, at least from my superficial reading of it, that in some way parallels these strange observations we're seeing is this postpolio syndrome where patients are showing up several years later with a return of similar neurological areas that are affected, and also with some of the vague pains and paresthesias that some of the divers complain of. I wonder whether there might be any parallel there. I know there are half a dozen theories now about the polio victims.

HALLENBECK: There was a recent review of that. Did you see that Hugh? What was the gist of it?

GREER: Well, I think the reason for the recurrent interest is that for years we thought that the reason patients who have had polio and have made a substantial recovery and then deteriorated in later years was that they've had a loss of anterior horn cells during their initial illness, and that those cells have been lost and do not recover. Then, as they get older and experience the natural attrition of more anterior horn cells, their attrition is simply much greater in those areas that were injured by polio. The reason for the recent interest and the recent review was the interest now in slow virus infection and the thought that this might be a persistent virus, that it is behaving like measles. These people who had polio actually have had a smoldering infection going on for years and years. I believe that the conclusion from what's been written recently is that the initial concept was the correct one and that up to now there isn't any evidence of slow virus. Is that the way you understand it, John?

HALLENBECK: I believe the article provided evidence that the progressing deficit develops due to the loss of nerve terminals which had previously proliferated to provide collateral reinnervation to compensate for the anterior horn cell loss.

YOUNGBLOOD: If you follow that thought, what you're saying is that since there are damaged anterior horn cells and that there is a loss of anterior horn cells with aging, therefore what would normally be perceived as the aging affect would be accelerated or occur at an earlier age because it's eating into the reserve, so to speak, some time earlier than would be anticipated.

HALLENBECK: Possibly, but the process would be different from the post-polio syndrome.

GREER: Tony Palmer's explanation for this was interesting and I don't know how far he's gone with it, but we thought about spinal cord damage from decompression sickness in terms of nerve fiber disease, instead of in terms of nerve tract disease. I think that's mostly the way we've all thought about it. But, in that meeting in Alabama, Palmer showed some slides. I think these were his goats in which he saw injury in the root entry zone and he saw ischemic changes in this area which he regarded as an area at the end of the penetrating circulation from the anterior spinal artery and the radial arteries. The reason that he gave for this recurrent "tonic, painful spasm" business, which to my mind is unique, is that some people with spinal cord decompression sickness have what appears to be radicular nerve root injury. That it was injury to nerve cells, not to long tracks. In these cases it appears that a lot of the cells had been lost, and the autoregulation, as John suggested, had been lost. The first one of these I ever saw or ever heard of was three years ago, and I've seen several instances since then.

LINAWEAVER: John, have you ever done consecutive animal studies where you didn't open the dog up but exposed it to repetitive decompression risk, causing spinal cord decompression sickness, then treating it and repeating the exposure to see if the dog is more susceptible?

HALLENBECK: We never did.

LINAWEAVER: Did you ever do a laminectomy on an animal, not do anything, and then use it as a control and expose the animal later?

HALLENBECK: No. They were all acute experiments.

LINAWEAVER: One of the two cases that Hugh and I presented at that paralyzed diver workshop was a classic Hallenbeck preparation. He has his own business and dives regularly. He never had a further problem. But the most incredible one was a professional diver who had a spinal cord lesion, had a prolonged and difficult treatment, and had to be flown up to Panama City. He had a neurogenic bladder for at least six months. He had abnormal signs when I saw him and turned him down for a physical. But, in the interim, he had been diving regularly to 190-200 foot on air, surface decompression on an O<sub>2</sub> profile, on a daily basis without any problems. Once he got over his bladder problem, he was cleared and he dove for seven years without a recurrence of any problem

at all. And he did not get bent at all. So, there at one end you have these cases that infarct in the same place. This diver did incredible, unbelievable decompression without any problem. So, you've got a wide spectrum.

BOVE: There are some interesting things that will be presented at the meeting in Japan on using evoked potentials in man and using MRI. I wonder whether this is becoming the state of practice now. Do you feel that a cord MRI scan ought to be done? It's a fairly useful piece of information, I think. So are the evoked potentials and I wonder if we need to start recommending a different diagnostic modality for cord injuries on these people. Whether we're going to make a commitment on the results about what happens afterward or not I don't know, but it seems to me we ought to be starting to do this kind of thing around the country.

YOUNGBLOOD: If nothing else, that would be a prospective study that would tell us two or three years down the road whether there was deterioration in these cords or not.

HALLENBECK: I think it would be good to recommend that. The MRI is really a sensitive device. At the American Academy of Neurology meetings this year, they had a neuro-imaging course and emphasized MRI. The point was made that this study is so good at visualizing the cord that it's going to make myelography obsolete. Course lecturers claimed that if somebody came to court with a complication of myelography at this point, they would side with the plaintiff, since MRI is at least as good as myelography and, of course, is noninvasive. I think it would be a very good technique to study diving-induced spinal cord problems.

GREER: I'll be interested to hear from the people who say that evoked potentials have been useful for this. I sure don't think they are.

YOUNGBLOOD: They're about 50 percent reliable.

GREER: Yes, about 50 percent.

YOUNGBLOOD: If it's positive, it's positive. But, if it's negative --

GREER: Well, even with the positives, there is a great deal of art in the interpretation, and I really mean art. They're very subjective.

YOUNGBLOOD: But the MRI shouldn't be that way. It's either there or it isn't. Phil Gaines has been getting some lesions in cords with those. Two millimeters resolution on MRI?

FARMER: Two millimeters?

GREER: That's pretty small. It's getting better all the time.

HALLENBECK: I guess the other question was migraine?

HALLENBECK: Decompression can provoke migraine. Migraine responds to hyperbaric oxygen treatment. One of the things that can happen with severe

migraine is nausea which can have a sudden onset particularly during cluster types of migraine headaches. Somebody could find themselves, I suppose, vomiting underwater which would be bad. One can also get fairly disoriented. There are forms of migraine in which a person can pass out. Basilar migraine of Bickerstaff is one type where actual loss of consciousness can occur. With complicated migraine, there could be a period of lack of motor function. In a strong current, loss of limb control would be dangerous.

