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Department of Physiology
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Edited by: Suk Ki Hong

Undersea Medical Society, Inc.
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University of Hawaii

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Edward Beckman
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Suk Ki Hong
Hermann Rahn

PARTICIPANTS

D. Adams
204 Makee Road, Apt. 1104
Honolulu, Hawaii 96815

A. J. Bachrach
Behavioral Sciences Department
Naval Medical Research Institute
Bethesda, Maryland 20014

D. G. Baker
2950 Manoa Road
Honolulu, Hawaii 96822

U. Balldin
Laboratory of Aviation and Naval Physiology
Solvegatan 19, S-223 62
Lund, Sweden

E. L. Beckman
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

P. Bennett
Duke University Medical Center
Durham, North Carolina 27710

R. C. Bornmann
Naval Medical Research and Development Command
National Naval Medical Center
Bethesda, Maryland 20014

C. V. Brown
4795 Somerset Drive
Riverside, California 92507

A. A. Buehlmann
University Hospital
Zuerich, Switzerland

L.-H. Chen
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

J. Craven
Marine Affairs Coordinator
University of Hawaii
Honolulu, Hawaii 96822

J. A. Cromer
Department of Anesthesiology
Duke Medical Center
Durham, North Carolina 27710

J. Dwyer
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

G. Egstrom
University of California, L.A.
MG218
Los Angeles, California 90024

D. H. Elliott
20 Solent Way
Alverstoke, Gosport
Hants, England

L. Fagraeus
Duke University
Durham, North Carolina 27710

V. Frattali
Naval Medical Research Institute
National Naval Medical Center
Bethesda, Maryland 20014

S. M. Gosovic
Naval Medical Institute
Yugoslavia

Y. Gotoh
38 Morohongo
Moroyama, Iruma-gun
Saitama, Japan 350-04

J. M. Hanna
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

C. A. Harvey
Submarine Medical Research Laboratory
Submarine Base
New London, Groton, Conn. 06340

E. Hayashi
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

K. Hayashi
Division of Hyperbaric Medicine
Krushu Rosai Hospital
522-2 Kuzuhara, Kokuraku
Kitakyushu City, Japan 800-02

T. R. Hennessy
University of Cape Town
South Africa

B. A. Hills
Marine Biomedical Institute
University of Texas Medical Branch
Galveston, Texas 77550

S. K. Hong
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

W. L. Hunter, Jr.
709 Chalice Street
Durham, North Carolina 27705

F. W. Ingle
2640 Dole Street #E305
Honolulu, Hawaii 96822

R. Johnson
2177 Aha Niu
Honolulu, Hawaii 96821

M. Kawashima
Division of Hyperbaric Medicine
Kyushu Rosai Hospital
522-2 Kuzuhara, Kokuraku,
Kitakyushu City, Japan 800-02

M. Kitano
Division of Hyperbaric Medicine
Kyushu Rosai Hospital
522-2, Kuzuhara, Kokuraku
Kitakyushu City, Japan 800-02

H. Konata
40 Yokohama City, Japan

F. K. Kurata
636 Ihe Street
Honolulu, Hawaii 96817

G. R. Kusic
Harbor Clearance Unit #1
FPO San Francisco 96601

Y. C. Lin
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

C. E. G. Lundgren
Laboratory of Aviation and Naval Physiology
Solvegatan 19, S-223 62
Lund, Sweden

Y. Mano
1-30-14, Kita-Karasuyama
Setagaya-ku, Tokyo, Japan 157

M. Matsuda
Japan Marine Science and Technical Center
Natsushima-Cho
Yokosuka 237, Japan

J. W. Miller
National Ocean and Atmospheric Administration
Rockville, Maryland 20852

T. O. Moore
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

J. F. Morlock
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

I. Nashimoto
38 Morohongo, Moroyama
Iruma-gun, Saitama, Japan 350-04

H. Nakayama
2-15 Natsushima-cho
Yokosuka 237, Japan

C. Paganelli
Department of Physiology
Biomed. Sci. T608
University of Hawaii
Honolulu, Hawaii 96822

J. Pegg
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

A. H. Purdy
National Institute for Occupational Safety
and Health
Public Health Service
Bethesda, Maryland 20014

F. N. Quandt
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

H. Rahn
Department of Physiology
State University of New York at Buffalo
Buffalo, New York 14214

M. A. Rokitka
580 Eggert Road
Buffalo, New York 14215

M. Singer
177 Belgrave Avenue
San Francisco, California 94117

K. H. Smith
Virginia Mason Research Center
Seattle, Washington 98101

R. M. Smith
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

R. H. Strauss
100 Walker Street
Cambridge, Massachusetts 02138

H. D. Van Liew
Department of Physiology
State University of New York at Buffalo
Buffalo, New York 14214

J. Van Ryzin
P. O. Box 1194
Kailua, Hawaii 96734

D. N. Walder
The University of Newcastle upon Tyne
United Kingdom NE1 4LP

D. G. Watt
943-D 9th Avenue
Honolulu, Hawaii 96816

G. F. Webb
815 Prospect Avenue
Honolulu, Hawaii 96813

P. Webb
Box 308
Yellow Springs, Ohio 45387

G. C. Whittow
Department of Physiology
University of Hawaii
Honolulu, Hawaii 96822

P. E. Winkelmann
3201 Forest Trail
Temple, Texas 76501

D. E. Yount
University of Hawaii
2505 Correa Road
Honolulu, Hawaii 96822

PREFACE

This volume contains papers presented at the International Symposium on Physiology of Man in the Sea, held on July 13-15, 1975, in Honolulu, Hawaii, U.S.A. in conjunction with the Sixth Underwater Physiology Symposium (July 6-10, 1975 at San Diego, California, U.S.A.). The symposium was sponsored by the Department of Physiology, University of Hawaii School of Medicine, and was conducted in cooperation with the Undersea Medical Society. The State of Hawaii Marine Affairs Coordinator's Office provided the major financial support (Task Order No. 50) while the NOAA Sea Grant (04-3-158-29) also contributed financially. I gratefully acknowledge this valuable support, without which this symposium could not have been organized.

The Department of Physiology of the University of Hawaii School of Medicine has been actively involved in research on the Physiology of Man in the Sea since 1968. Generous support provided by the NOAA Sea Grant, the Office of Naval Research, and the State of Hawaii Marine Affairs Coordinator's Office gave us an opportunity to develop a multidisciplinary research program on human performance in the sea. This program, which started out in 1968 with a study on cardiorespiratory functions of Hawaiian SCUBA divers, led to completion of a major dry saturation diving experiment (Hana Kai II) in which five divers spent 17 days at 18.6 ATA in March - April 1975. During these years, we accumulated a wealth of information on the physiology of man under various diving stresses, and it became necessary to share our experiences with other colleagues in the world. Consequently, this symposium was organized to: 1) reassess the University of Hawaii's "Man-in-the-Sea" program in relation to similar programs in other parts of the world; 2) exchange scientific information in areas of our current interest (e.g., underwater exercise, effects of inert gases, saturation diving, decompression sickness); and 3) consider direction of future research on the "Man-in-the-Sea" program. These objectives were met by the excellent program set up by the Local Organizing Committee, and I express my sincere gratitude to all members of the Committee.

The symposium was attended by approximately 60 scientists from Japan, Sweden, Switzerland, South Africa, the United Kingdom, the United States, and Yugoslavia. Since the group was relatively small, there were uninhibited discussions on scientific matters. The Local Organizing Committee expresses its sincere appreciation to all attendees for their active participation in the discussions.

The University of Hawaii Conference Center, under the direction of Mr. Harold Brown, handled all administrative matters excellently in coordination with Mrs. Jane Inouye, the Secretary of the Local Organizing Committee. I am greatly indebted to them for their splendid efforts.

The publication of this volume was made possible through the special effort made by Dr. Charles Shilling on behalf of the Undersea Medical Society, to whom I extend my special thanks. I also express my deep gratitude to Mrs. Shirley Dudziak for her valuable help in editing this volume. The Undersea Medical Society prepared this symposium for final publication. My thanks go to Marthe W. Beckett, technical editor of UMS, and Carla Walsh, who typed all of the final copy.

Suk Ki Hong,
Editor

SESSION I: PERSPECTIVES--MAN-IN-THE-SEA

SESSION I: PERSPECTIVES--MAN-IN-THE-SEA

A. U.S. NAVY PROGRAM: ROBERT C. BORNMANN

The purpose of the Navy's Undersea Biomedical Research Program is to provide the biomedical knowledge necessary for safe deep submergence operations and support of our submarine fleet, and to be able to salvage, reconnoiter, or build safely anywhere on the continental shelf (defined as underwater down to 850 fsw). The program output is software; no equipment is produced, although the information generated is applied to biomedical design criteria for effective diving equipment and to the development of safe, efficient operational procedures.

Funding for the program is five million dollars annually, 60% for Advanced Development, and 20% each for Exploratory Development and Basic Research. Of that total, approximately 47% is expended in Naval Medical Research and Development Command laboratories, principally at the Naval Medical Research Institute in Bethesda, Maryland, and at the Naval Submarine Medical Research Laboratory in Groton, Connecticut. About 8% goes to other Navy laboratories and activities and 45% goes to civilian research through Office of Naval Research (ONR) contracts. Coordination with ONR is close, and an active liaison is maintained in concept development and program planning, including collaborative funding of some contracts, and contract monitoring and review.

Potential customers are represented by 3,000 Navy divers of all types and 25,000 submarine personnel. Naval manned underwater operations can be divided into Underwater Demolition Teams (UDT) and SEAL units, Explosive Ordnance Disposal (EOD) specialists, Ship Repair and Ship Salvage groups, and deep-diving teams prepared to search for and retrieve objects lost underwater and to do deep salvage of sunken airplanes and small ships. The Seabees have units working on underwater construction. Submarine fleet operations have taken on an increasing strategic importance in the last decade, and an ancillary program for submarine personnel rescue or individual submarine escape has been maintained. There is also a small naval group involved in the increasingly important operation of small submersibles.

Saturation diving, in which divers remain under pressure for days or weeks, has made extremely deep diving operations safe and practicable. In the typical saturation deep-diving operation, the divers live in a Deck Decompression Chamber (DDC) on board a surface vessel. The DDC is compressed with diving gas, generally a mixture of helium and oxygen, to a pressure equivalent to that of the water depth at which the divers are working. At the beginning of a work period, a team of divers enters a Personnel Transfer Capsule (PTC) which is "mated" to the DDC and pressurized to an equal gas pressure. The two hatches or doors which make independently pressurized units

of the PTC and DDC are closed. The connecting tunnel is depressurized, the massive clamp holding the two together is released, and the PTC, with its diver occupants, is lifted by a crane and placed in the water.

The PTC is lowered through the water until a depth is reached where the water pressure outside equals the gas pressure inside the PTC. At this point the outside hatch of the PTC can again be opened, and the divers can exit into the water to begin their work. At the completion of their shift, the divers re-enter the PTC and seal the hatch. The PTC is lifted to the surface and out of the water and repositioned over the DDC mating skirt. The clamp locks the PTC securely onto the DDC, and the small connecting tunnel is again pressurized. Minor adjustments are made to equalize the pressures in the three vessels precisely, and the separating hatches are again opened. The divers climb down into the DDC to eat, refresh themselves, or rest until their next work shift. The major decompression ascent to the surface, which takes many days, is deferred; all the divers are decompressed at one time in the DDC.

Such saturation Deep Diving Systems (DDS) are installed on the USS ELK RIVER (IX501), a diving support vessel in San Diego, on the USS PIGEON (ASR 21), and the USS ORTOLAN (ASR 22), two submarine rescue vessels in San Diego and in Norfolk, and on a diving barge of the Atlantic Fleet Support Force in Norfolk. A world record dive at sea was made to 1143 fsw near Panama City, Florida, with this equipment in 1975.

The gas atmosphere within the DDS is an artificial one and must be continually adjusted. Oxygen pressures are generally maintained mechanically at a partial pressure close to that which exists normally in air at the surface. Carbon dioxide produced by the occupants must be removed. The increased pressure is produced by compressing metabolically active gases with an inert diluent, such as helium or nitrogen. Understanding the potential biomedical problems within the DDS would include a knowledge of the effects on the human organism of a range of oxygen and carbon dioxide pressures, the effect of pressure per se, as well as the effects of nitrogen or helium, which are not truly inert at high pressure. Contaminants such as carbon monoxide and hydrocarbon gases must be identified and their potential toxicity understood.

The presence of inert gas in the diving gas mixture necessitates a controlled ascent or decompression to prevent the occurrence of decompression sickness. This research program deals not with decompression schedules or tables, which must be linked to the equipment used for any dive, but with the fundamental principles of inert gas transfer within the body which are necessary for the formulation of such schedules. Decompression sickness does occur in diving, and it can be extremely serious; high priority in our program is given to developing better methods of therapy for that injury.

The purpose of underwater operations is to perform work, and there is little purpose in risking a diver in the water unless we can maintain his human faculties of vision, hearing, manual dexterity, and good judgment to a large extent. The focus of our effort in human engineering and performance research is directed to this end.

Cold is pervasive and potentially crippling in the deep ocean, and biomedical aspects of cold have been an important element in designing protective diving equipment for deep diving. As our naval operations move into the Arctic and under the polar ice, cold protection will become more important in shallow diving. Aseptic bone necrosis has been a major occupational injury in tunnelling and compressed air work for some time. More recently it has been discovered that such changes can be noted on bone radiographs from divers as well. A recent Navy survey has shown that asymptomatic radiographic changes can be found in the bones of less than 3% of Navy diving personnel. Severe necrosis can cause painful and crippling arthritic deformation, however, and we have as yet no understanding of how the injury is precipitated or of its natural progression. We hope to find the answer through research.

When a diver is placed in the cramped confines of his DDC, he relies on the Navy to maintain him and to deliver him safely. The Navy medical department is responsible for the treatment of any injury or illness he may suffer within the DDC. The requirement for decompression makes an emergency evacuation almost impossible. Limitations in visualization and communication may make diagnosis, even of simple problems, very difficult. Even with a corpsman or a medical officer pressurized in the chamber, there are logistic problems in obtaining necessary diagnostic and treatment instruments, and there is some question about the safety or adequacy of common medications under high-pressure conditions.

The Navy has only recently begun construction of new high-pressure research and test facilities to support the diving program. The Experimental Diving Unit has been moved to the grounds of the Coastal Systems Laboratory in Panama City, Florida, to take over the operation of the new Ocean Simulation Facility there. That chamber complex has a pressure capability of 2250 fsw and an enormous wet chamber, large enough to maneuver a small submersible in. Panama City will become the center for the development and testing of diving equipment for the Navy.

Now taking form in Bethesda is the building which will house the Environmental Health Effects Laboratory (EHEL) of the Naval Medical Research Institute (Fig. IA-1). Four chambers of that pressure facility, including a wet pot, will be capable of pressures to 2300 fsw and two additional connecting chambers will be rated to pressures of 3400 fsw. Operations are scheduled to begin in EHEL in calendar 1978.

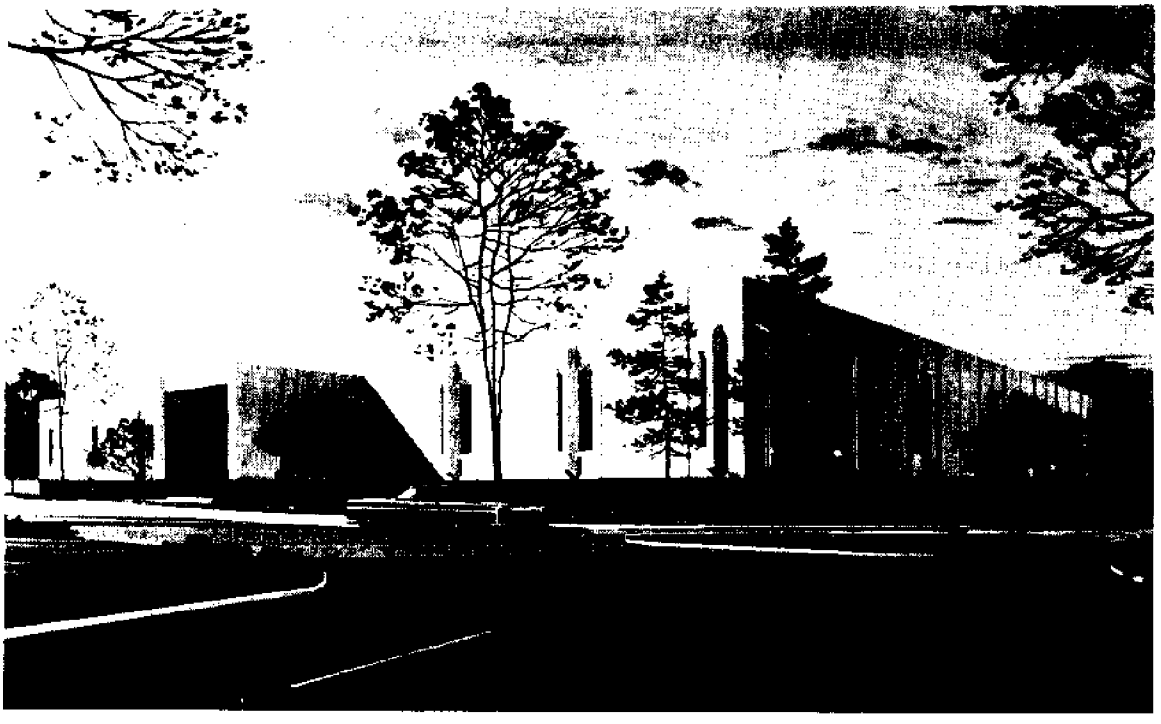

Fig. IA-1. Architect's drawing of Environmental Health Effects Laboratory at Naval Medical Research Institute in Bethesda, Maryland

There are two unique maneuvers which span the gap between normobaric submarine operations and hyperbaric diving operations. They are individual escape from a submerged submarine, and crew rescue from that submarine using a McCann Chamber of a Deep Submergence Rescue Vehicle (DSRV).

In submarine operations, the interior of the pressure hull is kept at near normal (1-ATA) pressure, and hatches are constructed to seal with pressure from the outside. It is possible (extraordinarily) for a passenger to open an external hatch if the interior pressure, usually in a specially constructed small rescue trunk, is raised to equal the external water pressure. In submarine ascent the individual is provided with a buoyancy vest to lift him rapidly to the surface. A hood can also be provided to keep water off the face and mouth of the escapee. As he ascends, the air expanding within the buoyancy bag escapes through relief valves placed to maintain a flow of air across his face. No other breathing device or source of air needs to be provided. A well-trained individual can escape even without a hood, but the risk of panic and of air embolism is much greater. An exposure suit can be added to the buoyancy vest to prolong survival on the surface while waiting to be picked up.