Migraine could also be confounding. There are migraine equivalents or so-called acephalgic migraine in which some sort of neurologic deficit occurs without the headache. That would be very puzzling if it occurred after a dive. You'd end up recompressing somebody.

LINAWEAVER: Do these tend to present with the same symptomatology each time?

HALLENBECK: Not invariably, but they often do.

FARMER: How does one diagnose acephalgic migraine? Is it a diagnosis of exclusion?

HALLENBECK: Yes, it's easiest in a young person without bad vessels, of course.

YOUNGBLOOD: You've got a nurse and a doctor at Duke both of whom get migraine attacks during decompression any time. I've disqualified a lot of people on that.

LINAWEAVER: They get migraine every time during decompression?

YOUNGBLOOD: Just about every time.

FARMER: Well, I can see the situation. You have a diver who comes up with a true acephalgic migraine and then you take him and put him on Table 6A and that makes his migraine go away. You congratulate yourself for treating another Type II decompression.

HALLENBECK: I think the problem is as Joe says. It can be confounding and I think that if somebody has migraine of sufficient frequency and severity, they'd better think it over pretty carefully before they start diving since it could be dangerous or create confusing situations.

VOROSMARTI: One of the other things I can see as a big problem with migraines is that you have so much pain that you get the old perceptual narrowing thing. The diver doesn't care about himself or his buddy. All he wants to do is get the hell out of there. I think that in addition to the problem you can get a lot of disorientation. That's another big problem. The diver doesn't really care about safety aspects.

LINAWEAVER: Some people really do get disabled or totally incapacitated, like this gal who had been worked up at the clinic before I saw her. That's how I knew she had migraine because it's not part of a normal questionnaire other than mild headaches.

ARMER: I think people with migraines of any significance are not allowed to be pilots. I think probably the same should be true of diving. Certainly commercial or scientific diving. The other problem is that it depends on a person's pattern. Some people will have classical migraine symptoms followed by clearing and then headache. Then the next time they may have one without any headache. There may be varying patterns within one person's migraine. Other people have absolutely identical symptoms time and time and time again. I might feel more comfortable with somebody like that diving than somebody that has variable symptoms. I still think that for any paid type of diving they ought to go to another field. There are certainly many, many migraine patients who are totally disabled during the attack. If they're not totally disabled with the pain that follows, they can certainly be totally disabled during the neurologic phase of it.

GREER: It's a quantity thing. There are an enormous number of people, maybe 60 percent of the world's population, who have some experience with migraine some time during their lives. It's really a question of how bad it is.



## METABOLIC CONSIDERATIONS

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### GENERAL

It is always difficult to generalize when talking about medical conditions. Here, as in any area of occupational and environmental medicine it is necessary to understand the nature of the recreational or working milieu in which the patient will be functioning. Each applicant must be considered individually in relation to his disease, its severity, the status of its control, the presence or absence of adverse reactions, and the applicant's cooperation and understanding of the disease process and the operational environment.

The role of the physician who examines divers to determine fitness to dive is varied. The physician's role in the medical evaluation of a sports diver is mainly that of an expert advisor who informs the patient, and perhaps his family and fellow divers, of the relative risks that any pathological process may pose in the course of diving.

In the commercial diving setting, the physician's role is quite different. The Handicapped Law, the Equal Opportunities Employment Act, and other federal and state legislation allow the examining physician to state whether or not any individual should perform a job. This decision is based on whether the job presents a risk to the individual, should he have a particular medical problem, or whether the individual, because of a particular health problem, would be a risk to his fellow workers. Moreover, most commercial diving companies have stringent physical standards which serve as preconditions for employment, and which allow the examining physician little latitude.

Physical standards for diving should be based on physiological considerations. These may be largely theoretical in nature. Additionally, standards need to be based, where possible, on accumulated experimental and clinical data. Setting standards is serious business, as they can adversely affect peoples' livelihood and dreams. They deserve our best efforts, and the most solid data that we can amass. Patients nowadays are often educated consumers who want well-founded answers; the law and ethical underpinnings of the practice of medicine also demand this of us.

### DIABETES MELLITUS

There are sound physiological and clinical grounds to recommend that individuals with insulin-dependent diabetic mellitus (IDDM) should not dive. Vascular disease, arterial, arteriolar and capillary, is the largest and most intractable disease in clinical diabetes. Unfortunately, this may develop at an early age and despite every effort by both patient and physician to maintain strict control of the diabetic state. The potentially deleterious effects of

this complication of diabetes mellitus on the kinetics of inert gas transfer, resulting in increased susceptibility to decompression sickness, are worrisome.

Recent studies have shown that platelets obtained from diabetic subjects manifest increased platelet aggregation in response to ADP, epinephrine, and collagen. This phenomenon appears to result from increased activity of the platelet prostaglandin synthetase system, resulting in increased production of cyclic endoperoxides or thromboxane A<sub>2</sub> (1). The role of the platelet and that of the prostaglandin system in initiating and participating in the processes of decompression sickness are not completely worked out. There is enough circumstantial evidence though, that these are important contributors to the pathogenesis of this disease. Perturbations in these systems in the diabetic could conceivably predispose to the development of decompression disease.

The adverse effects of insulin reactions in the diabetic patient have constituted the principle basis for advising the insulin dependent diabetic not to dive. Hypoglycemia is the most common complication of insulin treatment in patients with diabetes mellitus (2,3). In the combined studies of Malins (4) and Paz-Guevara, et al., (5), comprising nearly 200 insulin-treated patients, only 7% of the subjects indicated that they had never had a hypoglycemic reaction. In a recent large survey of patients with IDDM (6), approximately 60% of the respondents indicated that they had at least one mild reaction per month; 26% indicated that they had at least one severe reaction within the preceding year, and that this reaction required assistance from another person or hospital attention. A study of 100 patients with IDDM reported that 90% had hypoglycemic reactions, and nearly 55% of these subjects had become comatose during a reaction (7). Thus, hypoglycemic reactions in insulin-treated patients with IDDM are the rule rather than the exception, and severe reactions are quite common.

The risk of hypoglycemia in insulin-treated patients with diabetes mellitus is determined in large part by the balance between the biologic actions of the administered insulin and those of counterregulatory factors, particularly glucagon and epinephrine. This balance is shifted in favor of insulin action and the risk of hypoglycemia is increased if insulin-dependent glucose utilization is increased as it is during exercise. While diving can be a leisurely activity, the necessity for severe exercise is omnipresent. As such, individuals with IDDM should not dive.

Because of the theoretically greater susceptibility to decompression sickness that diabetics may incur, individuals with diabetes mellitus who take oral hypoglycemic agents for control are not qualified to dive, except recreationally under well-controlled circumstances.

Diabetics controlled by diet alone may dive recreationally.

#### AGE

Ever since Pol and Watelle (8) reported the first systematic study of caisson disease in 1854, age has been considered an important factor affecting susceptibility to decompression sickness. Catsaras (9), Snell (10), and von Schrotter (11) reported a higher incidence of compressed air illness in older than in younger workers. This was supported by studies of experimental

decompression sickness reported by Hill and coworkers (12) and Boycott et al. (13). During World War II, extensive observations were made on the incidence of altitude decompression sickness in Air Force trainees, chiefly in the age range from 18 to 28. A total of 52,313 subjects were reported by Gray (14). For a given exposure, the 18-year-old subjects had a relative risk of 23%, the 23-year-old subjects a risk of 50%, and the 28-year-old individuals a risk of 77%.

Paton and Walder (15) have shown that after the age of 40 the bends rate for compressed air workers rises steeply, and that the older the man starting compressed air work, the sooner he will suffer decompression sickness. Summitt, et al. (16) found that the older the diver, the more unlikely it is that he will obtain complete relief from decompression sickness symptoms on recompression, and the more likely that he will suffer a recurrence of symptoms during the subsequent decompression.

All divers over 40 deserve close scrutiny because of the increased incidence of cardiovascular disease in this age group. Because of this, and the strong evidence that the incidence of decompression sickness increases substantially in divers beyond the age of 40-45, one is faced with the necessity of excluding certain individuals from diving. Alternatively, one may consider establishing depth-time restrictions for divers in this age group. For example, one may restrict divers in this age group from undertaking dives requiring decompression. I am loathe to let anyone over the age of 55 dive, although there are certainly exceptions to this edict.

The age limit that the U.S. Navy uses of 45 seems reasonable. An age limit of 45 for commercial diving is appropriate, but provision has to be made for the exceptional individual who exceeds this age. (Moreover, there are legal considerations as the U.S. Navy discovered with its shipyard divers.) For scientific divers beyond the age of 45, consideration should be given to imposing depth-time limits to lessen the severity of decompression stress. The same approach may be best to employ for sports divers.

#### OBESITY

Because of the high solubility of nitrogen in oil and because of the relatively poor vascularity of fatty tissue, it has long been held that obesity increases susceptibility to decompression sickness. Smith reported that "heavy" individuals suffer a higher incidence of decompression sickness than "spare" individuals (17). Boycott and Damant (18) found a higher incidence of fatalities from experimental decompression in guinea pigs and rabbits with high body fat content than low. This was confirmed by work by Gersh and Hawkinson (19), and by experimental studies of lean and obese rats by Philp and Gowdey (20).

Obesity is a difficult characteristic to determine in human subjects. Accordingly, various indices of obesity have been correlated with susceptibility. Gray (14), using data on 48,726 Air Force trainees, reported a relationship between linear density (Weight/Height) and group susceptibility to altitude decompression sickness. He noted that if a particular flight produced 50% decompression sickness in the group with a linear density of 2.3, the risk likelihood of the group with a density of 1.8 was approximately 20%, and the

group with a density of 2.8 was 81%. These general trends in aviators were confirmed by Wehlham et al. (21), and by Swann and Rosenthal (22).

Since weight is a poor index of body fat, fatness in Tyne tunnel workers was estimated by use of skin calipers. Results, reported in the Medical Research Council report (23), indicated a statistically significant relationship between skin-fold thickness and susceptibility to decompression sickness, although this relationship was not significantly precise to be used in predetermining the suitability of an individual for compressed air work.

A recent study by Dembert et al. (24) analyzed health risk factors for development of decompression sickness in U.S. Navy divers. These authors presented compelling evidence that obesity is a contributory factor. Divers with skinfold thickness of 118-172 mm (triceps + abdominal + supra-iliac) were at 10 times greater risk for development of decompression sickness than individuals with thinner skinfold thickness.

An additional important implication of obesity is that it may imply a lack of physical fitness. It therefore is reasonable to restrict individuals who are frankly obese from engaging in diving activities of any sort.

While using body weight as an index of obesity is not the most desirable technique it can provide a first approximation of the degree of obesity. As a general rule, body weight should not be more than 20% of the recommended weights for men and women in the latest Metropolitan Life Insurance Company tables. Use of skinfold calipers for measurement of degree of body fat is more desirable.

#### PREGNANCY

Little information exists about the effect of diving during pregnancy on pregnancy outcome. The information that is available is essentially of three types: retrospective surveys and case reports; a few studies which have looked for teratogenesis in animals subjected to hyperbaric exposures; and studies which have examined bubble formation in fetuses and their mothers resulting from hyperbaric exposure.

Bangasser (25) and Bolton (26) performed retrospective surveys of women who dove while pregnant. While Bangasser reported no effect of diving on pregnancy outcome, Bolton found that women who engaged recreational scuba dives during early pregnancy had a higher incidence of fetal malformations than did women who dived prior to, but not during, pregnancy. However, the actual rate of fetal malformations found among the women who dived during early pregnancy was not higher than that seen in the general female population. (I don't find this observation comforting, since I would expect women who participate in scuba diving to be in better health, more physically fit and of better socioeconomic status than the population at large. As such, they should have a lower incidence of fetal malformations.) Recently, Turner and Unsworth (27) reported a case of a child with multiple congenital abnormalities who was born to a mother who dove extensively during the first trimester.