In 1970 a team of Royal Navy submarine-escape instructors from HMS DOLPHIN in Gosport made repeated and successful ascents from a Royal Navy submarine submerged to depths considerably in excess of the 200 odd feet from which Captain Bond and Commander Steinke had made two world-record ascents with American equipment a decade earlier. The biomedical research program necessary to support a USN deep-escape capability has since been completed at NSMRL.

Doctrinal emphasis in the U.S. Navy for rescue of the crew of a disabled, bottomed submarine has always been upon the use of a 1-ATA rescue chamber or rescue submarine. Two DSRV's at Submarine Development Group One in San Diego have been designed for submersible operation to 500 fsw and also have the capability of mating with a submarine in distress at depths down to 2000 fsw. Procedures for this latter operation are now being worked out. The DSRV's can operate either from the new ASR's (ASR 21 class) or from specially modified nuclear "mother submarines."

The Naval diving systems that have been discussed are capable of operation to 850 fsw, although considerably more work remains to be done to exploit that capability safely, especially at pressures within the deeper range. Extension of this capability down to diving depths of 1500 fsw appears a reasonable goal now, and we have been asked to do so, with a long-range objective of determining if 2500-fsw diving depths are attainable and at what physiologic or economic cost.

Continental shelf bottom depths lie under 10% of the oceans' surface, an area equal to one quarter of the world's land masses. Bottom depths 1000 fsw deeper than this cover a significant geographic area and possess the potential for mineral-resource exploitation. Submersibles capable of going to 20,000 fsw, however, would be capable of operating under 98% of the world's ocean surface. Whether the requirement for a specific Naval operation will call for manned diving, manned submersibles, unmanned remotely controlled submersibles, fixed 1-ATA ocean-floor structures, or an integrated mixture of these components, the responsibility for a medical program to support the development of these Naval capabilities will fall to the submarine medical service and its civilian counterpart.

Problems in diving medicine research currently include the following:

1) Underwater Breathing Apparatus. There is a critical need for bioengineering design criteria to improve underwater breathing apparatus for use at very great depths, to be used with present Deep Diving Systems within their deeper range capabilities from 600-1000 fsw, and for future use with Deep Diving Systems to attain depths to 1500 fsw or deeper. We need to provide adequate volume flow at low resistance to prevent carbon dioxide buildup and retention by the diver.

2) Faster Decompression. European decompression schedules are roughly twice as fast as U.S. Navy ones: ascent from a 1000-fsw dive in 6 days versus 11 days with USN decompression. This may result from their use of higher oxygen levels, which might expose divers to increased risk from hyperbaric oxygen toxicity. An investigation is underway to determine if the decompression benefit is real and how it can be best achieved while avoiding oxygen toxicity.

3) Aseptic Necrosis of Bone in Diving. Prevalence of non-symptomatic bone changes is less than 3% in Navy practice, which compares with the situation in the best-regulated commercial diving practice. More must be known about this injury, its pathogenesis, and its natural history.

4) Personnel. A fundamental, continuing problem is to find qualified, trained researchers with good ideas to work in our new high-pressure research facilities.

Goals for the research program in Diving Medicine are to: 1) improve the personal equipment of the diver to enhance his effectiveness and improve safety; 2) give the diver more working time in the water; 3) safely decrease decompression time requirements; 4) eliminate aseptic bone necrosis as a diving problem; and 5) extend diving capability to its limit.

B. MAN-IN-THE-SEA PROGRAMME IN THE UNITED KINGDOM: DAVID H. ELLIOTT

For those concerned with research into the safety and efficiency of man under the sea, it is beneficial to review perspectives broader than those of specific research projects, many of which were covered at the Sixth Symposium on Underwater Physiology. It may be profitable to spend a few minutes looking at the whole picture of diving, particularly those medical problems in the United Kingdom which have arisen from the recent growth of international diving activity in the North Sea.

It is useful to consider the United Kingdom program for the support of diving operations in the areas of occupational health, emergency services, research, and training.

The offshore exploration in the North Sea has had an effect on many different sectors of medicine. The large populations of migrant workers brought into relatively remote areas of Scotland have caused problems for the local health services which need not concern us in detail, but which serve to illustrate the impact upon local communities of a rapidly growing offshore industry. The concept of offshore medicine has been widely used in the U.K. to cover all those medical problems which may be associated with the ships and rigs of the North Sea. The logistics of providing an industrial health service offshore are being solved by the oil companies and their subcontractors. While there is room for further development in areas such as survival after a helicopter is lost in these cold waters, the majority of offshore problems are related to the diving industry.

Perhaps the most important development in this field has been the introduction of legislation, the Offshore Installations (Diving Operations) Regulations, which came into effect on 1 January 1975. The various government agencies which share responsibilities in the North Sea now have some measure of control over diver safety and health. In addition to existing rules specifying the minimum provisions for compression chambers and other diving equipment, there are regulations which require every diver working in the British sector of the North Sea to have an annual medical examination by a doctor approved for this purpose by the Secretary of State of the appropriate government department. This physical examination is laid down in the CIRIA (Construction Industry Research and Information Association) "Principles of Safe Diving Practice" and was originally based on that used in the Royal Navy for the experimental deep-diving team. It is extensive but needs no further elaboration here. No special medical skills are required to conduct such an examination but the interpretation of the results in terms of the fitness of a particular diver to continue his profession does require that the examining doctor have a special knowledge of diving and its hazards. For this reason the Regulations state that the approved doctor must have attended a suitable course of Underwater Medicine and occasional refresher training.

There is much potentially valuable information in the collected medical records of these divers and there are proposals to establish a data bank. However, if correlations are to be sought between the results of the long-bone X-rays and the possible etiological factors of osteonecrosis, much more information about individual diving activity and decompression incidents needs to be collected. To assemble this data completely would not only be an enormous task but would also necessitate overcoming the natural reluctance of commercial diving companies to disclose the details of their decompression tables.

It is not surprising that the attention of the British media has been riveted on the fatal diving accidents in the North Sea. It is clear that professional diving is a high-risk occupation and that some of these deaths might have been avoidable. Indeed the civilian physician has come in for particularly harsh criticism, some of it ill-informed and quite unjustified.

Offshore Emergency Medical Service

The emergency arising from a major disaster at sea, such as the collapse of a rig, requires coordination among several authorities and need not concern us here. For lesser accidents there must be an offshore emergency medical service. It must provide cover for accidents quite unrelated to diving and, although these are in the majority, their management is relatively straightforward. The diving emergency which requires a specialist probably concerns either decompression sickness which is beyond the scope and experience of the diving supervisor or some traumatic accident to a diver while he is still at depth.

The first such emergency medical service in the United Kingdom was organized at Great Yarmouth some years ago for the gas fields in the southern North Sea. A group medical practice has now been formed into the Institute of Diving Medicine to provide a center for such services. At Aberdeen, a center for the current and extensive offshore operations in the bleak and inhospitable waters of the northern North Sea, the Institute of Offshore Medicine, has been established by the University of Aberdeen with support from the U.K. Offshore Operators Association. Its functions include providing emergency medical service for the offshore industry. It is also intended that the Institute will become the center of an offshore occupational health service and of related research activities. It is not for me to comment upon these civilian institutes, both of which have their merits, their aspirations, and their problems, but it is my personal view that they have been established none too early to meet the urgent demands with which they are currently expected to cope.

U.K. Diving Research Programs

I will not spend time on the organization of financial and governmental support for the U.K. diving program; suffice it to say that responsibilities are divided among many authorities and that there is no single coordinated U.K. program outside that of the Ministry of

Defence. To review the U.K. research field I have chosen to proceed not by research topic but on a geographical basis. This, of course, exposes me to possible criticisms of omission and bias, but it is my intention and hope to be impartial.

Scotland, Ireland, and Wales

At Aberdeen, where most oil and diving companies have their North Sea headquarters, there are to be at least two commercial deep-compression chambers, but these are reserved for emergency and training purposes and no research program has been planned around them. The Institute of Offshore Medicine at the University of Aberdeen is so recently established that there is no active program of research beyond those items which are already related to its existing surgical hyperbaric unit and its interests in cold weather exposure. Scotland's only Department of Occupational Health is at the University of Dundee but, while it has plans for research based on a commercial chamber there, Dundee was considered by the U.K. Offshore Operators Association to be too far south from the Aberdeen heliport to become the base for its operations.

At the Western Infirmary, Glasgow, there has been an active program based on its surgical hyperbaric unit for many years. Much of this work should already be familiar to you, but current studies into the mechanisms of pulmonary oxygen poisoning and edema are worthy of special mention.

At Edinburgh there is, as far as I can discover, no medical research program directly related to diving interests, but one should acknowledge that city's Professor of Medicine, Professor Donald, not just for his work on acute oxygen toxicity in the 1940's but for his continuing participation as a consultant in this field.

Pending oil exploration of the Celtic Seas, diving activities based in Ireland and Wales are minimal.

England

The work done at Newcastle needs no introduction from me. The Medical Research Council's Decompression Sickness Panel and Registry of Compressed Air Workers have pioneered health surveys and, with the RNPL (Royal Naval Physiological Laboratory) have provided safe decompression tables for compressed air workers. The MRC (Medical Research Council) classification of the radiological lesions of aseptic bone necrosis is acknowledged worldwide and its collection of bone X-rays now includes a significant number of commercial divers. The recent research program there has investigated the origin of micronuclei in the body, the use of ultrasound to detect early intravascular events during decompression, and animal studies of aseptic bone necrosis (which are discussed by Professer Walder later in this Symposium).

In the Department of Pharmacology at Oxford University, Professor Paton has a long-standing research program in the hyperbaric research field. With the Department of Physical Chemistry they were among the first to use the exotic gases, such as SF_6, as a research tool in this field and they have made fundamental contributions on the mechanisms of narcosis and the pressure-reversal of anesthesia. Related animal studies of convulsions in very deep diving are continuing at the MRC Clinical Research Center near London. Other Oxford studies have included decompression performance using the exotic gases, pulmonary edema, the origin of intravascular bubbles and studies of the influence of absolute pressure upon the threshold for oxygen toxicity.

At the risk of making some unintentional omission, it seems fair to say that there is no other biomedical work in the civilian diving sector. The only unit capable of human deep-diving research in the United Kingdom is that at Alverstoke. In the Royal Navy there is a continued saturation diving program. The safe accomplishment of 250-meter, 7-day saturation diving tables in our wet and dry chamber, the Deep Trials Unit at Alverstoke, was reported at San Diego (Sixth Symposium) and the first dives to confirm the validity of these tables are being conducted at sea.

At the Royal Naval Physiological Laboratory a methodical program, free from any operational demand for haste, has been initiated recently to look at the physiological problems related to the depth limit of helium-oxygen diving. It is anticipated that this project in the RNPL deep dry chamber (700 m) will run for several years, its pace and the depth to be attained being determined solely by academic progress. It is also worth noting, among so much worldwide pioneering research into man-in-the-sea, that RNPL, like many other laboratories, is also concerned with a number of the basic problems which are still incompletely answered at shallower depths. These include the calculation of decompression tables for nonsaturation dives, a 5-year follow-up of the bone necrosis survey in naval divers, mechanisms of oxygen toxicity, hematological studies, aspects of respiratory physiology, and the evaluation of underwater breathing apparatus.

However, the Royal Navy has one program that is unique: research into submarine escape. This program, also perhaps applicable to smaller submersibles and to 1-ATA sea-bed habitats, is based on a need for survivors to escape without any intervention by surface support (such as the use of a Deep Submergence Rescue Vehicle (DSRV)). The safe escape of men from submarines as deep as 600 fsw by the technique of very rapid compression while breathing compressed air was proved in the open sea five years ago and is now in operational service. Safe escapes deeper than this depth are undoubtedly possible in an emergency with existing equipment and, while a laboratory program at RNPL continues to examine ways of extending the safe limits of the Royal Navy escape system, current work is focused upon those accidents which occasionally occur during submarine escape training. We believe that our training techniques are as safe as

they can be made and that our treatment of burst lung and air embolism is as good as is available anywhere, but we are nevertheless charged with the responsibility of searching for any possible improvements. This research into pulmonary barotrauma and its complications is directed into three principal channels: the retrospective investigation of the patient following an incident, including a detailed clinical study at the Royal Naval hospital (Haslar) in an attempt to locate possible etiological factors; a study of breathing patterns in the hooded suit during ascent through the water; and a pathophysiological study of pulmonary barotrauma and cerebral arterial air embolism and the possible development of new therapies (a field of research also relevant to certain diving casualties).

While the work on air embolism may be considered to have some relevance to other clinical forms of cerebral infarction, its importance stems from the need for safety in naval training and has thus been allocated a high priority.

U.K. Diver Training Program

To complete the picture of U.K. diving activities I would like to mention some of our training programs. To meet the urgent need for more helium-oxygen supported divers in the North Sea a government-sponsored training center is being established in the deep waters of Loch Linnhe on the west coast of Scotland. Beyond essential medical cover, there are no firm plans for any biomedical involvement at this stage. The need to train the rig medics in the basic principles of diving is acknowledged even though this is regarded as being a responsibility of their employers.

The approved physicians who perform the annual medical examination of commercial divers in the British sector of the North Sea are required to have attended a suitable course of instruction in underwater medicine. One must welcome the recognition by government authorities of the importance of training in this field, even for those physicians who remain ashore. At present the only such course in the U.K. is the introductory course held for medical officers in the Royal Navy. The slots allotted to civilians in this course, held at the Institute of Naval Medicine, Alverstoke, are now fully booked with a 12-month waiting list. However, the number of physicians already approved should be sufficient to meet the present demand.

A major difficulty, and one that is not confined to the North Sea, is that of training the civilian physicians who might fly out to the site of a diving emergency. There is no formal program for such training in the U.K. The majority of those who have volunteered to undertake this responsibility have attended the Royal Navy's introductory course and one or two of them have been able to stay for a longer period with us at RNPL. For those who may need to enter a compression chamber in order to provide the primary care for an injury or illness which has occurred at depth, perhaps this training

is sufficient; but for those who may be called upon to attend a diver at depth when the incident is due to a diving accident or a decompression disorder, the 1-week course is clearly inadequate. The minimum qualification for such a physician should be training as a professional diver, naval or commercial, plus the experience of having been involved with an experimental or operational diving team for some years. Such standards mean that there can be no quick response to the need for more physicians in the Offshore Emergency Medical Service in the North Sea.

Concluding Remarks

In spite of these many problems, the rapid expansion of diving off the coast of the United Kingdom during the past decade has brought us one advantage. The problems of diving have been brought to the attention of the general public. Some might say that the attention given to diving fatalities by the public media has been excessive and not always well-informed, but at least there is now awareness at all levels of society of the hazards of diving and the importance of diving to the national economy. While the U.K. has no coordinated program outside that of the Ministry of Defence, there is a feeling of quiet optimism amongst those in this field of research that the next decade will prove to be even more important in the development of U.K. underwater activities.

Summary

The United Kingdom program in support of diving operations is reviewed with particular reference to those problems which have arisen as a result of the recent growth of international diving activity in the North Sea. Occupational health services and offshore emergency medical services are among the essentials which this development has necessitated. The introduction of the Offshore Installations (Diving Operations) Regulations in early 1975 marked the beginning of statutory controls covering nearly all aspects of commercial diving operations. Research in the U.K. into the problems of diving is reviewed on a geographical basis and the current program of naval research into aspects of submarine escape is considered to be of particular importance.

C. MAN-IN-THE-SEA PROGRAM IN JAPAN: MOTOHIKO MATSUDA

In the early 1960's, undersea habitation experiments were performed by French and United States marine scientists; the success of these experiments, which advanced the technology of undersea habitation using saturated diving techniques, stimulated the interest of marine scientists and engineers in Japan. In 1965 the Science and Technology Agency of the Government of Japan provided a grant for comprehensive study of the more recent advances in these fields with the intention of launching similar experiments in Japan. Using this grant, a group of scientists associated with the Tokyo Medical and Dental University conducted a successful saturation diving experiment to a depth of 100 m in 1967.

In 1968, the Agency contracted with the Japan Association of Underwater Exploitation to perform an engineering study and to construct an undersea habitation system, consisting of an undersea habitat, a buoy equipped with a Deck Decompression Chamber (DCC), and a Personnel Transfer Capsule (PTC) aboard to support the habitat from the sea surface. The construction of the whole system was completed by the end of March 1971. In early September of the same year, the Japan Association of Underwater Exploitation conducted a test of the engineering performance of this habitation system in shallow water off Yakosuka, Japan. Japan Marine Science and Technology Center (JAMSTEC), founded in October 1971, took over the Association to conduct further experimentation with this system.

The first series of undersea habitation experiments using saturation diving techniques, named SEATOPIA, was launched in 1971 by the Science and Technology Agency of the Government of Japan as one of the pioneering projects in the field of marine science and technology. The SEATOPIA Undersea Habitation Experiment was planned by the Agency to develop an operational saturation diving system. The ultimate goal of the habitation experiment was for four aquanauts to stay at a 100-meter depth for one month. To achieve this, two habitation experiments at 30- and 60-meter depths were scheduled, and JAMSTEC started preparations for the experiment at a 30-meter depth immediately after taking over control of the project from the Japan Association of Underwater Exploitation.

Japan Marine Science and Technology Center (JAMSTEC)

To realize the orderly exploitation and harmonious utilization of the seas, it is indispensable to set up an appropriate system composed of marine-related knowledge and techniques in various fields of science and technology. Because of the enormous financial outlay and the need to muster specialists from a variety of fields to accomplish this aim, a unified effort of the government and concerned private circles is required. With this in mind, JAMSTEC was established in October 1971 as one of the leading technical research and development organizations in the field of marine development.

JAMSTEC is an incorporated body promoted by the civilian side and authorized by the government in accordance with legislation entitled "Japan Marine Science and Technology Center Act." It is under the administrative control of the government through the Science and Technology Agency, and is run with funds furnished by business circles and the government. JAMSTEC will carry out its mission as one of the promoters and technical pioneers of marine development, in view of the national policy and activities leading to marine development in Japan and elsewhere, and also in consultation with industrial and academic circles. International cooperation is another matter of interest. JAMSTEC expects to be an important asset to Japan in this respect. The mission of JAMSTEC is to raise further the levels of marine science and technology in Japan by carrying out a variety of research and development work, providing and managing experiment facilities for common use, and by training researchers and engineers.

For the present, the principal tasks of JAMSTEC fall into the following four categories.

Development of pioneering techniques. Comprehensive research and development of some pioneering techniques which are needed in various aspects of the exploitation and utilization of the seas will be implemented by JAMSTEC. These techniques include underwater work, conservation of the marine environment, and deep-sea surveys.

Arrangement and maneuvering of the large-sized research facilities for common use. Aiming at efficiency, large-sized and costly research facilities are constructed at and maintained by JAMSTEC for making common use of research sponsored by the government, industries, and academic communities. At present, the following facilities are planned: a hyperbaric water vessel, a diving training tower, a towing test tank incorporating a wavemaker, an underwater acoustic test tank, a deep research submersible, and a research ship. An undersea simulator and a diving-training pool are already offered for common use. A towing tank and hyperbaric water vessel are under construction.

Training of marine-oriented scientists and engineers. As an essential way of promoting marine science and technology, JAMSTEC endeavors to organize training courses in marine science and technology for scientists and engineers with various technical backgrounds. Educational programs for deep divers and technicians who are to engage in underwater projects are also to be organized.

Information and documentation services on marine technology. In connection with the acceleration of marine technological development, JAMSTEC implements a series of information services including collection, analysis, and evaluation and compilation as well as dissemination of useful technical information. Information exchange with overseas institutions concerned with marine science and technology are earnestly desired. There are five departments of which the following two are related to the Man-in-the-Sea program: the Manned Undersea

Technology and Engineering Department, and the Training Department of the Administration Office. The Manned Undersea Technology and Engineering Department has three research groups: technology and engineering for undersea habitation and manned undersea projects; equipment and tools for diving; and undersea physiology and medicine.

The main facilities of JAMSTEC are described below. According to the construction plan, various kinds of facilities required for research and development and also for personnel training are to be constructed so that JAMSTEC may perform its functions efficiently as the integrated and advanced research and training organ for marine development. Further, a tract of land with a sea area of 15,000 square meters is planned for reclamation and will be used for open-air experiments. At present, the following facilities are available at JAMSTEC:

Diving training pool. A total floor space of 1,570 square meters.

Surface support vessel. A surface support vessel, from which the entire experiment is controlled, is moored to the sea bottom, and supplies are provided to other systems. Dimensions of the support vessel are: length, 40 m; width, 11 m; depth, 3 m; draft, 1.5 m; displacement, 600 tons.

Deck decompression chamber (DDC). Dimensions of the deck decompression chamber are: length, 7 m; width, 2.5 m; height, 2.3 m; weight, 9.3 tons.

Personnel transfer capsule (PTC). Dimensions of the personnel transfer capsule are: diameter, 2.3 m; weight in the air, 8.4 tons; pressure-resistant hull, 1.8 m x 2.8 m.

Simulator for 500-meter depth. The undersea simulation system is designed to create on land a high pressure environment of up to 50 ATA corresponding to a sea environment of 500 meters in depth. This system is used to study diving physiology, medicine, work and instruments, and also to train divers.

Habitation at 30-meter depth. Although no serious danger was anticipated in testing the habitation system's mechanical performance in shallow water, extended periods at a depth of 10 m might present difficulties. Before the beginning of this experiment, fatalities were reported in the SEALAB III experiment conducted in the United States and the HELGOLAND experiment in West Germany. In Japan a preliminary simulated dive conducted in the DDC met with a small accident. In order to avoid such danger, the habitation system and its associated instruments were re-examined, remodeled, and improved.

Twelve divers were selected for habitation from professional divers and trained in basic diving physiology and technology. A simulation dive was carried out on board the support vessel, moored

in the shippond of the Maritime Self Defense Force in Yokosuka. Four divers stayed at 4 ATA for 46 hours; ambient gas was composed of 7.5% oxygen, 28.25% nitrogen, and 64% helium. Temperature in the DDC was held at $28^{\circ}C \pm 2^{\circ}$, and relative humidity at 70% ± 10%. Decompression to normal pressure required 29 hours, but took place without any abnormal symptoms.

In selecting the site of actual habitation at sea, the following conditions were considered. To settle the habitat on the sea bottom with stability and safety, a flat and level bottom with high ground-proof pressure was required. To ensure safe diving and operation in the sea, high transparency and good visibility as well as lack of strong current were desirable. As a site which met with the above conditions, a spot in Tago Bay on the western coast of Izu Peninsula (a central part of Japan facing the Pacific Ocean in Shizuoka prefecture) was selected.

Habitation at a 60-meter depth. Experimental habitation at a 30-meter depth yielded valuable knowledge and experiences related to diving physiology and technology. Based on this experience, a habitation at 60 meters was attempted.

The selection of divers who were expected to stay in the habitat was made in April 1973. A simulated dive in the DDC was conducted twice during the months of July and August on board the support vessel, moored to a berth in Yokosuka Harbor. In the first habitation, a medical officer joined the group and made medical observations and physiological studies in the DDC. Temperature in the chamber was usually maintained around $28^{\circ}C$, but was intentionally lowered to $26^{\circ}C$ in order to study the effect of cold in a hyperbaric environment. The humidity was controlled at approximately 60%, and the divers stayed for 168 hours at 7 ATA. Decompression to normal pressure required 71 hours.

Because of cooperation in medical and physiological studies from universities, including the University of Hawaii, and a food manufacturing company, a great deal of knowledge and data were obtained in these dives. The results of these diving experiments were reported at the Sixth Underwater Physiology Symposium held in San Diego in July 1975 by Dr. Nakayama (1), and two comprehensive reports were recently published in Undersea Biomedical Research (2,3).

The actual habitation in the sea occurred in late September in Tago Bay where the habitation at a 30-meter depth had taken place during the previous summer. Four divers stayed for three days in the habitat and medical and physiological studies were carried out while they were in the habitat and working in the sea. The temperature in the habitat was maintained between 29 and $30^{\circ}C$ and partial pressure of oxygen and carbon dioxide were satisfactorily controlled.

The observations on the physiology of divers in their habitat were almost consistent with those obtained in the simulated dive in the DDC. In the sea divers felt the breathing gas was very cold because of the high thermal conductivity of helium and resulting heat loss. This suggests the need to supply warmed gas for the experiment at greater depth.

After completion of the stay in the habitat, divers were decompressed in the DDC on board the support vessel without any trouble. Sixty-six hours were required to get from 6.3 ATA to 1 ATA.

The habitability of the support vessel for those working on board had been much improved, but further improvement in accommodations was considered necessary for experiments of a longer duration.

Experiment at 100-meter depth. The ultimate goal of the SEATOPIA Undersea Habitation Experiment is set at 100 meters in the sea; the preparation for this experiment started in October 1974. The results and experience obtained in two previous experiments made it essential, for the success and complete safety of future experiments, to re-examine and overhaul the whole habitation system in Spring, 1975. The first simulated habitation experiment was conducted in June-July using a simulator recently equipped by the Japan Marine Science and Technology Center, Natsushima, Yokosuka. The second simulation dive was conducted at the end of July in collaboration with the University of Hawaii. This simulated habitation lasted 18 days, including pre-dive, decompression, and post-dive periods. The experimental spot in the sea is not under investigation and the experiment will be carried out in late 1975; saturated divers will probably be kept in the DDC on board the support vessel instead of in the habitat while they are not working in the sea.

Future Plans

The highly successful SEATOPIA project will terminate this year, and next year will mark the beginning of a new era for Japan Marine Science and Technology Center. Using the newly completed facilities, we plan to extend the depth capability eventually to 300 m (approximately 1,000 feet) by 1979. In reaching this goal, we plan to ask for as much cooperation as possible from our international partners.

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SESSION II: ENERGETICS OF EXERCISE IN WATER

SESSION II: ENERGETICS OF EXERCISE IN WATER

A. IMMERSION EFFECTS ON PULMONARY, CIRCULATORY AND GASTROINTESTINAL SYSTEMS: C. E. G. LUNDGREN

As soon as a diver enters the water he is subjected to certain stresses, some of which may be fully apparent even before he has begun his dive. The functions of the respiratory, circulatory, and gastrointestinal systems are influenced by pressure differences between the breathing gear and different parts of the diver's body. Once submerged, the depth difference between the diver's breathing apparatus and chest does not change as he proceeds down. Therefore, immersion effects remain independent of the absolute depth. The main difference between a surface swimmer with the water level at his mouth and a fully submerged diver with a breathing valve in his mouth is that the diver can change posture so that the direction of the depth differences acting on him may change.

Certain fundamental aspects of immersion can be studied in the non-immersed subject who is exposed to positive and negative pressure breathing. However, because of the differences in external hydrostatic pressure at different levels of a diver's body, the two situations are not strictly comparable. For instance, the transmural pressure gradients of the extrathoracic airways will be considerably larger during pressure breathing than during immersion for any given transthoracic pressure difference (referred to the centroid in the case of immersion). Negative pressure breathing (-20 to -22 cmH_2O) appears to have only a slight effect on central blood volume (29), whereas head-out immersion causes a marked intrathoracic blood pooling (2). This short review will be limited mainly to observations in immersed subjects. Furthermore, it is immersion in the erect position that has received most attention from various investigators, and usually during actual diving with conventional gear the diver's thorax will be exposed to pressures which are higher on the outside than on the inside. Therefore, the model for most of this discussion will be the erect subject exposed to head-out immersion or the erect diver wearing a breathing valve at the mouth level.

The pressure centroid of his chest is at a point approximately 7 cm posterior to and 19 cm caudal to his sternal notch (26). The outside pressure on the chest is higher than the inside pressure by about 35 cmH_2O. However, the diaphragm is only exposed to part of the pressure head exerted by the external water column. As demonstrated by Agostoni, Gurtner, Torri and Rahn (1) during head-out immersion, the increase in transdiaphragmatic pressure is only about 14 cmH_2O although the height of the external water column at the level of the diaphragm during head-out immersion may amount to about 25 cmH_2O. This is so mainly because during immersion the abdominal wall is stretched toward the abdominal cavity and recoils outward with a pressure of about 13 cmH_2O. Nevertheless, it is

upward movement of the diaphragm more than a compression of the rib cage that determines the reduction in lung volume. For a detailed analysis of the interplay between forces on these different structures at head-out and intermediate levels of immersion, the reader is referred to the authors just mentioned (1), and Craig and Dvorak (15). These forces may cause a decrease in the functional residual capacity (FRC) to about 50% to 60% of its normal value.

As can be inferred from Table IIA-1, this decrease is mainly achieved by a reduction in expiratory reserve volume (ERV), but several authors have also found small reductions in residual volume (RV). An increased intrathoracic blood volume is presumably responsible for the RV reduction. As will be shown later in detail, a large amount of blood is redistributed into the thorax from the periphery during immersion. This extra amount of blood may occupy space at the expense of lung air. Alternatively, airway closure may render part

Table IIA-1. Summary of observations of relative changes in vital capacity (VC), residual volume (RV) and expiratory reserve volume (ERV) introduced by head-out immersion

Reference	VC, % change	n	RV, % change	n	ERV, % change	n
Agostoni et al. (1)	-9	2	-16.8	4	-70 (from 35.8% to 10.8% of VC)	8
Salldin et al. (5)	-7.8	12				
Brozek et al. (10)			- 8.0	9		
Carey et al. (12)	-0.7	8	+ 5.8	8	-47.9 (from 26.1% to 13.7% of VC)	8
	-5.5*	8	+20.1*	8	-51.8* (from 28.2% to 14.4% of VC)	8
Craig and Dvorak (15)	-8.9	11			(from 37% to 10% of VC)**	
Craig and Ware (16)	-4.0	21	- 4.0	21		
Dahlbaeck and Lundgren (18)	-7.7	20			-74.0 (from 40.2% to 11.3% of VC)	20
Dahlbaeck (17)	-4.3	6	No change	6		
Flynn et al. (20)	-2.8	5	- 7.0	5	-53.0 (from 39.4% to 19.0% of VC)	
Hamilton and Mayo (22)	-6.4	20				
Hong et al. (23)	-3.3	7				
Hong et al. (24)***	-8.0	4			-72 (from 35.4% to 11.7% of VC)	
Jarrett (25)	-4.3	5				
Jarrett (26)			- 8.0	3		

Data are % of controls during non-immersion. Depth of immersion varied between suprasternal notch and mouth (nipples in (22)); body posture was either sitting or standing; comparisons are either between group means or individual paired comparisons. *After apneic dive training. **Deduced from graph. ***Compared to control value while immersed to the xiphoid process.

of the RV inaccessible for measurement with gas dilution techniques unless a very large tidal volume is employed. It is noteworthy that in so doing Dahlbaeck found no systematic change in RV during immersion (17). Blood redistribution into the thorax may also be partly responsible for the well-known reduction in vital capacity (VC) of about 3-10% which is induced by head-out immersion (Table IIA-1). Based on effects of Valsalva maneuvers counteracting intrathoracic blood pooling, Hong, Cerretelli, Cruz and Rahn (24) estimated that 60% of the VC reduction was due to intrathoracic blood pooling, the remaining 40% being caused by mechanical influences on the chest. Using diastolic pressure cuffs around arms and legs to prevent intrathoracic blood pooling, Hamilton and Mayo (22) obtained results from which it can be calculated that about 60% of the VC reduction during immersion is caused by blood pooling. Dahlbaeck (17), on the other hand, found that the entire reduction in VC (4.3%) during head-out immersion could be avoided if blood redistribution into the thorax was prevented by a special technique (Fig. IIA-1), exempting the legs and gluteal region from immersion.

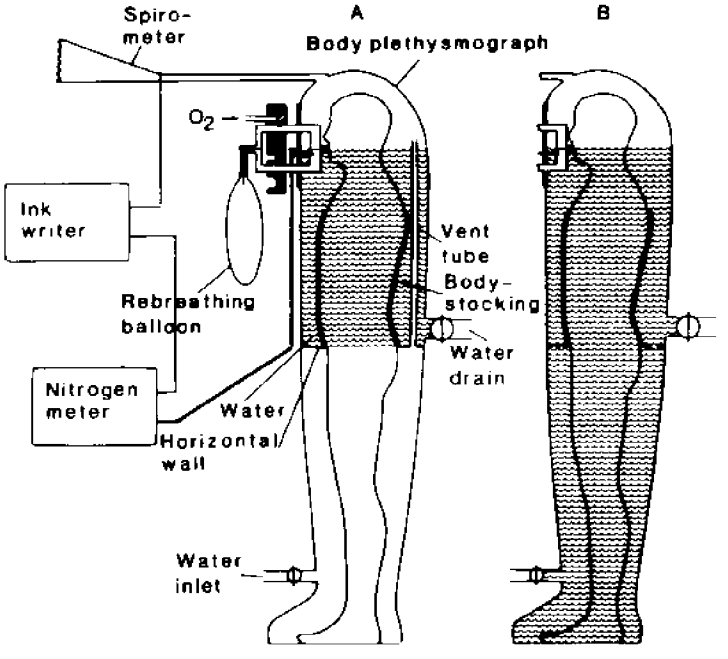

Fig. IIA-1. Experimental arrangement for separating effects of intrathoracic blood pooling from mechanical effects on lungs during immersion. In A, thorax immersion allows blood pooling in legs; in B, blood from legs is redistributed into thorax during head-out immersion (17).

Another effect of intrapulmonary blood pooling is an increase in lung compliance (9,24,38). In the study by Bondurant, Hickam and Isley (9), lung compliance changed from 0.168 liter/cmH_2O during non-immersion to 0.116 liter/cmH_2O during head-out immersion.

The reduction in FRC and the intrathoracic blood pooling interfere with normal lung ventilation. Thus, a proportion of the lung which constitutes 2.0% of the vital capacity may not be ventilated during immersion as demonstrated by Dahlbaeck and Lundgren (18). In accordance with this, Bondi and Bennett (8) have recorded an increased closing volume of the lungs during head-out immersion. More recently Dahlbaeck (17), using the thorax immersion technique, has shown that intrathoracic blood pooling is responsible for 75% of the gas trapping. Furthermore, the parts of the lungs which are ventilated show a more uneven ventilation (31), and it appears that this unevenness of ventilation is combined with a mismatch of perfusion. Cohen, Bell, Saltzman and Kylstra (13) observed an increased $AaDO_2$ during immersion. This poorer ventilation-perfusion adaptation appears to be confined to a limited, presumably basal region of the lungs, because it was not evident in regional lung function studies by Arborelius, Balldin, Lilja and Lundgren (3), who compared large apical and basal parts of the lungs using ^{133}Xe spirometry. They found that in head-out immersion there was a more even alveolar ventilation to perfusion ratio ($\dot{V}/\dot{Q}$) than in the non-immersed state. Thus $\dot{V}/\dot{Q}$ for the apical regions changed from 1.56 to 1.17, and from 0.78 to 1.24 for the basal regions. This overall effect on the lungs may be expected because when the FRC is reduced largely by an upward displacement of the diaphragm more basal alveoli are likely to approach the lower, flatter part of the pressure volume curve. Thus, they would tend to become less ventilated. Ventilation in apical alveoli would improve because they would move to the steeper part of the pressure volume curve. The more evenly distributed ventilation was matched by an improved apical perfusion which presumably resulted from the increased intrapulmonary blood volume during immersion and the increased perfusion pressure (2).

It has been hypothesized that closed-off parts of the lungs may become over-expanded and suffer rupture when a diver makes a free ascent, especially if he further reduces lung volume by exhaling excessively (18). The volume of trapped gas in the lungs held at maximal expiratory position during immersion may amount to more than 5% of the VC (18).

Even with normal breathing the trapping of pulmonary gas in immersed subjects may become a problem. Oxygen breathing for two hours during head-out immersion caused a mean reduction in VC of 22.4% in 13 subjects (5). Atelectasis formation apparently caused this VC reduction, since deep inspirations, although initially inducing cough and chest pain, finally restored VC.

The maximal voluntary ventilation (MVV) is reduced by about 15% in head-out immersion (20,34). This MVV reduction may be the result

both of the transthoracic pressure of water opposing inspiratory effort and of increased flow resistance because of reduced airway diameter if the FRC is reduced. However, observations of FRC during MVV maneuvers and simultaneous immersion seem to be lacking. There is indirect evidence that the inertia of the water does not significantly limit the ventilatory capacity during immersion (20).

As for airway resistance during quiet breathing, Agostoni et al. (1) found that it increased by 58% during head-out immersion. The reduction was attributed to the decrease in lung volume. Hong et al. (24) measured respiratory work during immersion at rest. Comparing immersion to the neck with immersion to the xiphoid process while employing a stable VT of 1.0 liter, they found that the total work of breathing increased by approximately 4000 g/cm (60%). Of this increase 75% was attributed to elastic work and the rest to dynamic work. This marked increase in dynamic work of breathing during immersion to the neck was attributed to an increase in flow resistance of the airways functioning at a lung volume where the ERV was reduced to 450 ml (from 1600 ml). The possibility that some of the airway resistance is caused by swelling of blood vessels in the walls of small air ducts remains to be investigated. In addition to the elastic and dynamic work of breathing during immersion, static work may be performed in maintaining the FRC at a level different from that obtained at relaxation. Thus, Jarrett (26) and Hong, Ting and Rahn (23) have evidence that there is a tendency during head-out immersion toward increased inspiratory muscular tonus. Similarly, we have recently seen signs of varying, sustained expiratory muscle tonus during immersion in the head-down position (unpublished observations). Such static work adds to the respiratory work (which is further increased because of high gas density in deep diving), and may increase the risk of exhausting the diver.