Studies (28,29) of animals which have received hyperbaric exposures have not shown deleterious effects on fetal development where the exposures were not

extreme. However, recent work by Gilman et al. has shown smaller fetal weights in animals whose mothers dived during pregnancy and who did not develop decompression sickness, than in fetuses of mothers who were dived, developed decompression sickness and were treated. In those animals which develop decompression sickness or where the exposures have been extreme during dives while pregnant, significant fetal abnormalities and stillborn animals have been frequently noted (29).

Studies which have addressed the issue of whether bubbles can occur in the fetus following dives of varying degrees of severity have shown that bubbles can be present in the fetus following dives when the mother has no manifestations of decompression sickness (30). The consequences of having these bubbles in a developing fetal nervous system are worrisome.

No firm conclusions can be drawn from the works cited above. However, there is ample cause in each area for concern, and a conservative approach is advocated. No diving during pregnancy is advocated.

#### REFERENCES

1. Halushka PV, Lurie D, Colwell JH. Increased synthesis of prostaglandin like material by platelets from patients with diabetes mellitus. *N Eng J Med* 1977; 297:1306-10.
2. Feingold K. Hypoglycemia: a pitfall of insulin therapy. *West J Med* 1983; 130:688-95.
3. Wilson DE. Excessive insulin therapy: biochemical effects and clinical repercussions: current concepts of counterregulation in type I diabetes. *Ann Intern Med* 1983; 98:219-27.
4. Malins J ed. *Clinical diabetes mellitus*. London: Erye & Spottiswoode, 1968.
5. Pas-Quevara AT, Hsu TH, White PL. Juvenile diabetes mellitus after 40 years. *Diabetes* 1975; 24:559-65.
6. Goldgewicht C, Slama G, Papoz L, Tchobroutsky G. Hypoglycemic reactions in 172 Type I (insulin-dependent) diabetic patients. *Diabetologia* 1983; 24:95-99.
7. Gin H, Roulet M, Brottier E, Augertin J. Semeiologie et vicu des hypoglycemies chez 100 diabeteques traites par I insuline. *diabetes Metab* 1984; 10:235-59.
8. Pol B, Watelle TJJ. Memorie sur les effets de la compression de l'air appliquee an crecsement des peuts a houille. *Ann Hyg Lang Fr Med Twr* 1854; I:241-279.

9. Catsaras M, Resherches M. Resherches cliniques to experimentales sur les accidents survenant par l'emploi des scaphandres. Paris: Bureaux du Progres Medical, 1890.
10. Snell EH. Compressed-air illness or so-called caisson disease. London: Lewis, 1986:251.
11. Hiller K, Mager W, von Schrotter H. Luftdruckerkrankungen. Wien: Alfred Holder, 1900.
12. Hill L. Caisson sickness and physiology of work in compressed air. London: Edward Arnold, 1912:xi,255.
13. Boycott AE, Damant GCC, Haldane JS. The prevention of compressed air illness. J. Hyg 1908; 8:342-343.
14. Gray JS. Constitutional factors affecting susceptibility to decompression sickness. In: Fulton JF ed. Decompression sickness. London and Philadelphia: Saunders. 1951:Chap 7.
15. Paton WDM, Walder DN. Compressed air illness. Special Report, Med Res Council 281, London: HMSO, 1954.
16. Summitt JK, Berghage TE, Every MG. Review and analysis of cases of decompression sickness occurring under pressure. U.S. Navy Experimental Diving Unit Rep 12--71, 1971.
17. Smith RH. The effects of high atmospheric pressure including caisson disease. Brooklyn: New York and Brooklyn Bridge Co, 1873.
18. Boycott AE, Damant GCC. Experiments on the influence of fatness on susceptibility to caisson disease. J Hyg 8:445-456, 1908.
19. Gersh J, Hawkinson GE. The formation and appearance of tissue and vascular gas bubbles after rapid decompression of guinea pigs from high pressure atmospheres. NMRI, Project x-284, Rep 1, March 1944.
20. Philp RB, Gowdey CW. Experimental analysis of the relation between body fat and susceptibility to decompression sickness. Aerosp Med 1964; 35.4:351-56.
21. Welham W, Blanch Jj, Behnke AR. A procedure for selection of diving and aviation personnel resistant to decompression sickness based on tests in a low pressure chamber. U.S. NRC, C.A.M. Rep 282, 1944.
22. Swann HG, Rosenthal TB. A survey of the incidence of decompression sickness with reference to some constitutional and environmental variants. Kingman Army Air Field, Kingman, AZ, 31st Altitude Training Unit, 1944.
23. Decompression sickness panel. MRC, 1968.

24. Dembert ML, Jekel JF, Mooney LW. Health risk factors for the development of decompression sickness among U.S. Navy divers. Undersea Biomed Res 1984; 11(4):395-406.
25. Bangasser S. Medical profile of the women scuba diver. IQ - 10, Int Conf on Underwater Education, Nov 9-12, 1978.
26. Bolton ME. Scuba diving and fetal well being: a survey of 208 women. Undersea Biomed Res 1980; 7:183-89.
27. Turner G, Unsworth I. Intrauterine bends? 1985; 905. Lancet.
28. Bolton ME, Alamo AL. Visceral malformations, resorptions, and birthweight among fetal rats exposed to air at increased atmospheric pressure. Bacharach AJ, Matzen MM, eds. In: Underwater physiology VII. Proceedings of the seventh symposium on underwater physiology. Bethesda, MD. Undersea Medical Society, 1980.
29. Gilman SC, Greene KM, Bradley ME, Biersner RJ. Fetal development: effects of simulated diving and hyperbaric oxygen treatment. Undersea Biomed Res 1982; 9:297-304.
30. Powell Mr, Smith MT. Fetal and maternal bubbles detected noninvasively in sheep and goats following hyperbaric decompression. Undersea Biomed Res 1985; 12.1:59-67.

#### DISCUSSION FOLLOWING PRESENTATION BY DR. BRADLEY

LINAWEAVER: I'd like to add one other comment to Mark's pregnancy thing that many people don't think about. That is the problem of getting cold and diving. We have cold water in California and it's not unusual to get chilled. Now, if you drop the hemoglobin just a few degrees centigrade, you really hold on to the oxygen. And you figure that the hemoglobin is only 40 percent saturated, plus or minus, in the fetal circulation. A degree of cord temperature drop is a significant amount of oxygen deprivation to that fetus. And so, for that reason, in addition to what Mark mentioned, it's our policy to advise women sport divers, not to dive when they're pregnant because of that additional risk. People in the Caribbean may not have that problem but in California we definitely do.

ARMER: Not true. If you really force our divers to answer the question, they will admit that they don't wear their wetsuits just because of coral cuts. They wear them because they get cold. Even in warm weather. They don't like to admit it. It hurts their macho image.

FARMER: Yancy, what's the consensus among people you come into contact with about pregnant women diving?

MEBANE: Of course we say no. There are so many reasons not to and I can't think of any reason a woman should dive while pregnant. It's a temporary

condition and the chances of problems are far too great. We tell them to wait a few months and the problem will solve itself.

DUEKER: What if they don't know they are pregnant?

MEBANE: We never have recommended interruption of pregnancy and that comes up from time to time. We just say don't dive any more during this pregnancy and have not had reports in that situation of abnormalities developing.

BRADLEY: I had the experience some years ago of having a woman sports diver develop decompression sickness down in the Gulf and who subsequently was treated with a Table 6, and then got low-back x-rays and discovered she was pregnant during this whole thing.

MEBANE: Did they interrupt her?

BRADLEY: That was what I recommended.

ARMER: In that light, considering that particular problem, there's a lot of discussion in private industry about allowing women at child bearing age to be exposed to reproductive hazards regardless of whether they're pregnant or not. They may be pregnant for some time before they know it. I suppose that could be argued for dives as well. I know we're addressing that problem right now with our animal keepers in the zoo who deal with the big cats. The potential of contracting toxoplasmosis is there. We haven't decided whether we're going to let pregnant animal keepers continue working in that area or not.

LINAWEAVER: Haven't the Courts decided that for us?

ARMER: Well, in private industry the Courts have said that industry can say it's not prejudice to forbid child-bearing-age women to expose themselves in certain occupations. There's not been much of it in the government though.

LINAWEAVER: I meant the other way around. The Courts have said that they can not do this.

ARMER: Just recently in Boston, they said that although the cases were going both ways, they seemingly were coming down on the side of industry being able to keep them out of those fields if it was a clear-cut reproductive hazard. It would be impossible for us to take all child-bearing age women and say you can't dive because you might get pregnant. That would be more than half of our scientific divers.

FARMER: There is a fairly quick pregnancy test that's available. I mean, if a sexually active woman is diving very actively, then she may wish to have this test once a month. It costs about \$7.00.

LINAWEAVER: You can buy it at the drug store.



FARMER: No, this is a blood test. That's a good point, Paul, because the one you get at the drug store is a urine test and it's much cheaper. It would be a good screen, but then if that's positive you need to go to that beta subunit of HCG. You get it back in three or four hours.

GREER: Let me try this. I'm not sure this is true, but I guess that the risk in diving versus pregnancy is about the opposite of the risk in drug exposure versus pregnancy in that the risk of teratogenesis from anticonvulsant medicines is not worse in the first part of the first trimester. The risk in diving, I would think, is the risk of oxygen deprivation and in early pregnancy the oxygen requirement of the little tiny implant is not very critical. It becomes increasingly critical as the fetus gets older. I'd guess that the risk is not very great.

LINAWEAVER: Next week there's a special concerns of women in diving workshop. That might be something that you could get to the chairman and ask them to address, is there a difference between the risk early on and in the middle, as the baby gets bigger.

FARMER: I would recommend that in an industrial or scientific situation particularly, that these women who are doing this on a regular basis and who are sexually active ought to do a pregnancy test once a month. At least you may have cut down the amount of exposure time by two thirds.

ARMER: I think maybe we could say, before you go on the trip, we'll check you to see if your pregnant. If we did it routinely to all women before they went on expeditions, we could get away with it. Once they're on the expedition, which might well be several months, it's pretty hard to do because they're usually remote areas without refrigeration, or other facilities. It would be quite difficult. Before they go, you probably could.

NEUMAN: The newer urinary screens for pregnancy are as accurate as blood tests. They detect at the level of between 25 and 50 international units per milliliter. So, they detect pregnancy essentially as it occurs.

FARMER: Our Institutional Review Board will not allow any test to exclude pregnancy for a woman engaging in an experimental subject unless she undergoes a beta subunit of HCG test. That's a blood test.

## GENERAL MEDICAL CONDITIONS

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Diving is a potentially vigorous activity practiced in an alien environment. Considerations of immersion, increased pressure, and isolation affect divers. The topic "general medical conditions" acts as a gathering place for those organ systems not specifically discussed earlier as well as for a general approach to medication and diving.

### VISION

The visual system like the ears and sinuses frequently is less than perfect. Unlike the ears and sinuses, flawed vision usually does not impede diving. Color vision should not be important in either diving or hyperbaric therapy. Standard techniques of visual correction are suitable in hyperbarics. The military imposes strict standards of visual acuity for diving which are not suitable for diving in general. Many divers have refractive errors that do not affect their diving. Others will require correction. It is sometimes possible to wear glasses under a mask. This requires close coordination of glasses style and mask design. Dislodging and fogging also limit this technique. Mask faceplates can be prescription ground or lenses can be bound to faceplates. Contact lenses have become increasingly popular in diving because of their function and convenience. Hard lenses are easily dislodged and have been implicated as sites of bubble formation in deep diving. Soft lenses typically fit more securely and are gas permeable.

The argument can be made that a visually impaired diver will be in trouble if his special mask (or lenses) is lost underwater. No human diver sees well underwater without a mask. Certainly, the corrective lens wearer needs to have a backup set of glasses or lenses available on the surface.