The respiratory frequency, tidal volume and pulmonary minute ventilation remain largely unchanged during immersion at rest (19,20, 24,36), as does end-tidal PCO_2 (20). Provided comparable work loads are applied both during non-immersion and immersion, this also appears true during exercise (14,20,33). Varying the intrapulmonary pressure to the point of discomfort on both the positive (+20 cmH_2O) and negative (-10 cmH_2O) side during immersion did not influence minute ventilation (36). The pressure centroid of the chest was determined at 7 cm behind and 19 cm below the sternal notch by Jarrett (26). In contrast, divers allowed to select the most comfortable intrapulmonary pressure preferred it to correspond to the pressure at the suprasternal notch in the prone and head-down position. In the erect position a level of 5-10 cm below the external auditory meatus was preferred during rest but when there was hyperpnea from any cause the level was 5 cm deeper (36). It is possible that this deviation from what appears to be the physiologically best pressure was to some extent caused by disturbances of respiratory muscle tone initiated by the requirement to hold on to a mouthpiece (26) and/or pressure sensations from the extrathoracic airways (28).

Repeated reference has been made to the possibility that immersion may cause intrathoracic blood pooling. In head-out immersion the amount of blood redistributed from the periphery amounted to 0.7 liter, as determined by Arborelius, Balldin, Lilja and Lundgren (2) who used the dye dilution technique for multiple measurements in two subjects. Part of this blood would be in pulmonary vessels, part in large extrapulmonary veins and part is accommodated by the heart (Fig. IIA-2). Thus, Guyatt et al. (21) found a distinct increase in the pulmonary capillary blood volume during head-out immersion and a

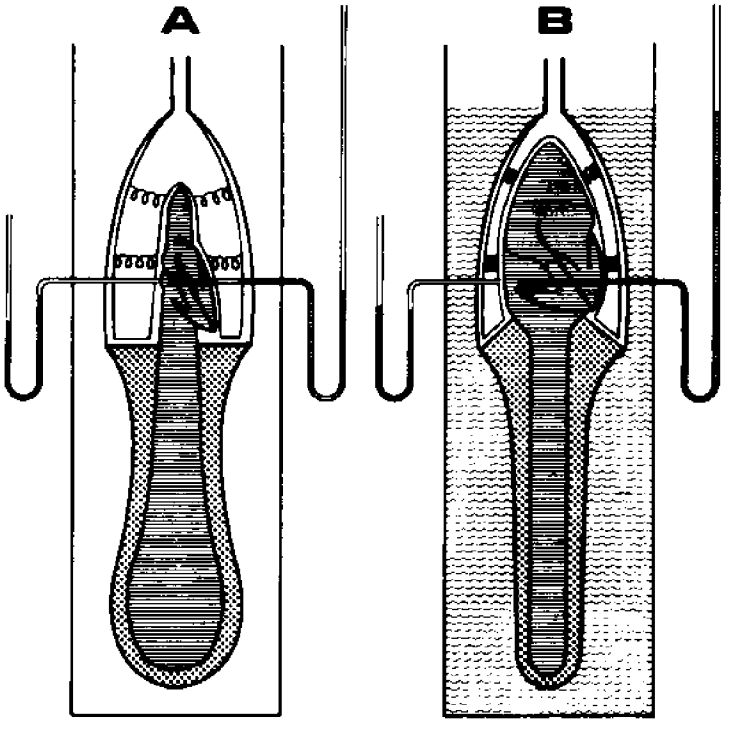

Fig. IIA-2. Schematic illustration of effect of immersion with head above water on distribution of blood between dependent regions of body and thorax. Springs indicate elasticity of lung tissue. Dotted areas: incompressible but resilient tissues. Hatched areas: blood. Left manometer (black fluid column) indicates pleural pressure; right manometer (hatched fluid column) indicates right atrial pressure. Difference between right atrial and pleural pressure reflects atrial transmural pressure gradient. A = erect body position in non-immersed situation, blood pooled in vascular bed below the heart. Atrial pressure is almost zero; pleural pressure is negative. Right atrial transmural pressure gradient is

small. B = erect body position during immersion with head above water; redistribution of blood towards heart and intrathoracic vascular bed distends these structures. Atrial pressure is raised; pleural pressure is almost zero. Right atrial transmural pressure gradient is increased. (Modified from Gauer (2))

consequent increase in pulmonary diffusing capacity by 16.7%. Lange, Lange, Echt and Gauer (30), using roentgenometry in 10 subjects, demonstrated an increase in heart size during immersion in the standing position of 180 ml (50%). In the earlier mentioned study by Arborelius et al. (2), the functional consequences of such a distension of the heart were measured in 10 subjects. Transition from a thermoneutral non-immersed situation (sitting in air at 28°C) to head-out immersion in thermoneutral water (35°C) caused a 35% increase in stroke volume. This increase was due mainly to a cardiac output increase of 32%, the heart rate remaining essentially unchanged. The transmural pressure increase in the right atrium was 13 mmHg on the average. The 30% decrease in peripheral resistance which was evident in these experiments may have been caused by changes in the autonomic control of the circulation due to activation of various receptors within the circulatory low pressure system (35). Part of the fall in resistance may also have been due to an abolition of myogenic precapillary sphincter tone as the external water column balanced the intravascular pressure. Earlier work made use of few subjects and less accurate indirect methods to determine cardiac output. Under neutral thermal conditions an enhancement of cardiac output by immersion was usually reported, ranging from a mean of 6% to 22% (2). The 12% reduction in resting cardiac output which was recorded by Rennie, di Prampero and Cerretelli (37) in water below 34°C was due to a reduction in heart rate and was considered a response to cold stress. During exercise the cardiac output values were the same in water as in air, presumably because of increased heat production which eliminated the cold stress. During moderate exercise stroke volume was high and heart rate low relative to air values, but stroke volume approached air values with more strenuous exercise. Balldin, Lundgren, Lundvall and Mellander (7) have demonstrated that the experimental conditions of Arborelius et al. (2) are conducive to a greatly increased peripheral circulation. They learned that head-out immersion induced a 130% increase in the rate of elimination of ^{133}Xe from a muscle. Tissue nitrogen elimination during oxygen breathing was increased 40% during the first 30 minutes of immersion in 35°C water and was 29% higher than during non-immersion over a 7-hour period (6). This enhancement of nitrogen elimination, further strengthened by increasing water temperature to 37°C, was used by Balldin to attain a considerable protective effect against experimental altitude decompression sickness in man (4). This method ought to be used to prevent and treat decompression sickness in divers. However, the same principle acting during the uptake of inert gas would promote tissue gas saturation.

Because of the considerable vertical length of the gastrointestinal system, it may be exposed to considerable pressure inequalities during

immersion. The magnitude of these pressure differences is closely tied to respiratory mechanics, and thus to breathing pattern (1) and immersion effects on the respiratory system. Johnson, Lin and Hong (27), in a study of five subjects, demonstrated an increase from 4.6 mmHg in gastric pressure (non-immersed) to nearly 20 mmHg during head-out immersion. Esophageal pressure rose from -6.0 mmHg to -0.8 mmHg. Thus, there was a gastric to esophageal pressure gradient which amounted to about 20 mmHg in immersion versus 10-11 mmHg in the non-immersed subject. Although the distal esophageal sphincter pressure always remained 11-15 mmHg higher than gastric pressure in their healthy subjects, the authors concluded that immersion may entail the risk of gastric reflux in individuals with insufficient competence of the distal esophageal sphincter.

The possibility that immersion may facilitate esophageal transport toward the stomach and thus cause problems in diving has recently attracted our interest. Of 2,053 sport divers responding to a questionnaire, 111 (5.4%) indicated that they had suffered significant gastrointestinal distension and discomfort after ascent because of aerophagia during the dive (32). One of the factors which may have contributed to the aerophagia in these divers was their steep head-first descents. Such descents may create a large gas pressure gradient between the oral cavity and the stomach. The theory that the attitude of the diver in the water may influence the degree of aerophagia was tested in simulated wet dives in a pressure chamber in which the subjects descended either head first or feet first to a depth of 30 m (11). Swallowing was used to obtain middle ear pressure equilibration. Gastric gas content was determined by underwater weighing. Feet-first descent caused a mean weight reduction ($\underline{n}$ = 10) equivalent to the ingestion of 0.11 liter of air (at 1 ATA) whereas head-first dives introduced more than twice as much air (0.29 liter) into the gastrointestinal tract. One subject who dove head first and made seven excursions between 25 and 30 m surfaced with considerable abdominal discomfort after ingesting an air volume of more than 1.3 liter.

To gain information on the conditions which promote gas transport into and through the gastrointestinal system during diving we are now studying the esophageal pressure profile during head-down and feet-down immersion. A representative example of the results of these experiments is shown in Fig. IIA-3. The pressure during head-up immersion is largely on the positive side relative to mouth pressure and increases toward the stomach, which corresponds to the results of Johnson et al. (27). By contrast, in the head-down position pressure is clearly on the negative side, particularly in the distal part of the esophagus. The esophagus is thus likely to receive larger amounts of air and convey them to the stomach whenever the tonus of the distal sphincter is lowered by swallowing.

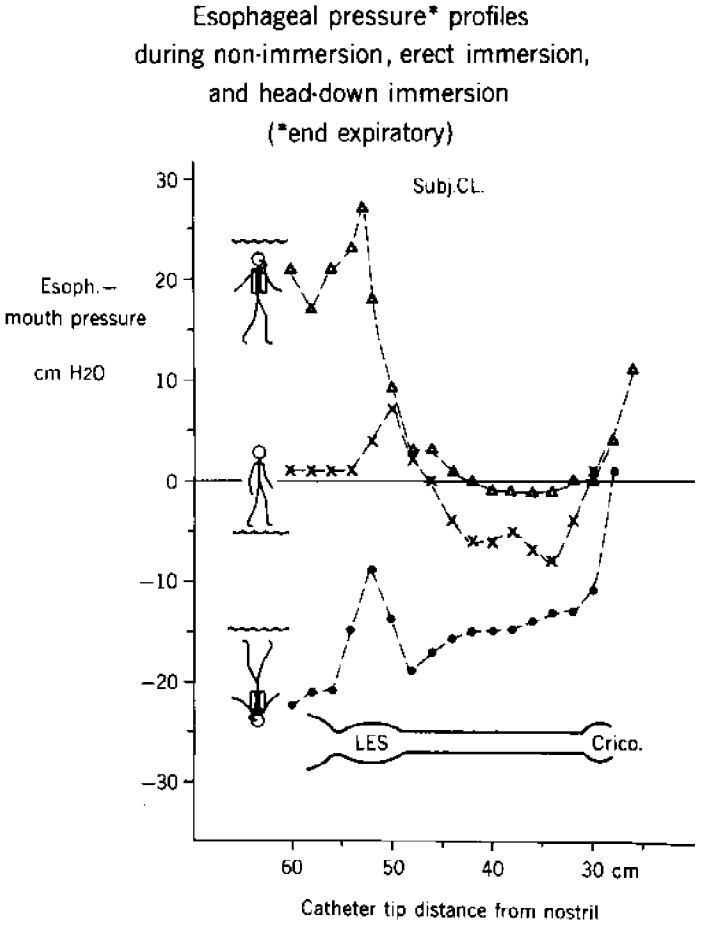

Fig. IIA-3. Esophageal pressure relative to pressure in oral cavity (ordinate) plotted versus position of tip of catheter (with 3 cm-balloon) in esophagus (abscissa) from one subject. Measurements were obtained by stepwise retraction of the catheter from stomach under 3 conditions, i.e., immersion in erect body position (upper curve), non-immersed standing position (middle curve), and head-down immersion. Schematic figure of esophagus with lower esophageal sphincter (LES) and cricopharygeus is shown for orientation.

Summary

This review deals with certain immersion effects on the respiratory, circulatory and gastrointestinal systems, mainly seen during immersion in the erect posture. Head-out immersion or erect submersion with the breathing valve at the mouth level causes lung compression with

increased elastic, dynamic and static respiratory work. Air trapping occurs in closed-off lung regions but overall ventilation-perfusion adaptation improves. Ventilatory minute volume and end-tidal CO_2 tension are not greatly influenced by immersion. Apart from direct compression effects, lung function is influenced by considerable intrathoracic blood pooling. This blood pooling increases venous filling of the heart, thereby inducing a 30% increase in cardiac output due to an increase in stroke volume. Peripheral circulation increases and greatly enhances inert gas transport. Increased stomach to esophageal pressure differences in the erect position may entail the risk of gastric reflux. In the head-down position high oral gas pressure relative to the esophagus and stomach may induce aerophagia, causing serious gastrointestinal distension in a diver as he surfaces.

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B. ENERGETICS OF SCUBA DIVING AND UNDERSEA WORK: JEFFREY DWYER

For years SCUBA divers, as performers of undersea work, were studied almost exclusively in terms of perceptual and motor skills, problem-solving capabilities, work methodology and ergometry, and team coordination (2,3,4,5,37). Physiological studies of diver performances were by no means unavailable, but most were limited to simple measurements of heart rate, breathing rate, or air consumption (18,36,38) coupled with descriptions of underwater tasks, task completion times, and error rates. A few early studies of oxygen consumption were also available but these were limited by the use of 100% oxygen systems to very shallow depths and light work loads (9,13). However, recent developments in underwater data acquisition systems (28) and respiratory gas sampling methods (11, 12) for air-breathing SCUBA divers have provided scientists with measurements of oxygen consumption and energy expenditure at depths to 190 fsw in the ocean (19). These measurements accurately describe the energetics of SCUBA diving and permit more precise and meaningful interpretations of underwater work than any other physiological parameter. Recent studies of the energetics of air-breathing divers working in the open sea, coupled with some early studies of $\dot{V}O_2$ in divers breathing 100% oxygen in open water and in special water tanks, now provide sufficient data to describe the working SCUBA diver as a source of power and a performer of physical work.

The rate of oxygen consumption has long been recognized as the best indication of the diver's energy expenditure during work or exercise. It is an accurate index of cardiopulmonary stress and an excellent basis for comparison with other physiological parameters and studies. In diving physiology research few studies of oxygen consumption are available due to the formidable technical problems of measurement during ocean dives. Yet it is true that $\dot{V}O_2$ measurements are vital to any rational organization of underwater work (16), assessment of the energy requirements of underwater tasks (10,13,34, 36), studies of the effects of the underwater environment on mechanical efficiency (13,14,34,35), and recognition of the physiological limits of undersea workers (9,19).

The early studies of $\dot{V}O_2$ during swimming with SCUBA and other forms of underwater work used 100% oxygen rebreathing systems. Oxygen uptake measurements were obtained by the tank pressure differential method but severe limitations were placed on depth and work load by the toxic nature of oxygen under pressure. Modern studies of $\dot{V}O_2$ in air-breathing SCUBA divers have used tank pressure differentials to determine the pulmonary ventilation and various gas collection techniques to obtain samples of mixed exhaled air for FE_{O_2} and FE_{CO_2} analysis. These sampling systems range in sophistication from simple rubberized bags to trap-exhaled gas bubbles (16,34,35), and from vacuumized SCUBA tanks for single sample collection (12,30), to the system devised by Dwyer (11), which is capable of collecting ten gas samples at several different depths during a single dive.

Basic Studies of Oxygen Consumption

In the mid 1950's Donald and Davidson (9) published reports of oxygen consumption experiments with working divers in a 12-ft tank, and in open water, which continue to be among the most complete studies of their kind. The subjects were Royal Navy divers equipped with diving suits, oxygen rebreathing apparatus, and weighted boots or swim fins. Experiments at minimum and maximum speed walking around the tank and over a muddy bottom in an estuary revealed $\dot{V}O_2$ rates which were surprisingly low. For maximum speed $\dot{V}O_2$ was 1.53 and 1.96 liter/min in the two respective environments. These values are in close agreement with data reported by Kurenkov (19) for divers walking at 15 m/min at a depth of 60 meters with helmet and diving suit. It is of interest to note that maximal walking efforts caused exhaustion in fifteen minutes yet the $\dot{V}O_2$ suggests that moderate work was performed. The $\dot{V}O_2$ for minimal speed in either area was between 0.6 and 1.0 liter/min, indicating that the range of oxygen requirements between minimal and maximal efforts in these experiments was quite narrow. The data also suggest that divers are incapable of utilizing their normal maximal oxygen uptake ($\dot{V}O_{2max}$) underwater. A lack of traction on the bottom and the aqueous environment apparently prevent the muscles from reaching the maximal rate and force of contraction (22,34). Reports by Donald and Davidson on oxygen uptake during underwater swimming are included in other sections.

Underwater arm work was also performed at 139 kpm/min, requiring $\dot{V}O_2$ of 1.15 liter/min. After 10 minutes the subjects reported "severe local fatigue in the arms and shoulders." In contrast, a self-paced underwater search mission, involving primarily the legs, required 1.47 liter/min. As in surface work, the amount of muscle mass involved, i.e., whether work is performed with the arms or legs, is an important consideration in assessing the underwater work stress from the oxygen consumption (1).

Only one study is available which measures $\dot{V}O_2$ during common, variable underwater labor. Gulyar and his associates (16) monitored several physiological parameters in air-breathing SCUBA divers at a depth of 20 fsw while they operated an undersea drilling facility. Oxygen consumption during "relative calm," i.e., at rest, was unusually high, averaging 1.41 liter/min. While clearing the drilling area with crowbar, air hammer, connecting hoses, pipes and positioning equipment, the $\dot{V}O_2$ increased to 1.66 liter/min. The divers had some difficulty maintaining positional stability during the calm period and this may have caused the high $\dot{V}O_2$. During the work period the handling of lines and tools apparently added to their positional stability so the divers could redirect the oxygen uptake to useful work. This would account for the small difference in oxygen uptake between the two periods and is in accordance with the findings of Streimer and his associates (34,35) for self-paced work.

Weltman and Egstrom (36) have recognized the bio-instrumentation problems of measuring underwater energy expenditure by $\dot{V}O_2$, and the apparent necessity of using easily measured parameters, such as heart rate (HR), for field evaluations of underwater work stress. However, their attempt to characterize the physiological load of various tasks by the heart rate response was not successful because oxygen-consumption/heart-rate curves were not used to calibrate heart-rate/work-stress analysis. Other investigators (10,20, 25) found that regression lines for $\dot{V}O_2$-HR data varied significantly with water temperature and pressure. Thus, the use of heart rate to assess underwater work may result in large errors which lead to underestimations of the energy cost and actual work stress.