Blind persons can and do dive, but obviously they must do so under very controlled conditions.

### MUSCULOSKELETAL

Problems with the musculoskeletal system outside of neuromuscular illnesses usually affect only mobility. Generally, swimming is a salubrious activity for those with musculoskeletal illnesses.

Speculation is ageless as to increased risk of decompression sickness in injured or surgically corrected joints. I am not aware of any proven association of decompression sickness with these conditions.

Divers with extensive surgical repairs, especially joint replacements, may be limited. This is one area where a distinction is suitable between commercial and recreational diving.

Severe arthritis of the hands may limit ability to operate diving equipment.

A person with aseptic bone necrosis should not dive.

#### GASTROINTESTINAL

Gastrointestinal illnesses are markedly affected by the potential for isolation in diving. As an example, a diver with a peptic ulcer, well controlled with diet, may be in distress on a dive expedition with irregular hours and/or diet. Certainly any gastrointestinal ailment worsened by emotional or physical stress is disqualifying for divers. Life threatening risks of hemorrhage, perforation, or bowel obstruction must be considered in those with gastrointestinal illnesses. Inflammatory diseases associated with severe diarrhea may result in dangerous degrees of fluid loss.

It would be wise to have an inguinal hernia repaired before diving to avoid the potential of incarceration or strangulation.

#### RENAL

Renal failure, regardless of cause, would be disqualifying for unlimited diving. Limited diving may be possible within programs designed for disabled divers.

#### MEDICATIONS

The subject of medications and other drugs in diving is enormous. Indeed, an entire UMS Workshop was devoted to it. Specific drugs have been discussed in relation to appropriate organ systems.

Drugs used to treat illnesses unrelated to diving should be considered for their own properties as well as for the illnesses treated. Divers should not use medications which affect the state of consciousness or which limit exertion. Thus, tranquilizers, sedatives, and stimulants are unsuitable. The question of drug interaction with pressure is moot since these drugs may be dangerous even on the surface. Cardiac medications may limit exertional increases in cardiac output and would be unwise even for divers with mild cardiovascular disease (i.e. uncomplicated hypertension).

Many medications are used for disqualifying illnesses. An example would be anticonvulsants. This may be true for acute as well as chronic illnesses. For example, antibiotics are not a problem unless used for dangerous ailments such as pneumonia.

The non-therapeutic drugs affect most people in the range from caffeine to alcohol and other drugs of abuse. Chronic, excessive use of alcohol is debilitating and implies that diving may be undertaken when under the influences of alcohol. Any other drug abuse should be similarly disqualifying.

Tobacco use should alert the examining physician to the increased possibility of lung and cardiovascular disease.

DISCUSSION FOLLOWING PRESENTATION BY DR. DUEKER

LINAWEAVER: The only reference that I've ever found concerning joint injury or surgery was in Bennett and Elliott. I can't remember the author's name, but I think he was associated with the tunneling industry. He said there was no correlation between the presence of a previous injury and the development of decompression sickness; however, if decompression sickness does occur, it is more apt statistically to occur in that previously injured area. So you take your choice.

FARMER: It doesn't predispose you to developing decompression sickness. But if you violate the tables and you get bent it's more likely at that site.

ARMER: Did you want to comment about intraocular pressure?

DUEKER: I thought about it and mulled it over, but I could not come to a firm conclusion.

BRADLEY: Does anybody have any data about that?

FARMER: I've always heard: Don't dive if you've got it.

ARMER: This is the thing. We do check for increased pressure and we check them because it's so easy to do. I've never had anyone who had glaucoma who was a diver. We've had a few borderline people. We've let them dive. We just checked them every six months instead of annually. I don't know what to do when I get one that has glaucoma.

GREER: Glaucoma is important for all sorts of reasons, but I can't see how diving would have anything to do with it.

BOVE: One issue which frequently arises concerns lens implants. Apparently there are a lot of people that have lens implants and have gone back to diving. The experience, so far, has been that they do all right. There's an old, general belief that lens implants have the propensity to fall out of place if you bumped your head or something. The newer ones, in the last five or six years, are anchored in a different way and they seem to be all right when you dive.

YOUNGBLOOD: How about keratoplasties.

DUEKER: I suspect the reason I didn't mention keratoplasty is that where I work it doesn't have a very good reputation. I think that they are so prone to failure that probably a good face squeeze can easily disrupt the tenuous nature of the keratoplasty.

BOVE: Well, the ophthalmologist will tell you differently because apparently the technique is to cut down through the cornea to the Descemet's membrane, a very tough membrane on the inside of the cornea, and you're supposed to cut down just to that membrane. That membrane provides most of the strength of the cornea. So most ophthalmologists feel that you insignificantly weaken

the cornea by cutting down through the lighter cells down the Descemet's membrane. From the ophthalmologist's standpoint, that would maintain the strength of the cornea. Disruption doesn't sound like a problem. The thing that they do mention is infection because for the first four to six weeks after the radialkeratotomy there are basically open infections during that time. That would be a time not to dive because you could get contaminated water in your eyes.

FARMER: What about glaucoma?

BOVE: There was some interesting concern about mask squeeze with glaucoma, because if you did get a differential pressure across the eye you might get in trouble with it. I don't remember all the details. But, it didn't make a lot of sense to me actually whether that was going to be real or not. For just regular diving, it's not a problem.

LINAWEAVER: I was glad to see that you brought up renal failure.

BOVE: I want to raise a question about that because if you say renal failure, you mean a whole spectrum of things from a creatinine of 1.4 to a creatinine of 7.0. I guess the question is what do you mean? There are a lot of people with low-grade, chronic polynephritis that have been cured and their BUN in 28 and their creatinine is 135.

DUEKER: I was talking about people that need to be dialyzed. That, to me, is failure.

ALEXANDER: Chris, do you want to comment on colostomies?

DUEKER: I wouldn't disqualify them, if that's what you're driving at.

LINAWEAVER: If they can tolerate the aesthetics themselves, if they're well adjusted to the situation, and they've got no other contraindication, I don't see any reason not to encourage them to dive.

DUEKER: I have been asked to comment on motion sickness. I think that medication is one of the biggest areas where motion sickness is a problem and people that have to take medications might be a problem in diving.

Motion sickness, for those of you who haven't had it, is a really serious problem. It's incapacitating to many people. If you do have a person who has motion sickness in the water, even if they don't get to the point of actually having emesis underwater, which is an obvious risk, they get extremely distracted by the motion sickness. In that sense, it's like cold. Even before you lose the ability to work your equipment, you get so focused in on your problem that you're not paying attention to what else is going on. There was a display at the UMS meeting in Long Beach that pointed out that Navy divers were allowed, at the discretion of the Navy diving medical officers, to use the scopolamine patch for preventing motion sickness. I talked to the presenter whose name, unfortunately, I don't remember right now. But, he said that their experience had been good. We

don't have a lot of hard data on this subject. The experience that is accumulating is that it probably works pretty well.

SPAUR: I was just saying I think it's far better to drug people and have them not ill; I think being ill is a far greater danger than the effects you get from scopolamine or phenergan and other drugs.

ARMER: It's quite a problem when you have the psychotic episode in a small boat. We've had one.

FARMER: Overdose?

ARMER: No, I don't believe so, not by history anyway. We give the commoner drugs whenever we can. We actually have about six divers who get no results from dramamine, so they use scopolamine and I give it to them with reluctance and I warn them and I tell the people they are diving with what to be concerned about. One woman had a wild reaction that lasted about five hours. She'd never had anything like that before.

FARMER: Did she try the patch beforehand?

ARMER: I think this was the second day she'd had it on. She had good results with it and she had used it once before with good results.

FARMER: But the second day?

ARMER: The problem, of course, is when you're diving, it gets washed out and doesn't last very long. But they have all figured out that putting clothing around it and plastic over it so that they get a good seal which will make it last several days. They say it lasts three days when they're diving.

MEBANE: Certainly what Andy is saying is not an isolated episode. If you look at the small-boat literature, it's replete with case reports.

YOUNGBLOOD: I'd just like to mention hemoglobinopathy since Chris left it out, because I just finished doing a quick survey for the Nigerian government about this problem. We've run into it in commercial diving areas and it has all sorts of sociopolitical implications. There's been an exchange of letters over the past year about this, often quite heated. It seems that, at least from the recent things that I could find in the aerospace literature and some of the others, for diving where there's not a decompression-related problem that the hemoglobinopathies in general should not be a problem as long as you're speaking of elevated  $PO_2$ 's. Things are complicated by our screening tests, particularly the sickle-dex type tests, which don't differentiate between the disease and the trait and that the trait is probably relatively innocuous, although there's a great deal of debate over percentages of abnormal hemoglobin which may occur in the trait. Theoretically, if you get blockage due to a bubble, in the hypoxic areas downstream from that you may well have sickling occur which will make the decompression sickness refractory or more refractory to treatment, and therefore those with the disease should certainly be excluded. In commercial situations, just like in military situations, logistical problems associated

with treatment or the consequences of having somebody disabled outweigh the equal opportunity benefits of being allowed to dive. My feeling, after reading all that material, is that in sport diving, as long as one understands what the risks might be in the incidence of decompression sickness that it should not be a contraindication.

MEBANE: I asked our hematologist at Duke about this and he said that the person with this disease obviously shouldn't dive, but the guy with the trait, if he did get into trouble and got hypoxic, his central nervous system would be so much more sensitive to the hypoxia than the red cells would be that he'd have nervous system damage long before he'd have a problem with general sickling. The same is true in the localized area of a person with the trait. He'd probably do more damage to other tissues before he really bothered the red cell.  $PO_2$  has to get so low to make the red cells begin to sickle.

LINAWEAVER: One reference that did come up in this JAMA letter-writing exchange was that Lobar clarified his original letter in which he had merely recommended that the relatively few sickle cell carriers, with high concentrations of hemoglobin S, 35 to 45 percent, should avoid potentially hypoxic situations. If we consider decompression sickness as a potential regional hypoxic condition or focally hypoxic, then maybe they should be testing to see if they have this level. That's an expense to somebody.

BOVE: I want to make one other point about medications, which is what I had some experience with these diving doctor courses. You really get a spectrum of the diving population. I had one guy that got severe narcosis at about 80 feet at a time when he was taking cough medicine with codeine and some decongestants, all these sedative agents. We had to drag him out of the water. I think there probably is an interaction in the sport diving level of deaths with sedatives and nitrogen narcosis. That might be one time in this zero-to-five atmosphere range where we could see some drug interaction, too.

GREER: There are a lot of anecdotes about scopolamine. I've seen a lady who got really pretty severely narcotized at 60 feet wearing a scopolamine patch comparable to people that do the same thing taking antihistamines. The thing that puzzles me about it is the unpredictability of it. This lady that you talked about who'd used it before with good results the second day, and then she got psychotic. With such tiny amounts of scopolamine, 0.5 milligrams in the whole thing, you just wouldn't expect that kind of unpredictable behavior. One reason may be that the millipore membrane does not distribute the drug as evenly temporally as it's supposed to, because I have seen the same thing happen. I've seen people use it one trip and get a good result and the next trip have something funny happen. So, it may be that the mechanical behavior of the membrane is not as good as it ought to be.

SPAUR: I would think that it's far better to use phenergan and ephedrine because you take it about a half an hour before and the dose peaks up, and then you're done with it, rather than having a continuing effect.

YOUNGBLOOD: Before we get off scopolamine, can we make sure for the record, and I'm sure we're all aware of this, but the danger of touching the patches and getting medicine in your eye.

LINAWEAVER: You don't even have to touch it. You can have it on the same side and dilate a pupil. You don't have to touch it.

GREER: I don't believe that.

LINAWEAVER: That's what Kizer reported. Dr. Farmer has volunteered to briefly discuss motion sickness.