Several investigators have measured oxygen consumption during fin-swimming, i.e., using a leg kick for propulsion with the aid of foot fins but without the use of the arms. A summary of some of the available data collected in pools and special water tanks is presented in Fig. IIB-1. Schaefer (32) found very consistent values for $\dot{V}O_2$

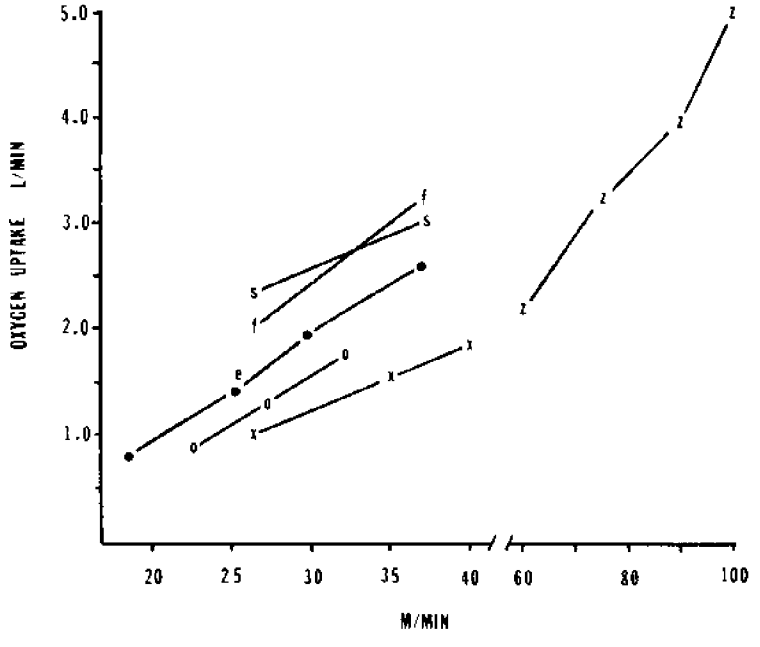

Fig. IIB-1. Oxygen cost of underwater fin-swimming. ● = Specht et al. (33): 3 fsw, 100% O_2; o = Goff et al. (14): 3 fsw, 100% O_2; e = Schaefer (32): 100% O_2 at 40 fsw; x = Foley et al. (12): 3 fsw, air, no protective suit; z = Kurenkov (19): 60 meters, air (open-sea swims); Donald and Davidson (9): 3 fsw, 100% O_2; s = with protective suit; f = without protective suit.

as his divers swam at 26 m/min around the perimeter of a large wetpot. As the depth increased from 20 to 30 and 40 fsw, the $\dot{V}O_2$ also increased in 60- to 100-ml increments, presumably due to the increased oxygen cost of breathing a gas of progressively greater density. Goff et al. (14) and Specht et al. (33) found similar $\dot{V}O_2$ for swims at 18-21 m/min in a moving water tank (the David Taylor Model Basin). At any speed, however, Goff's divers consumed less oxygen than those in Specht's group. The difference was roughly 300- to 400 ml/min at speeds beyond 25 m/min. Specht et al. and Lanphier (21) reported the oxygen uptake at 24.6 m/min to be 1.40 and 1.30 liter/min, respectively. In contrast, Foley, Billings, and Huie (12) found $\dot{V}O_2$ at 40 m/min which were about the same as that reported by Specht's group at 30.7 m/min. This discrepancy, and other differences in oxygen uptake reported by several investigators, may be due to different drag configurations among subjects.

This idea is supported by the findings of Goff et al. (14), who discovered differences in drag and $\dot{V}O_2$ as great as 0.5 liter/min between two groups of divers swimming at the same speeds but wearing wet- or dry-type diving suits. Furthermore, Goff et al., Specht et al. and Dwyer (10) have stressed the critical importance of the correct, near-horizontal body position in determining the oxygen cost of a swim. Divers swimming with a feet-up or feet-down attitude may require as much as 1.0 liter/min of O_2 more than divers swimming with the correct body attitude. Individual differences in mechanical efficiency and swim fin configuration may also be responsible for the lack of agreement among investigators on the oxygen cost of underwater swimming.

Open-Sea Studies of Oxygen Consumption During Fin-Swimming

The oxygen cost of fin-swimming in open water has only been measured by three research groups over a period of 17 years. In 1956 Goff, Frasetto, and Specht (13) studied Navy divers swimming in tropical and subtropical waters with oxygen rebreathing equipment. Two hundred open-water swims were made over distances ranging from 500-2,000 yards (461-1846 meters). The divers swam at a self-paced rate, but regardless of distance the average speeds varied between 20 and 24 m/min. In water temperatures of 25°C, without protective suits, the $\dot{V}O_2$ at 16.5, 25.0, and 32.0 m/min were 1.25, 1.40, and 2.20 liter/min, respectively. The measurement at 25.0 m/min agrees closely with data reported for fin-swimming in special water tanks (32,33). However, at faster speeds the $\dot{V}O_2$ was higher in the ocean.

After a period of 16 years, during which no studies of oxygen consumption in open water took place, Russell, McNeill, and Evonuk (30) published a comprehensive study of the physiology of divers swimming at three depths with modern wet suits and compressed-air SCUBA. Swims were performed at 30 m/min at 1, 2, and 3 ATA in a fresh water lake. The $\dot{V}O_2$ at these depths were 1.65, 1.80, and 1.93 liter/min,

respectively. Since water temperature was the same at each depth (5.5°C), the progressive increase in $\dot{V}O_2$ appears to be the result of the increased work of breathing. However, because the wet suit provides less cold protection with increasing depth due to suit volume compression, a large portion of the excess oxygen consumption was perhaps the result of greater cold stress at 2 and 3 ATA. The $\dot{V}O_2$ are in fair agreement with the data of Goff et al. (14) and Specht et al. (33).

Foley's divers (12), who swam in a warm pool with nearly identical equipment except that they wore no wet suit, swam 10 m/min faster than the other divers, but still had $\dot{V}O_2$ of 1.8 liter/min. The difference in swim speed for a particular oxygen uptake rate highlights the effect of the diving suit, i.e., drag configuration and, perhaps, cold stress on work efficiency. In a study by Kurenkov (19), divers swam at a depth of 60 meters (7 ATA) in the ocean. Their equipment and breathing apparatus were not described but it may be assumed that the divers wore wet suits and some form of compressed-air SCUBA. Kurenknov did not describe his research apparatus, but $\dot{V}O_2$ in excess of 5.0 liter/min at swim speeds of 100 m/min were reported. At 60 m/min oxygen consumption was 2.2 liter/min.

No device presently available measures the physical work output of freely swimming divers, but Dwyer (10) has used a unique underwater ergometer to control the work output of divers swimming in the ocean at depths to 100 fsw. In this comprehensive study 12 physiological parameters were measured under 12 work-depth conditions. The ergometer has been described by Pilmanis, Henriksen, and Dwyer (29). It is a drag board which the diver holds in front of his body as he swims with fins, using a leg kick. A drag indicator located on the ergometer indicates to the diver the added resistance he is working against. This resistance is in addition to the unknown amount of work required to move his body and life-support equipment through the water. At any level of resistance the swim speed was found to be constant. Therefore, work loads were reproducible, work performed in moving the ergometer through the water could be calculated, there was no need for external pacing to maintain a constant swim speed, and the diver actually moved through the water as he would if he were transporting a tool or other resistive object to an underwater work site. Data acquisition was made possible by a unique underwater recording system (28) and an underwater gas sampler designed by Dwyer (11). Physiological measurements were made during rest and with three work loads at 33, 66, and 99 fsw (Table IIB-1).

Resting oxygen consumption at 33 fsw was 385 cc/min, 38 cc/min greater than measurements made with the subjects at rest on land prior to a bicycle ergometer work test. At 66 and 99 fsw, the $\dot{V}O_2$ was essentially the same (see Table IIB-1), representing an increase of 45 cc/min over the shallow depth. These rates are higher than those of Donald and Davidson (9), who found $\dot{V}O_2$ of

Table IIB-1. Experimental results during underwater ocean swimming at 33, 66, and 99 fsw, against resistances of 1.5, 2.0, and 2.4 kg and during rest

Depth, fsw		33	66	99
$\dot{V}O_2$, liter/min	Rest	0.385	0.431	0.429
	1.5 kg	1.760	2.370	1.982
	2.0 kg	2.538	2.867	2.715
	2.4 kg	3.279	3.325	3.465
Energy Cost, kcal/min/m^2	Rest	0.986	1.118	1.103
	1.5 kg	4.537	6.209	5.112
	2.0 kg	6.535	7.419	7.024
	2.4 kg	8.471	8.587	8.946
Heart Rate, beats/min	Rest	74.0	68.0	75.0
	1.5 kg	134.0	143.0	138.0
	2.0 kg	159.0	155.0	151.0
	2.4 kg	172.0	164.0	163.0
VA, liter/min	1.5 kg	29.58	41.17	34.92
	2.0 kg	44.81	54.79	50.04
	2.4 kg	65.40	57.44	58.12
PA_{CO_2} mmHg	1.5 kg	47.19	46.02	48.65
	2.0 kg	43.07	41.89	42.56
	2.4 kg	42.71	50.75	50.94

Data from Dwyer (10)

250 cc/min for fully equipped divers at 12 fsw. However, Russell et al. (30) reported oxygen uptake of 512 and 755 cc/min in air-breathing SCUBA divers at 1 and 3 ATA, respectively. The water temperature in this case was a constant 5.5°C, yet the reductions in the thermal protective capacity of wet suits with increasing depth probably resulted in an added cold stress at the deep depth. Shivering was observed at each depth but it was more severe at 3 ATA. In Dwyer's experiments the temperature was 11°C at 99 fsw but no shivering was found. The highest resting $\dot{V}O_2$, 1.41 liter/min, was reported by Gulyar et al. (16) for fully equipped, air-breathing divers at 20 fsw in open water. Difficulties in maintaining positional stability significantly elevated the $\dot{V}O_2$, but Gulyar and his associates also believe that a "conditional-reflex reconstruction" of the impending tasks also contributed to the unusually high oxygen uptake for underwater rest. This psychologically induced pre-work intensification of physiological functions may be related to the extent of the subject's diving experience or familiarity with the

task (15,38). In Dwyer's study rest was followed at each depth by work which required 1.7 to 2.3 liter/min of O_2. Individual heart rate data suggested that in some cases consideration of the impending work may have been a factor in preventing complete relaxation.

Mean oxygen consumption for the heaviest work load (2.4 kg at 24.5 m/min) showed a progressive increase with depth, which conforms to the usual relationship between the parameters reported by Russell et al. (30) and several investigators who worked with hyperbaric chambers (6,19,20,31). The differences in $\dot{V}O_2$ at different depths are sufficiently small to be attributed to the effects of increased gas density and possibly lower water temperatures. However, at the other two work loads, 1.5 and 2.0 kg, the mean oxygen uptake was highest at 66 fsw, but always higher at 99 fsw than at 33 fsw. This unusual pattern and the large $\dot{V}O_2$ differences between depths at these resistances suggests that some factor or factors altered the total work load or efficiency of the divers at 66 fsw. Dwyer pointed out that some of the divers had operational difficulties at that depth. In some cases over-inflation of the buoyancy compensator occurred and two subjects swam with feet above the depth plane. Others felt that they properly inflated the compensator but swam with a feet down attitude similar to that reported by Goff et al. (14). The effect of these errors in swim position was sufficient to cause the $\dot{V}O_2$ pattern described above, although some of the divers did swim in the correct position. One diver who swam with a feet down attitude had a $\dot{V}O_2$ at 66 fsw which was 1.03 liter/min greater than his rate at 33 fsw and 0.755 liter/min greater than his $\dot{V}O_2$ at 99 fsw. Goff and his associates found equally large differences in oxygen uptake between correctly performed swims and swims in which a feet up attitude was observed. The implication is that the oxygen cost of any particular swim and the usual depth-$\dot{V}O_2$ relationship may be significantly changed by variations in mechanical efficiency related to swim attitude or differences in drag configuration. These changes are most likely to occur when divers transport equipment or other materials around underwater work sites, or when they change depth without precise compensations for shifts in buoyancy caused by wet-suit compression.

Based on a regression line for oxygen uptake and land work output, Dwyer estimated his underwater work loads to be equivalent to 900, 1250, and 1600 kpm/min performed on land. Subjective evaluations by the divers were in agreement with these estimates. Of course the actual physical work output was much less than these figures because of the inefficient nature of fin-swimming. Several studies of underwater work suggest that divers seldom exceed $\dot{V}O_2$ of 2.5 liter/min and therefore rarely approach a work stress equivalent to 1100 kpm/min performed on land. Further, since self-paced underwater arm work and fin-swimming require no more than 1.7 liter/min of O_2, and often as little as 1.0 liter/min (13,34,35), typical SCUBA dives involve a moderate work stress.

Maximal Oxygen Consumption Underwater

The maximal oxygen consumption ($\dot{V}O_{2max}$) of swimming SCUBA divers has received the attention of very few investigators (23,26). Part of the difficulty of studying the $\dot{V}O_{2max}$ underwater lies in the selection of criteria for the maximal aerobic power. Diving researchers do not administer two different work loads, as suggested by Astrand and Rodahl (1), to determine that no increase in oxygen uptake occurs despite an increase in work output, nor is the blood lactate measured for levels in excess of 70-80 mg/100 ml blood. Instead, the underwater test for $\dot{V}O_{2max}$ has become a single swim in which the subject swims as fast as possible until he feels exhausted. Methodological improvements in this test seem warranted since the $\dot{V}O_{2max}$ may be reached at work loads which are not maximal and the diver need not make a maximum effort. Other improvements in underwater ergometry, physiological data acquisition systems, and the utilization of highly trained and motivated divers may yield more reliable measurements of the underwater $\dot{V}O_{2max}$ than those presently available. Further, agreement among hyperbaric physiologists on a standard underwater $\dot{V}O_{2max}$ test is needed, from both methodological and physiological points of view.

Stationary underwater fin-swimming, where the divers are restrained by a cable attached to weights outside a water tank, resulted in a $\dot{V}O_{2max}$ which was 74% of the value found for land work on a bicycle ergometer (23). In this study air was breathed from a modified two-hose regulator at 3 fsw. Morrison (27) attempted $\dot{V}O_{2max}$ tests with mixed-gas apparatus (N_2-O_2) while his divers swam against a trapeze, restrained by weights, at their maximal sustainable effort for 10 minutes in a wet pot. In each series of experiments the $\dot{V}O_{2max}$ was much less than expected in view of earlier reports of the oxygen required in fast underwater swimming by Donald and Davidson (9). At 6 fsw the $\dot{V}O_{2max}$ ranged from a low of 2.1 liter/min to a high value of 3.4 liter/min for six divers, while at 78 fsw three divers consumed 2.0, 2.5, and 2.8 liter/min. When the trials were performed at 176 fsw only four of six divers were able to complete the swims and $\dot{V}O_{2max}$ ranged from 1.7 to 3.2 liter/min. Four divers reported difficulties such as headaches and feelings of being out of breath, which implied that carbon dioxide retention was a possible cause of early termination in some of the swims. Other investigators (9,10,12,19) have reported $\dot{V}O_2$ which approximates the maximal aerobic power but only two such studies were performed in the ocean. In swims with fins at speeds described as fast (31-42 m/min) in shallow tanks, the $\dot{V}O_2$ ranged from 2.63-4.15 liter/min, with a mean of 3.16 liter/min (9). When diving suits were worn some divers showed an increase rather than no change in $\dot{V}O_2$, but the mean fell to 3.09 liter/min. In a similar study by Foley et al. (12) one diver consumed 2.94 and 2.45 liter/min of O_2 during two separate swims at fast speed. The highest value for $\dot{V}O_2$ during underwater fin-swimming was reported by Kurenkov (19) at a depth of 60 meters in the open sea. At speeds greater than 100 m/min, three divers

exceeded 5.0 liter/min of O_2. Air was used as the breathing gas, but Kurenkov did not describe the breathing apparatus or his data acquisition methods. Dwyer (10) reported $\dot{V}O_2$ between 3.3 and 3.89 liter/min for work performed on a mobile ergometer at 99 fsw in the ocean. Subjective impressions of the divers suggested that they worked very close to their maximal aerobic power but comparable land $\dot{V}O_{2max}$ data was available for only one subject. That diver was working at 91% of his $\dot{V}O_{2max}$ while fin-swimming, but he developed acute awareness of respiratory impairment, a severe headache, and a PA_{CO_2} close to 60 mmHg.

Within the range of air-supported SCUBA dives, the gas density may increase several times and lead to alveolar hypoventilation and carbon dioxide retention (10,17). An interesting question concerns the depth at which restricted pulmonary function, rather than $\dot{V}O_{2max}$, will become the work-limiting factor. Experiments in a hyperbaric chamber at 7.8 ATA indicate that when gas density reduces the MVV below the $\dot{V}E$ required for normal elimination of carbon dioxide, CO_2 retention may become excessive and force the diver to terminate his work even at moderate $\dot{V}O_2$ (24). Differences in breathing resistance between valves used in hyperbaric-chamber exercise experiments and standard underwater breathing apparatus (17) and differences in MVV in dry and wet hyperbaric environments (26) suggest that at depths in excess of 100 fsw in the sea, air-breathing SCUBA divers may encounter intolerable PCO_2 as their metabolism increases before they are able to reach 100% of their $\dot{V}O_{2max}$. It follows that two factors of primary importance in the maximal performance of underwater work are: 1) the PCO_2 which a diver is willing, or physiologically able, to tolerate; and 2) the percentage of his $\dot{V}O_{2max}$ that can be utilized before he reaches his critical PCO_2. Both of these factors may vary with depth and diving experience.

Mechanical Efficiency and Self-Paced Work

The diver's mechanical efficiency is rarely determined, because of the difficulties of measuring oxygen consumption and physical work output underwater. Mechanical efficiency is defined as the percentage of the energy expenditure that appears as work output (1, 14), and it is computed by the equation

$$ME\% = W \cdot 100/E - e$$

where W is the work output expressed in kilocalories, E is the gross energy expenditure, and e is the resting metabolic rate.

Goff, Brubach, and Specht (14) and Specht, Goff, Brubach, and Bartlett (33) measured the mechanical efficiency of fin-swimming divers over a wide range of speeds at a depth of 3 feet. Oxygen uptake was determined by the closed-circuit method and physical work output was measured by a unique drag technique. A special moving water tank, the David Taylor Model Basin, was used to create water speeds of 19 to 38 m/min, which allowed the swimming diver to

maintain a constant position relative to researchers and instrumentation at the edge of the tank. To determine the work output, the drag resistance was measured by a strain gauge at various water speeds as the divers were suspended in the flowing water by a cable. Measures of water speed and drag were used to calculate the amount of work which would have been done by the diver had he been swimming freely at the selected speeds. The divers were then released from the cable and swam by kicking with fins against the flowing water while the $\dot{V}O_2$ was measured. It was assumed that under these conditions the diver was producing a force, by kicking, which equaled his personal drag resistance.

Goff and his associates found that the mechanical efficiency ranged from 1.2 to 5.6% in untrained swimmers, but did not vary systematically with swim speed or work output. A study of the data in conjunction with motion pictures of the experiments indicated that variations in the mechanical efficiency of any particular swimmer "could be directly related to swimming technique and the state of buoyancy of the subject." The importance of swimming technique was demonstrated by additional experiments with selected untrained swimmers. After instruction for improving the leg kick the oxygen consumption at 22 m/min was reduced 28% and the mechanical efficiency increased from 1.47 to 2.07%. By correcting a feet-up body position and high kick, one subject reduced his $\dot{V}O_2$ by more than 0.8 liter/min. The significant effect of errors in body position on the $\dot{V}O_2$ was subsequently confirmed for ocean swims by Dwyer (10). Specht and his associates (33) used highly experienced Navy divers as subjects, yet large interindividual differences in mechanical efficiency were found, along with evidence of a learning effect in kick rate as the experiments progressed. At speeds of 21.5 m/min and slower, the efficiency averaged 3.6% and ranged from 2.0 to 8.0%. At faster speeds the average was unchanged but the range was less than 1.5%. The data also indicated that the oxygen cost of swimming a nautical mile is essentially the same at 24.6 and 27.6 m/min (0.8 and 0.9 knots). At both faster and slower speeds the efficiency tended to fall, and at 1.0 knot the oxygen requirement was too high to be maintained for long periods of time. The calculated oxygen cost of swimming a nautical mile in the moving water tank agreed closely with a previous report for ocean swims by underwater combat divers (13).

Swim speed data, in miles per hour, and drag resistance, in pounds, reported by Goff et al. (14) may be used to calculate the work output of fin-swimming divers in conventional units, i.e., kpm/min. At speeds of 21.6, 25.0, and 31.0 m/min, the work output was 37.0, 47.0, and 76.9 kpm/min, respectively. On the basis of the oxygen uptake for these work rates, corresponding work outputs on a standard bicycle ergometer would be 533, 649, and 811 kpm/min. It is apparent that underwater fin-swimming is a relatively inefficient method of propulsion. The drag configuration of the diver, the viscosity of the water, and the method of applying force combine to produce a low mechanical efficiency which results in high levels

of work stress and remarkably low work output rates. In contrast, stationary cycling work involving the arms and legs simultaneously, in a seated position, is performed with equal efficiency in air and water (8). However, stationary cycling with the legs only, in a prone position, is performed with a mechanical efficiency of 13% in water compared to 16% on land (7).

Streimer and his associates (34,35) used a different approach to study the mechanical efficiency of divers. They measured the energy cost in liters of oxygen/min/HP for self-paced arm work on a flexion-extension task (hack saw) and a rotary task (grindstone). At a depth of five feet in a swimming pool the oxygen uptake remained within very narrow limits (0.98 to 1.13 liter/min) while the work output varied as task difficulty was altered by changing the resistance and/or lever arm length. As the tasks became more difficult the work output actually increased, with essentially no change in oxygen consumption from the least difficult work conditions where the work output was lowest. Thus, the energy cost of self-paced arm work was greatest, 62.9 liter/min/HP, when task operation was least difficult, compared to 26.3 liter/min/HP when it was most difficult. Streimer and his co-workers theorized that, as task operation difficulty, i.e., resistance, increased there was a reduced need for stabilization forces to keep the diver in position and the muscular efforts could be redirected to the increased production of useful work. Conversely, as operation difficulty decreased the need for stabilization forces increased and these muscular efforts were provided by reducing the work output rather than increasing the oxygen uptake. These findings seem to be confirmed by Gulyar et al. (16), who found oxygen uptake of 1.41 liter/min during rest while they were exposed to currents, surge, and variations in buoyancy. When the divers began clearing the bottom of rocks, connecting hoses and cables, and operating a drill, the $\dot{V}O_2$ increased by only 200 cc/min because they could redirect their muscular efforts from stabilization to the production of useful work. Thus, a diver is most efficient when his task inherently provides for body stabilization while extraneous muscular efforts are minimized.

Self-paced work studies were also performed at 33 and 66 fsw in the ocean. At either depth, $\dot{V}O_2$ was remarkably constant despite innate efficiency differences between the two tasks. Between depths self-paced work rates showed no change but $\dot{V}O_2$ was consistently greater at 66 fsw. Thus, the divers met the added oxygen cost of breathing at the deeper depth by increasing $\dot{V}O_2$ rather than decreasing the work output.

Summary

Within the past twenty years a limited number of studies of oxygen consumption in SCUBA divers has been published. However, sufficient data is presently available to describe the energetics

of SCUBA diving and undersea work. The oxygen uptake of SCUBA divers while idle or at rest is significantly influenced by cold water, gas density, pre-work muscular tensing, and the need for muscular efforts to maintain positional stability.

The oxygen cost of fin-swimming has been found to vary among different groups of highly trained divers because of differences in equipment drag, fin configuration, and mechanical efficiency. The mechanical efficiency of fin-swimming under these conditions is approximately 3.5%, but it may be as low as 1.0% and as high as 8.0% with highly trained military divers. Divers who are permitted to work and swim at self-paced rates probably experience no more than a moderate work stress at relatively shallow depths. However, slight changes in positional stability, swim attitude, buoyancy, and drag may greatly increase the oxygen cost of a particular task and raise the work stress to high levels.

The maximal aerobic power ($\dot{V}O_{2max}$) has received very little attention in diving research. In a pool, divers have reached 78% of their land-exercise $\dot{V}O_{2max}$ by tethered swimming. Other attempts to reach $\dot{V}O_{2max}$ by fin-swimming at maximal effort at 178 fsw were inconclusive due to a lack of comparable land-exercise data. Still other studies of $\dot{V}O_2$ during fast or maximal swimming efforts indicate a wide range of oxygen uptake, from 2.45 to 4.15 liter/min. In one open-sea study, a diver was able to work for four minutes at 91% of his land-exercise $\dot{V}O_{2max}$, but this effort resulted in an acute awareness of respiratory impairment, a severe headache and a PA_{CO2} close to 60 mmHg at 99 fsw. The role of $\dot{V}O_{2max}$ in the performance of undersea work has yet to be clearly determined. Furthermore, there is evidence suggesting CO_2 retention as the work-limiting factor during ocean dives rather than cardiovascular factors.

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C. HEAT EXCHANGE DURING UNDERWATER EXERCISE: JAMES F. MORLOCK AND T. O. MOORE

According to Nadel et al., "There have been several studies of energy production during swimming. However, determinations of the energy losses during resting exposure in the water have only been by indirect methods. Very few attempts have been made toward prediction of energy losses during swimming and no actual data have been obtained to this time (3)."

Prior to the above study Bullard and Rapp (1) summarized the literature as follows: "As one looks at the literature in this field, it is disheartening to see the many small bits and pieces in which the information exists. Future impetus must be toward more complete studies in which wide ranges of water temperature, varied metabolic rates, varied body compositions, and physical conditions are assimilated."

The following experiments were undertaken to investigate how the combined factors of water temperature, subcutaneous insulation, work load, and repetitive exposure affect the rate of heat loss from the skin surface (HSKIN) while exercising in water.

Methods

Each subject was asked to arrive at the laboratory at 9:30 a.m. on the day of the experiment. Prior to arrival he was asked to arise in his usual manner and eat a typical breakfast. Due to the length and severity of the forthcoming exposure, no attempt to commence in a basal state was made. Upon arrival the subject was asked to change into a brief nylon racing swim suit and to voluntarily void both urine and feces.

Next, the subject sat quietly in a nylon-webbed chaise lounge adjacent to the water tank and began breathing via a SCUBA double hose mouthpiece (U.S. Divers, Santa Ana, California) which he used exclusively throughout the experiment.

The experiment began with a 14-minute control period during which the subject sat quietly in the chaise lounge. This was succeeded by three 30-minute immersed exercise periods, each followed by a 60-minute non-immersed recovery period. This particular protocol was chosen to simulate a typical working diver's day.

During each of the three recovery periods the subject was wrapped in a cotton-rayon blanket covered with a radiation-preventing, and evaporation-inhibiting, space blanket (Thermos Division, King-Seely Thermo Co., Norwich, Conn.). Recovery room, and therefore inspired gas relative humidity, was measured by a lithium-chloride sensor, the resistance of which varies in proportion to the relative humidity to which it is exposed (Hygrodynamics Universal Hygrometer Indicator,

American Instrument Co., Silver Spring, Maryland). Inspired gas relative humidity while breathing from the SCUBA tank was assumed to be zero.

Inspired and expired gas temperatures were measured using 26-gauge copper-constantan thermocouples placed outside the valves in the SCUBA mouthpiece. These two temperatures as well as recovery room temperature were recorded on a multipoint potentiometer (Honeywell Co. Inc., Fort Washington, Penn.).

An underwater bicycle ergometer (2) was used to elicit a controlled and variable work load (Fig. IIC-1). The following modifications were made in the depth of the tank and the gas collection system to enable the subject's head to be completely immersed. The water tank depth was increased from 146 cm to 171 cm, and a 2 × 4 inch capping which prevented water from splashing out of the tank was placed on top of that. The gas collection system used was the SCUBA system previously described (2), except that the mixed expired gas was collected directly in the 350-liter Tissot spirometer instead of in meteorological balloons. To prevent free flow of the SCUBA regulator due to the hydrostatic pressure gradient from the regulator to water surface, the expired gas line had to be interrupted. Expired gas was vented from the mouthpiece to a 15-cm diameter, 70-cm high polyethylene cylinder sealed at the top and open at the bottom. The cylinder was mounted vertically adjacent to the SCUBA bottle on the inside wall of the water tank so that half of its volume was below the water level and therefore filled with water; the other half protruded above the water surface and contained air. The expired gas was vented inside the cylinder at the level of the regulator. It then bubbled through the water-occupied volume, collected in the vertically adjacent gas volume, and was vented directly to a 350-liter Tissot spirometer, which was used to measure expired volume.

To maintain the water bath at any given temperature below that of the climatic room the following control system was incorporated (see Fig. IIC-1). The water in the exercise tank could be pumped from the tank through a 65 × 20 cm heat exchanger and back into the water tank. The heat exchanger, which was placed in a thermally insulated ice chest filled with 55.0 liters of cold water, was constructed from 14 meters of approximately 1.3-cm (1/2 in) diameter, thin-walled copper tubing. An electric pump (model 11810-37, Jabsco Inc., Costa Mesa, Cal.) was automatically turned on whenever the water bath became warmer than the desired temperature. This was accomplished by a YSI Model 63 RC Temperature Controller (Yellow Springs Instruments Inc., Yellow Springs, Ohio). The entire electrical portion of the system was connected to a microsecond ground fault circuit breaker (Bell Electric Co., Chicago, Illinois) to prevent accidental electrocution of the subject by short circuiting.

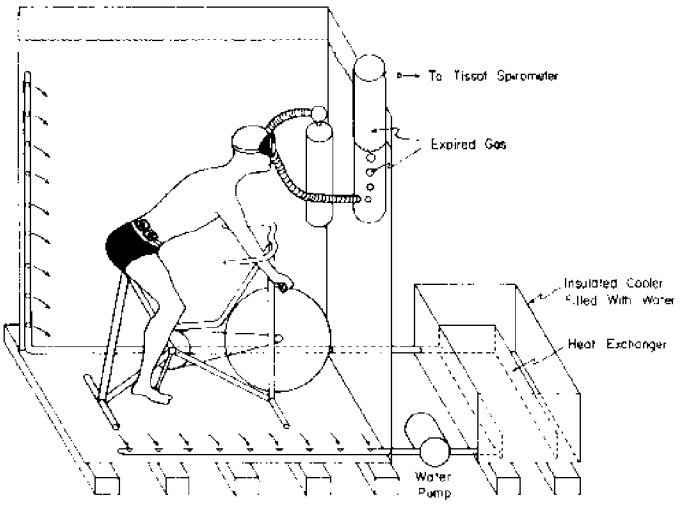

Fig. IIC-1. Schematic of experimental setup

Calibration experiments revealed that, for each degree centigrade difference between recovery room and water bath, the water in the cooler increased 0.018845°C per minute, when the water was mechanically stirred so that the water surface was similar to that when the subject was exercising in the tank (n = 7, r = 0.995). By measuring the increase in the cooler chest water temperature for a given period of time one is able directly to measure the amount of heat lost from the subject's skin surface while he is exercising on the bicycle ergometer, assuming a bicycling efficiency of 23% (6) and knowing the rpm at which the subject was pedalling (2). In other words, if the water tank temperature is held constant, all the heat absorbed by the cooler is caused by the summation of the following: heat flux from the climatic room to the water tank and cooler, rate of increase in heat content of the water due to mechanical work being done on the water, and rate of heat lost from the subject's skin to the water. Knowing the first two enables one to calculate the third, if the rate of temperature increase in the known volume of water in the cooler is measured.

The capacity of the heat absorbing system was not large enough to hold the exercise tank temperature constant during the high rates of skin heat loss when the subjects were initially immersed in the cold water. However, by the end of the immersion period the water tank temperature had returned to the desired level, thus enabling accurate determination of the average skin heat losses experienced over the entire 30-minute immersion period.

Pertinent physical characteristics of the four subjects used in the study are presented in Table IIC-1. Each subject was asked to complete the previously described protocol at three work loads and four water temperatures (27.5, 25.0, 22.5, and 20.0°C). The work

loads were chosen to elicit an oxygen consumption of 1.0, 1.5, and 2.0 liters/min in 30°C water (2). This corresponds to pedalling rates of 46, 55, and 61 rpm respectively. The subjects and work loads chosen were such that the anticipated O_2 consumption would be less than 60% of any given individual's maximum aerobic power to ensure that the imposed stress would be primarily thermal.

Table IIC-1. Physical characteristics of subjects

Subject	Age, years	Height, cm	Weight*, kg	Mean Skinfold**, mm	$\dot{V}O_2$ max, liter/min
(RD)	31	178	70.48 ± 0.19	6.32 ± 0.09	4.08
(JM)	28	184	67.72 ± 0.53	7.74 ± 0.18	3.75
(DB)	25	180	74.63 ± 0.33	10.21 ± 0.15	3.35
(MAC)	22	187	101.66 ± 0.44	11.13 ± 0.28	3.52

*Mean ± SE for 5 measurements taken over course of experiment. **Mean ± SE for 5 measurements taken at 10 sites over course of experiment.

Because of the excessive thermal drain, the leanest subject (RD) refused to go back into the water at 22.5°C the third time at all three exercise loads (rectal temperature 33.2° for 46 rpm), and was therefore not asked to attempt the colder 20°C water series. Subject DB refused to do the 20°C water series because of other commitments and the extreme stress that subject JM was experiencing in the 20°C water.

Results and Discussion

Total body heat loss will be divided into two separate pathways. HSKIN, which is by far the greater of the two at 1 ATA, will be reported first. Respiratory heat loss (HRESP), the second of the two pathways, was calculated according to the principles outlined by Webb (7).

Figure IIC-2 illustrates that if one singles out water temperature as the only independent variable, one obtains a definite, inverse linear relationship between skin heat loss and water temperature. These means were obtained by averaging the results from four subjects, working at three different work loads and three consecutive exposures, for each of the four water temperatures.

Similarly, by singling out repetitive exposure as the sole independent variable (Fig. IIC-3), one notices a trend of progressively

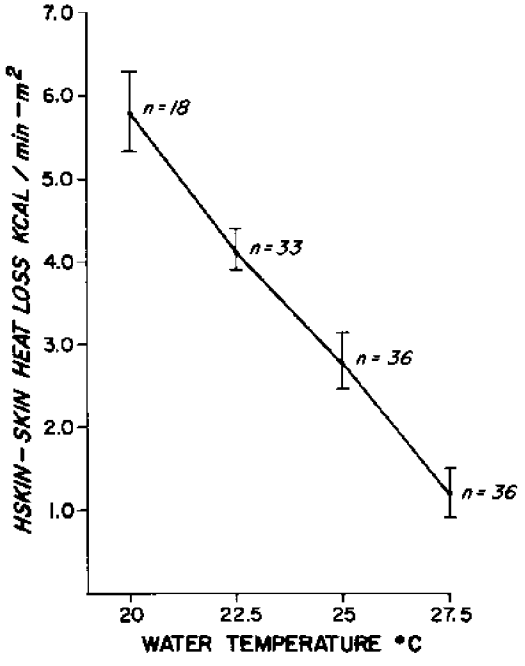

Fig. IIC-2. Heat loss from skin as a function of water temperature while exercising in water. Values are means ± SE, obtained for given desired water temperature.

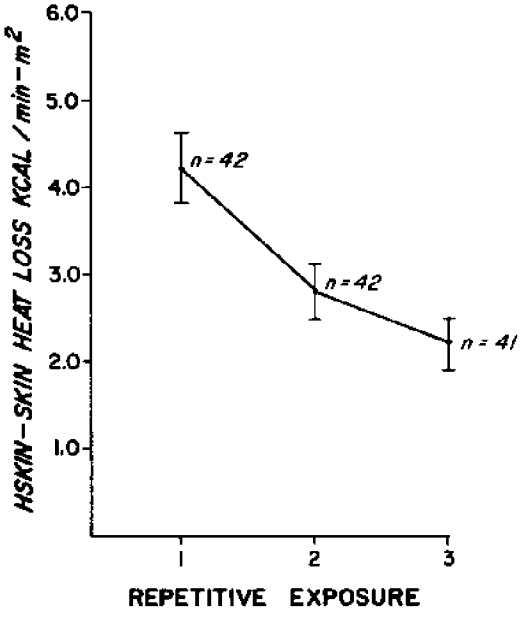

Fig. IIC-3. Heat loss from skin while exercising in water as a function of repetitive exposure. Values are means ± SE, obtained for a given 30-min repetitive exposure; each exposure was separated by a 60-min recovery period in air.

diminishing heat loss with each successive repetitive exposure. The reader should note that these are the means of HSKIN obtained for four subjects, three work loads, and four water temperatures for each of the three repetitive exposures.