FARMER: Motion sickness is a serious problem. The reasons for motion sickness are not well understood. The best reason is the neural mismatch theory. During the first year of life, your central nervous system was programmed so that vision, proprioception, and vestibular inputs would fit certain parameters. In most situations, motion sickness occurs when you're on a rocking vessel and vestibular inputs indicate that linear and angular acceleration is occurring; proprioception also indicates that this is happening. But if you're not looking at the vessel or the waves, your visual input doesn't fit. The most classic case is when you're in a cabin of a ship and the ship is moving. The vestibular stimuli says it's moving; proprioception says it's moving, but vision says this is not happening. You're in the cabin and keeping the same relative location in space. That's when motion sickness occurs. Current thinking indicates that the best way to prevent it is not watch the horizon, because then you get a visual mismatch, but to go in the bow of the ship and watch the ship meet the sea. Here you are at the bow of the ship. Here comes a roll and all input indicates that the ship is going to go up and it's going to go down. Hence, no neural mismatch. Once you have motion sickness, it is very difficult to manage. The only way to treat it once it is present is by sedation and, in my view, this is totally incompatible with underwater survival. Unfortunately, most of the "drugs that prevent it" are not, in my view, completely safe to use on or in the water. They are potential problems. The best drug that I have heard about so far that would have less problems is scopolamine, but I'm hearing today that there are problems with scopolamine, also. There are two effective drugs for treating vertigo. One is droperidol. Obviously, you're not going to use this. The other one is Valium, which is also not appropriate for dives. Droperidol and Valium are the only two chemical compounds that have been shown to suppress the symptoms in acutely labyrinthectomized animals.

Here is the diver out on the boat who paid \$2,000 for the diving trip; he's throwing up over the rail and he hasn't gone into the water yet. He's obviously sick, yet he is getting ready to try to get his tank on. Are you going to give him a shot of Valium and let him jump into the water? Obviously not! So you're in a situation where you should advise people that if they are highly susceptible to motion sickness, they ought to take up some other form of recreation besides diving. The same thing with scientific diving and commercial diving. Take up some other occupation. It is true there is great deal of adaptation. Similar to the phenomenon of space motion sickness where the saccule and utricle become angular accelerometers instead



of linear accelerometers. A significant period of adaptation occurs. The astronauts, if they can get through the first two or three days, experience a lessening of the symptoms of space motion sickness. Some of this will occur with divers. But, the problem is that they need to be on a vessel during the time of adaptation. The typical scenario is that divers go out on the dive boat in the morning and become ill with motion sickness. The dive boat returns; they may skip the afternoon dive and try again the next morning. Again they become ill. Maybe, on the last day of a week's diving trip, some adaptation has occurred. I think it is a dangerous situation where the problem itself is potentially dangerous and the methods of treatment plus diving create hazardous conditions.

BOVE: I'm just curious about scopolamine because on these adult diving trips with physicians, a lot of people use the patches, and they don't really relate a lot of problems. I'm not sure what the statistic is. I think a lot of people are using them, and we get reports on the major complications. I'm not sure what the story is in general because I see a lot of people using them and they swear by them. Now, we've always told everybody, put them on at home a month before you go diving and try them for a couple of days and make sure that nothing's going to happen, which seems to be a more rational way to do it than stick it on while you're on the boat and find out you're going to go nuts out in the middle of the ocean.

FARMER: I agree.

BOVE: I think there are some isolated incidences and a lot of them are in small kids. Somebody sticks a patch on 12 year old and he gets an overdose. I'm not sure that the adult population has a high incidence. As I said, I think you're going to hear about the major reactions. It seems to be a pretty useful drug.

GREER: I agree with that entirely. There are occasional anecdotes. I suppose that's going to happen with any drug that has to do with neurotransmitter. But, I think its a real breakthrough.

ARMER: We certainly are using it because for divers who have good reactions to it, it is a miracle; it solves their problem and they seem to have no difficulties. But, that one who reacted badly doesn't dive anymore.

BOVE: One more point. I have been reviewing some work on motion sickness. The best model for motion sickness is to put somebody in a Barany chair and have him nod his head. Within about 10 seconds, he'll be vomiting all over the place. You can do this blindfolded or not blindfolded. The point is that I think some of it is because of two mode motion.

FARMER: That's not like the seasickness and motion sickness we've been talking about; its like the old aviation sickness, the graveyard spinning, guys pile in the plane; there's the runway right there and he's going to make this high speed turn and come in to the runway. His accelerometer is set to accommodate this high speed turn, but, then he turns his head down and he induces an entirely different acceleration force. He actually ends up getting a false sensation and he thinks the plane is going over one way, so

he kicks it in the other direction and he goes into the ground. That is the type of thing when they put them in a spinning chamber and have them put their head down. That introduces a mismatch between the canal and saccular system. That's different from seasickness. In seasickness there is a mismatch between vision and proprioception and overall vestibular input. Here you get a mismatch, you know, I guess you can have some vessels that are moving so violently that when you reach down to pick up something, you're analogous to the pilot of the plane. But, here you have a mismatch between structures within the vestibula and it is not at all clear that this can be used to predict the other one. That remains to be seen.

## CONCLUSION

The consensus standards presented and discussed in this workshop and the reason behind their adoption have been described. It became clear during the discussion that many of the decisions regarding the disqualifying nature of the condition in many instances were based on potential hazards to and the logical outcome of a specific medical condition involving a diver under pressure in the aquatic environment. It was clearly recognized by all attendees that risk data and morbidity and mortality statistics with relationship to health and/or medical conditions in diving are lacking. It is strongly recommended that a registry of diving-related illnesses, and accidents be established, similar in concept to the registry of diving deaths that is currently run by the University of Rhode Island. Such a registry would require funding, standardized submission of information, absolute security from a medical confidentiality point of view, and assurance of long-term organizational status. The present Diving Alert Network has attempted to perform this function but has been somewhat limited in its ability by the lack of reporting, even from network treatment facilities. Compared with other public health matters, diving accident statistics certainly are of low priority because of the relatively low incidence of problems associated with this activity. The low incidence of morbidity and mortality speaks well for the current diving training agencies' good work and, perhaps, to prior and current medical standards that have been established and promulgated from other sources.