Since HSKIN is so dependent on water temperature, as illustrated in Figure IIC-2, water temperature was chosen as the primary independent variable when attempting to determine if any secondary factors interact with water temperature to influence HSKIN. No consistent relationship was found between HSKIN and mean skinfold thickness (MSF) of any subject. However, if one puts the two leanest subjects into one group and the two more stout subjects into another group, a tendency toward a secondary inverse relationship between HSKIN and MSF can be seen (Fig. IIC-4).

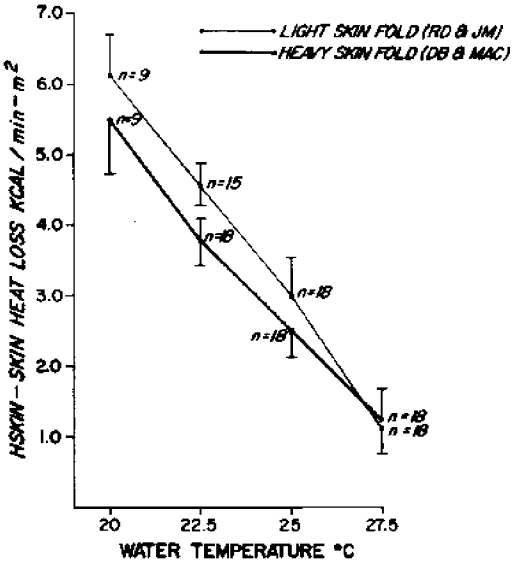

Fig. IIC-4. Heat loss from skin while exercising in water as a function of water temperature and light and heavy skinfold.

Since minute ventilation is known to be directly related to work load, and respiratory heat loss is known to be directly related to ventilation, a positive correlation between respiratory heat loss and work load was expected. This relationship is graphically illustrated in Fig. IIC-5.

To determine the relative importance of each of the four independent variables considered in determining the rate of skin heat loss, the data were analyzed by the statistical technique of multiple regression (4). The results of this statistical analysis are summarized in Table IIC-2.

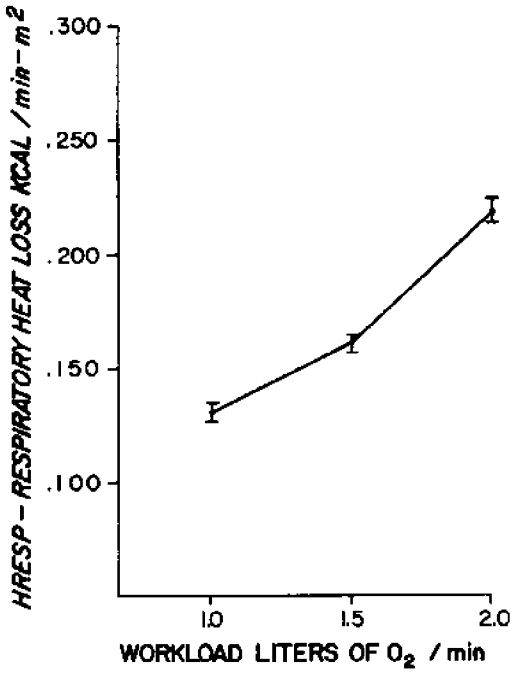

Fig. IIC-5. Mean respiratory heat loss for 30-min exercise period in water as a function of work load (see text). Values are means, ± SE, obtained for a given work load.

Table IIC-2 may be interpreted to mean that the overall regression equation when all four independent variables are considered is significant at $P = 0.005$ level and has a multiple regression coefficient of 0.756. Furthermore, 57.2% of the measured variations in skin heat loss can be explained by the four variables considered. Finally, the standard error of skin heat loss is 1.555 kcal/min-m^2. This latter value may be interpreted as the average error in predicting skin heat loss from the multiple regression equation.

Table IIC-2 also illustrates that skin heat loss is definitely not dependent on work load, which is directly related to pedalling rate and water velocity. Therefore, skin heat loss is not related to water velocity over the range of work loads tested. This is consistent with Rapp's (5) prediction based on a theoretical analysis that non-respiratory heat loss is independent of swimming speed. This non-dependence of skin heat loss on water velocity is also in agreement with the conclusion of Nadel et al. (3) that the heat transfer coefficient between the skin surface and enveloping water is essentially independent of water velocity.

Since the B values (slopes) associated with mean skinfold and work load are not even significantly different from zero at

Table IIC-2. Multiple regression analysis of skin heat loss with water temperature, repetitive exposure, mean skinfold and work load

Analysis of Variance	Degrees of Freedom	Sum of Squares	Mean Square	F
Regression	4	381.80	95.45	39.45†
Residual	118	285.32	2.42	

multiple r = 0.756

r square = 0.572

SE = 1.555

Variable	B	SE	F
Water Temperature	-0.625	0.0563	123.24**
Repetitive Exposure	-0.940	0.1731	29.48*
Mean Skinfold	-0.100	0.0757	1.75
Work load	-0.140	0.3548	0.16
Constant	21.449		

*Significant at P = 0.25; **significant at P = 0.10;
†significant at P = 0.005.

P = 0.25 level, a second multiple regression analysis considering only water temperature and repetitive exposure was done. The results of this analysis are summarized in Table IIC-3.

The results of this second analysis indicate that, if one only considers water temperature and repetitive exposure as independent variables, the multiple regression equation is significant at P = 0.025 level and has a multiple regression coefficient of 0.752. In addition, 56.7% of the measured variation in skin heat loss may be explained by the two variables considered with the same standard error of skin heat loss when all four independent variables

Table IIC-3. Multiple regression analysis of skin heat loss with water temperature and repetitive exposure

Analysis of Variance	Degrees of Freedom	Sum of Squares	Mean Square	F
Regression	2	376.90	188.45	77.92†
Residual	120	209.21	2.42	

multiple $\underline{r}$ = 0.752

$\underline{r}$ square = 0.567

SE = 1.555

Variable	B	SE of B	F
Water Temperature	-0.617	0.0559	121.73**
Repetitive Exposure	-0.951	0.1729	30.226*
Constant	20.152		

*Significant at $\underline{P}$ = 0.25; **significant at $\underline{P}$ = 0.10;
†significant at $\underline{P}$ = 0.025.

were included. The predictive equation is as follows

$$HSKIN = -0.617\ (TH_2O) - 0.951\ (REP) + 20.152$$

where HSKIN = heat loss from skin, kcal/min-m^2

TH_2O = water temperature, ^oC

REP = repetitive exposure number

The above equation allows one to estimate the expected skin heat loss knowing only the water temperature and the number of previous 30-min dives a given diver has made that same day, assuming no artificial rewarming between dives. Standard error in the above estimation can be expected to be ± 1.55 kcal/min-m^2.

A liberal estimate of the accuracy of the calorimeter may be made by knowing the following facts. The cooler which absorbed the

heat, which was added to the water tank over the 30-min exercise period, contained nearly 55 kg of water. Estimated error of the change in cooler temperature over the 30-min period is at most 0.2°C (Thermal couple recorder manufacturer claims an accuracy of 0.14°C). Specific heat of water is approximately 1 kcal/°C-kg. This leads to an estimated accuracy of 0.366 kcal/min.

In addition to this, the HSKIN calculation necessitated the assumption of a known constant bicycling efficiency of 23%. Shepard (6) says that this can vary from 21 to 25%. Assuming the largest of the three work loads, this leads to an additional error of 0.20 kcal/min. For an average surface area of 1.8 m^2, if the above two possible errors were maximized for all 123 cases, one might expect the maximum error of HSKIN to be 0.315 kcal/min-m^2. Yet the standard error of Eq. 1 is 1.555 kcal/min-m^2. It is therefore concluded that factors other than the four independent variables of Tables IIC-2,3 are needed to predict HSKIN with a greater accuracy than Eq. 1. We feel that the most likely factor would be a quantitative index of a given subject's ability to vasoconstrict the protective shell or, more generally, increase his effective insulation while exercising in water at various temperatures.

Conclusions

Heat loss from the skin surface of a working diver is not dependent on either exercise level or mean skinfold thickness over the ranges of the two variables tested. On the other hand, skin heat loss is inversely and linearly related to water temperature. This relationship is modified by the number of previous exposures in a given day. A predictive equation has been determined which allows one to estimate the expected skin heat loss of a working diver knowing only water temperature and number of previous exposures and assuming no artificial rewarming between dives. The standard error of this estimation can be expected to be ± 1.55 kcal/min-m^2.

ACKNOWLEDGMENTS

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CHAIRMAN'S SUMMARY: S. K. HONG

Since the first step of diving involves immersion in water to the neck, it was appropriate to begin this symposium with discussions of physiological effects of head-out immersion. Dr. Lundgren gave an excellent review of man's respiratory functions during immersion. He pointed out the existence of air trapping in the lung during immersion, which might lead to an accident during a submarine escape. Another important point was the facilitation of N_2 washout during immersion, which relates to the kinetics of saturation and desaturation with inert gases in a prolonged diving situation. If we can shorten the time for decompression following a saturation dive by immersing a diver in water to the neck, this will contribute much to diving technology. Cardiovascular data presented by Dr. Lundgren were also valuable because the depth in the study is greater than that in many other such studies in the literature.

Gastrointestinal problems associated with diving have not been appreciated until now. Recently, Johnson et al. (4) reported that the gastroesophageal pressure gradient increases greatly during immersion to the neck, predisposing the diver to gastric reflux. Dr. Lundgren's study indicates that the opposite is true during a head-down dive, which leads instead to aerophagia: gas will move from the esophagus to the gastrointestinal tract, where it expands during ascent, resulting in occasional severe abdominal pains. This is a new direction for research and more studies should follow this line in the future.

One theory advanced by Gauer and Henry (3) concerns a consequence of cardiovascular changes during immersion in the regulation of body fluid volume. They attributed immersion diuresis to the stimulation of volume receptors in the low-pressure system, which is supposed to inhibit the ADH system. However, such ADH inhibition has not been demonstrated. Epstein and Saruta (2) showed an inhibition of the renin-aldosterone system, which could cause a diuresis during immersion. Within the last year, two groups of investigators, Epstein et al. (1) in Miami and Kurata et al. (unpublished data) in Hawaii, produced direct evidence for the inhibition of the ADH system during immersion. According to Kurata et al., the urinary excretion of ADH decreases from 27.5 U/min before immersion to 8.9 during 3-hr immersion in water of critical temperature; the urinary aldosterone excretion decreased by 50% during immersion. These recent findings not only prove the original hypothesis (inhibition of the ADH system), but also implicate an additional mechanism (inhibition of the renin-aldosterone system) in the development of immersion diuresis.

Dr. Dwyer gave an excellent review of the energetics of underwater exercise. He has developed excellent techniques for underwater measurement of $\dot{V}O_2$ and $\dot{V}CO_2$ in resting and exercising divers. However, there are still many technical problems in making these measurements and the

results are thus highly variable. Resistance to breathing offered by the conventional SCUBA gear is so high that it is very difficult to interpret the results. For example, many investigators report significant CO_2 retention during underwater exercise, which may limit the magnitude of underwater work load. Studies conducted in our laboratory by Dressendorfer indicate that the maximal aerobic power does not significantly change either during head-out immersion or in an 18.6-ATA heliox environment, as long as the diver uses a low resistance breathing valve (unpublished data); there was no evidence of significant retention of CO_2 in these experiments. These results indicate that the physiological system is capable of supporting the maximal work load underwater, but that conventional underwater breathing apparatus is not.

Dr. Morlock developed a simple calorimetric technique and came up with very important conclusions on heat exchange during underwater exercise. He pointed out that water temperature and the number of previous exposures in a given day are the major determinants of heat loss underwater. Other factors, such as skinfold thickness and work load, do not seem to have much effect. However, the range of skinfold thickness in this study is narrow, and a more critical evaluation on the effect of subcutaneous fat may be in order.

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SESSION III: PHYSIOLOGY OF INERT GASES

A. BINARY AND TERNARY DIFFUSION OF WATER VAPOR IN He, N_2, AND He-O_2 AT INCREASED PRESSURES: CHARLES V. PAGANELLI AND FRED K. KURATA

The rate at which one gas diffuses through a second or through a mixture of other gases has important physiological consequences for animals whose natural habitat is the atmosphere. Pulmonary exchange of oxygen and carbon dioxide at the alveolar level involves diffusion in the gas phase, as does gas exchange in the avian embryo, which takes place through the pores in its shell. Insensible water loss from the skin also involves gaseous diffusion of water vapor from the surface of the skin into the surrounding air. Binary or mutual diffusion of one dilute gas in another occurs at a rate which is inversely proportional to total ambient pressure, according to the Chapman-Enskog theory (9). This theory also shows the molecular composition of the binary mixture to be an important determinant of diffusivity in the gas phase; diffusion of one gas in another of low molecular weight, such as hydrogen or helium, will occur more rapidly than through a heavier gas such as nitrogen or argon.

In experiments on water vapor loss from eggs in air, a process which is known to be diffusion-limited, Paganelli et al. (7) showed a reduction in this water loss with increasing pressures, and an augmentation at below-ambient pressures. The relations between water loss and pressure followed theoretical predictions below 1 atmosphere absolute (ATA), but deviated significantly in the direction of larger-than-expected water losses at pressures of 2 and 4 ATA. Water vapor loss into a helium environment at 1 ATA occurred about 2.4 times faster than into nitrogen at an equal pressure. The relation between water vapor diffusion and pressure has special significance for the water economy of eggs which are incubated at altitude, as Rahn and Ar (8) have shown.

Insensible water loss in man, for which water loss from eggs serves as a model, is a major element in maintaining water balance. Human subjects who live at increased environmental pressure, as in saturation diving, should show reduction in insensible water loss and a concomitant readjustment of other avenues of water loss, e.g., urine flow. The presence of helium in the atmosphere should also modify insensible loss. Hong (5) has measured such changes; his results and conclusions are discussed in this symposium.

In view of the foregoing considerations, it is important to measure the influence of pressure and the molecular species of the second gas on the binary diffusion coefficient of water vapor. This study is designed to measure the binary diffusion coefficients of the water-vapor-helium and water-vapor-nitrogen systems as a function of pressure. Since water vapor diffusion usually occurs in a ternary or quaternary system, the effect on diffusion of

introducing a third gas (O_2) into a water-vapor-helium system was also investigated.

Principle of Measurement

We have adapted the method of Schwertz and Brow (10) to the study of water vapor diffusion at elevated ambient pressure. They measured the rate of evaporation of water from a capillary tube into a second gas whose water vapor pressure was kept at 0. The water vapor pressure (P_{H2O}) at the gas-water interface in the capillary tube is the saturation vapor pressure at the temperature of the experiment (25°C), while at the mouth of the capillary $P_{H2O} = 0$, as shown in Fig. IIIA-1. Thus water vapor diffuses steadily down its gradient

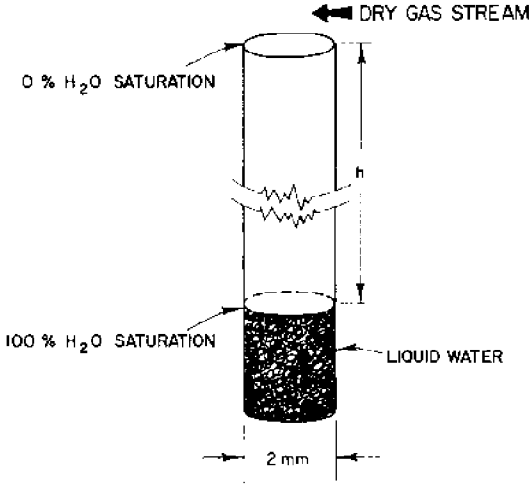

Fig. IIIA-1. Schematic drawing of capillary diffusion tube

and out of the capillary tube, and the level of liquid water in the tube falls. From the rate at which the water level falls, one may calculate D, the binary diffusion coefficient of water vapor in the second gas. The equation governing this case of one-dimensional diffusion with a moving boundary is

$$D \frac{\partial^2 c}{\partial x^2} - v \frac{\partial c}{\partial x} = \frac{\partial c}{\partial t}$$

where C = particle concentration of water vapor; x = distance along the axis of the capillary, positive direction downward; v = rate of displacement of the gas-liquid interface; t = time.

Details of the solution of Eq. 1, with the assumptions involved, are

presented in detail by Schwertz and Brow (10), who give the following expression for the binary diffusion coefficient $\underline{D}$ derived from the solution of Eq. 1

$$\underline{D} = \frac{(h^2 - h_o^2)}{2(t - t_o)} \cdot \frac{de}{ds} \cdot \frac{Ps}{P} \cdot \frac{1}{\ln(P/(P - Ps))} \qquad (2)$$

where h, h_o = heights of the water vapor-gas column at times t and t_o, respectively, (cm); de = density of water at experimental temperature, $(g \cdot cm^{-3})$; ds = density of saturated water vapor at experimental temperature, $(g \cdot cm^{-3})$; P_s = saturation vapor pressure of water at experimental temperature (Torr); P = experimental pressure (Torr).

In practice, one measures h, the height of the water vapor-gas column at increasing times t, and plots $(h^2 - h_o^2)$ vs $(t - t_o)$, where h_o and to are the initial values of h and t. $\underline{D}$ is then calculated from the slope of the line, together with known values of the constants in Eq. 2 and a measured value of P. Constants used were: de = 0.9970 $g \cdot cm^{-3}$, $d_s = 2.304 \times 10^{-5}$ g · cm^{-3}, P_s = 23.73 Torr, all at 25°C.

Materials and Methods

Diffusion Tubes. The glass capillaries used as diffusion tubes were straight-bore 200-µl capillary pipettes, 70-80 mm in length and 2 mm in ID. Uniformity of bore was checked by mercury weighings and found to vary by no more than 0.7% from point to point along the capillaries. Capillaries were cleaned in reagent-grade acetone, concentrated HNO_3 and distilled water, and were filled with freshly boiled distilled water immediately prior to an experiment.

High Pressure Chamber. Three capillaries were mounted parallel to each other about 1 cm apart near the front of a high-pressure chamber, shown schematically in Fig. IIIA-2. The chamber was

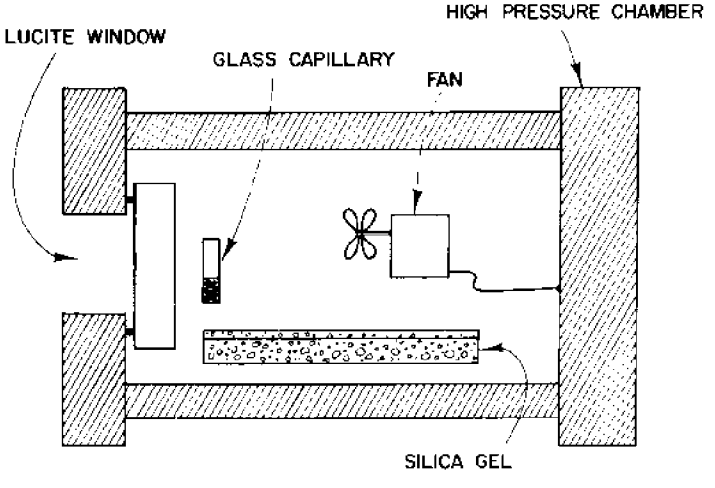

Fig. IIIA-2. Diagram of high-pressure chamber with a capillary (not to scale)

constructed of ASTM schedule 160 steel pipe, with a 1-in thick plexiglass viewing port, 3-in in diameter. Its maximal working pressure was 50 ATA. The chamber was equipped with a light source, an induction-motor fan to provide stirring, a tray containing indicating silica gel to keep P_{H_2O} in the chamber at 0, and a calibrated thermistor for measuring chamber temperature (YSI #427 thermistor and model 46 TUC telethermometer, Yellow Springs Instrument Co., Yellow Springs, Ohio). Pressure within the chamber was measured with a direct-drive Bourdon-tube gauge, calibrated to an accuracy of 0.25% of full scale or about 2.5 lb in^{-2}, according to manufacturer's specifications (Roylyn Precision Direct Drive Gauge, 3-D Instruments, Inc., Anaheim, Cal.).

Cathetometer. Measurements of h, the length of the water vapor-gas column, were performed using a cathetometer readable to .001 cm (Precision Tool and Instrument Co., Ltd., Surrey, England). Eleven replicate determinations of distance between the top of a capillary tube and the water meniscus yielded a standard deviation of ± .0013 cm. This degree of precision is made necessary by the slow rate of water evaporation from the capillaries and hence the small differences between positions of the meniscus in time. In experiments conducted at 50 ATA of N_2, for example, h changed by less than 0.03 cm in the course of 6 days.

Gases. Commercially available He and N_2 of high-purity grade were used. Manufacturer's specifications list high-purity He as 99.995% He and high purity N_2 as 99.99% N_2. He-O_2 mixtures were prepared from high purity He and 99.6% O_2 in our laboratory and analyzed either with a Beckman OM-11 oxygen analyzer (Beckman Instruments, Inc., Fullerton, Cal.) or with a Quintron model R gas chromatograph (Quintron Instrument Co., Inc., Milwaukee, Wis.)

Temperature. The pressure chamber was placed in a constant temperature chamber (model 1247LA, Hotpack Corp., Philadelphia, Penn.) which was capable of maintaining a set temperature to about ± 0.2°C. The temperature within the high pressure chamber itself showed even smaller fluctuations, of the order of ± 0.02°C, because of its large thermal mass. Temperatures were measured with YSI thermistors calibrated against precision mercury thermometers with 0.05°C scale divisions. As Schwertz and Brow (10) pointed out, evaporation may cool the gas-water interface significantly below the ambient temperature in the high-pressure chamber if it proceeds too fast, and thus may lead to erroneously low values of D. Evaporation is accelerated as the liquid level is brought closer to the open end of the capillary. In the present experiments, the liquid level was kept at least 6 cm below the open end of the capillaries for all experiments conducted at 1 ATA, where evaporation is most rapid. During an experiment in which water vapor was diffusing into He, the actual temperature difference between the gas-water interface and the bulk water phase was measured with a copper-constantan thermocouple made of .005-in wire, a size small enough not to occupy any significant fraction of

the surface area of the capillary. The voltage output of the thermocouple was measured with a Hewlett-Packard model 419A null microvoltmeter; the temperature difference recorded in this fashion was less than 0.01°C.

Sources of Error. One possible source of error lies in the assumption that diffusion of water vapor out of the capillary is the rate-limiting process, i.e., that removal of water vapor from the mouth of the capillary by convection is sufficiently rapid to maintain $P_{H_2O} = 0$ at this point. Schwertz and Brow (10) checked this assumption by measuring diffusion coefficients at several volumetric flow rates in their apparatus from 50-300 $cm^3\ min^{-1}$ and found no influence of flow in this range. In the present experiments, convective mixing was produced routinely within the high-pressure chamber by a small fan which caused a linear flow velocity near the tops of the capillaries of 100 $cm\ sec^{-1}$ at 1 ATA. The binary diffusion coefficient of water vapor-helium was measured under this type of convective mixing and also with a 500 $cm^3\ min^{-1}$ flow of helium from a pressure cylinder directed at the tops of the capillaries. The two experiments gave values of $\underline{D}$ which agreed within 3.8%.

Possible errors in cathetometer readings caused by thermal expansion of contraction of water attendant on temperature fluctuations of the order of those which occurred during diffusion experiments were calculated and found to be negligible. Similarly, although some vapor pressure lowering is to be expected as He or N_2 under high pressure dissolve in water, the fractional lowering of water vapor pressure caused by dissolution of N_2 at 50 ATA, for example, is less than 0.1%. Pressure per se increases water vapor pressure at a given temperature, but again, the effect is slight. For example, it may be calculated that the equilibrium vapor pressure of water is increased at 50 ATA by about 3% over its 1 ATA value (11).

Results

Diffusion measurements were made at 25°C on the binary systems $H_2O_{(g)}$-He and $H_2O_{(g)}$-N_2 at approximately 1, 4, 10, 20, and 50 ATA. Diffusion of water vapor in the ternary system $H_2O_{(g)}$-He-O_2 was also measured in gas mixtures of 1.6% O_2-98.4% He and 26% O_2-75% He. Data were obtained in the form of values of h vs t, and for binary systems were plotted as $(h^2 - h_o^2)$ vs $(t - t_o)$ for each of 3 diffusion tubes at each pressure and gas combination. Linear least-squares regression analysis was performed on the data from each capillary, and the three slopes so obtained were averaged before calculation of $\underline{D}$ by Eq. 2. The data fall very closely on each regression line, as shown in Fig. IIIA-3, a representative plot for $H_2O_{(g)}$-N_2 at 1 ATA. Correlation coefficients were never less than 0.997 and averaged 0.999 over all experiments. Plots of $(h^2 - h_o^2)$ for $H_2O_{(g)}$-He and $H_2O_{(g)}$-N_2 at pressures of 1, 4, 10, 20, and 50 ATA

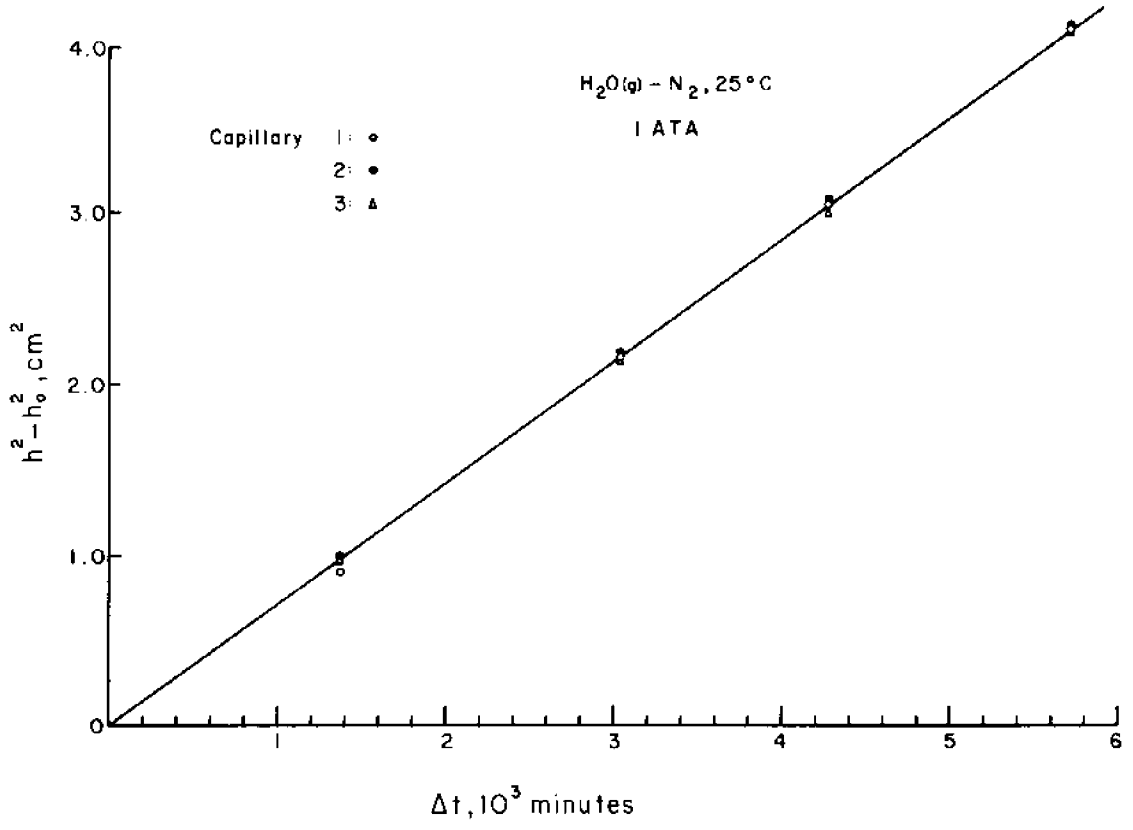

Fig. IIIA-3. $(h^2-h_o^2)$ in cm^2 vs elapsed time Δt in units of 10^3 min. Water vapor diffusion into N_2 at 1 ATA, 25°C. Each symbol represents a separate capillary.

are shown in Figs. IIIA-4,5, respectively. The reduction in slope with increasing pressure is immediately evident. Corresponding binary diffusion coefficients are given in Table IIIA-1. The diffusion coefficients listed at 1 ATA in Table IIIA-1 were actually obtained at prevailing atmospheric pressure (P_B), which deviated from 760 by 4-5 Torr at most over several months; these diffusion coefficients were then corrected to 1 ATA by multiplying by $P_B/760$. Estimates of the standard deviation in $\underline{D}$ in Table IIIA-1 were obtained by combining standard deviations obtained from the linear regression analysis for the 3 individual slopes in each experiment, according to rules for propagation of error (1).

For comparison, Schwertz and Brow (10) list experimental values of $\underline{D}H_2O,He$, and $\underline{D}H_2O,N_2$, at 34°C and 1 ATA of 0.902 cm^2 s^{-1} and 0.256 cm^2 s^{-1}, respectively; Lee and Wilke's (6) value of $\underline{D}H_2O,He$, at 25°C is 0.908 cm^2 s^{-1}. Data from the present experiments are in good agreement with these numbers.

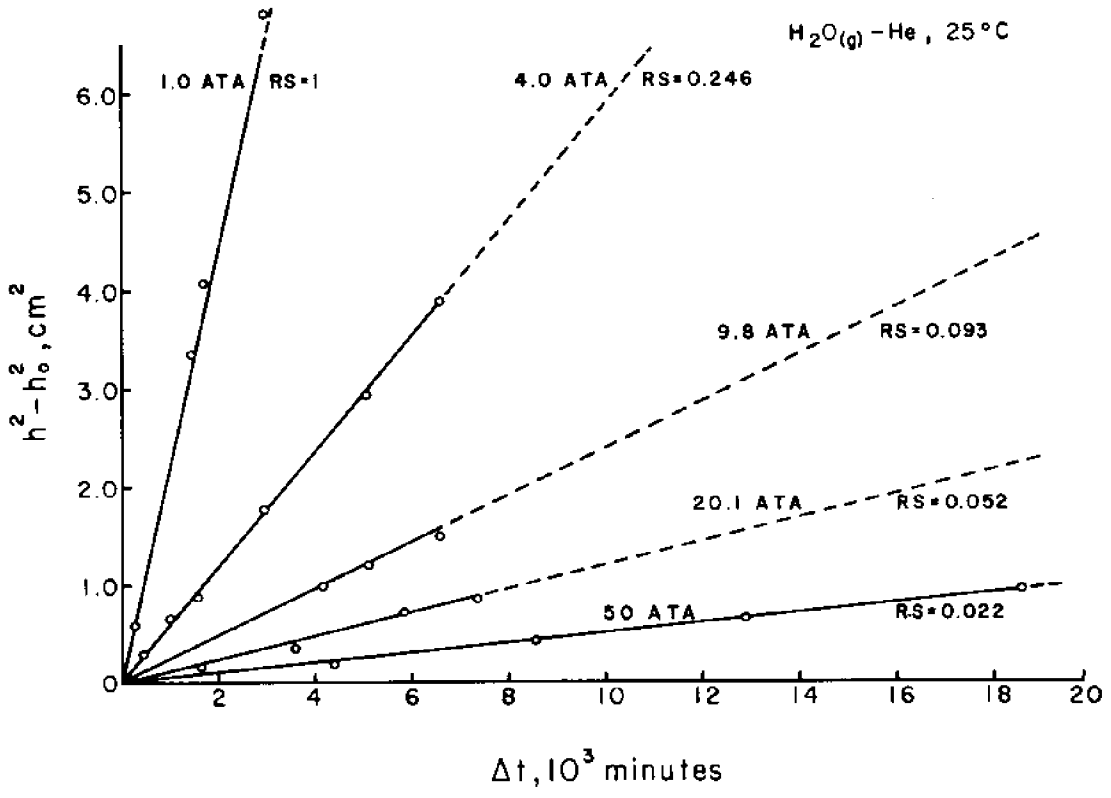

Fig. IIIA-4. $(h^2-h_o^2)$ in cm^2 vs elapsed time Δt in units of 10^3 min. Water vapor diffusion into He at the indicated pressures. RS = slope relative to that at 1 ATA.

The last column in Table IIIA-1 gives the ratio of $\underline{D}_o$, the value of $\underline{D}$ at 1 ATA, to $\underline{D}$ at the experimental pressure. According to Chapman-Enskog theory this ratio at moderate pressures (atmospheres in multiples of tens) should be equal to P/P_o, or simply to P, since P_o = 1 ATA. Figure IIIA-6 is a graph of $\underline{D}_o/\underline{D}$ vs P/P_o. Experimental data are plotted as points, and the line of identity is drawn. It is clear that $\underline{D}_o/\underline{D}$ deviates from Chapman-Enskog theory above 4 ATA for H_2O-N_2, and above 20 ATA for H_2O-He; the deviations are more pronounced in N_2 than in He, and become larger at the higher pressures. Comparison of the He and N_2 data at the same pressure shows that water vapor diffuses from 2.5 to 3.3 times more readily through He than through N_2, depending on the pressure.

In the ternary system H_2O-He-O_2, flux of water vapor out of the capillary tube was used to characterize the diffusion process, since it is not in general possible to assign a single diffusion coefficient to a system involving three gases (2). For ternary

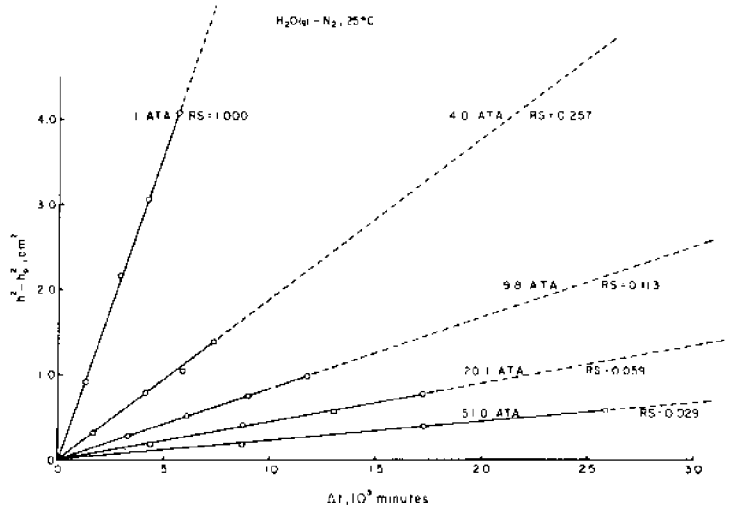

Fig. IIIA-5. $(h - h_o)$ in cm vs elapsed time Δt in units of 10 min. Water vapor diffusion into N_2 at the indicated pressures. RS = slope relative to that at 1 ATA.

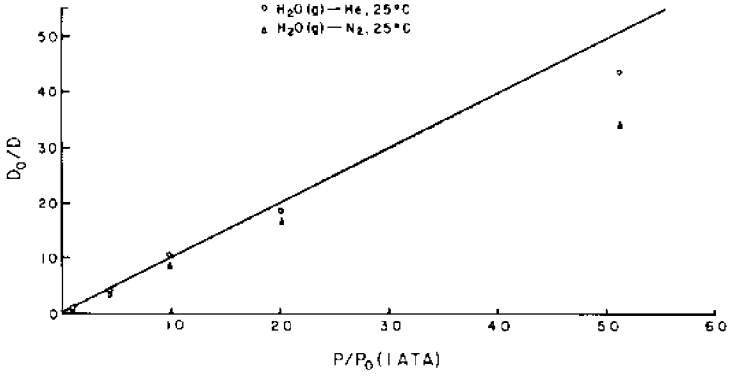

Fig. IIIA-6. $\underline{D_o}/\underline{D}$, ratio of binary diffusion coefficient at 1 ATA to that at experimental pressure P, vs P/P_o where P_o = 1 ATA. Points are experimental values; line of identity has been drawn.

Table IIIA-1. Binary diffusion coefficients at 25°C. D_o/D = ratio of binary diffusion coefficient at 1 ATA to that at experimental pressure.

Gas System	Pressure, ATA	D, cm^2s^{-1}, ± % SD	D_o/D
$H_2O(g)$-He	1.00	0.836 ± 0.2%	1.00
$H_2O(g)$-He	4.06	0.209 ± 0.8%	4.00
$H_2O(g)$-He	9.85	0.0849 ± 1.5%	9.85
$H_2O(g)$-He	20.12	0.0444 ± 2.0%	18.8
$H_2O(g)$-He	50.88	0.0188 ± 0.9%	44.5
$H_2O(g)$-N_2	1.00	0.253 ± 0.4%	1.00
$H_2O(g)$-N_2	4.10	0.0672 ± 1.5%	3.76
$H_2O(g)$-N_2	9.84	0.0297 ± 0.8%	8.52
$H_2O(g)$-N_2	20.05	0.0159 ± 1.3%	15.9
$H_2O(g)$-N_2	51.01	0.00749 ± 2.6%	33.8

% SD values determined as described in text.

systems h was plotted directly against t, as shown in Fig. IIIA-7. Good straight line fits to the experimental data were obtained by linear regression analysis, and dh/dt, the slope of the line, was determined. Correlation coefficients averaged 0.999 over all experiments. To a close approximation, dh/dt is a direct measure

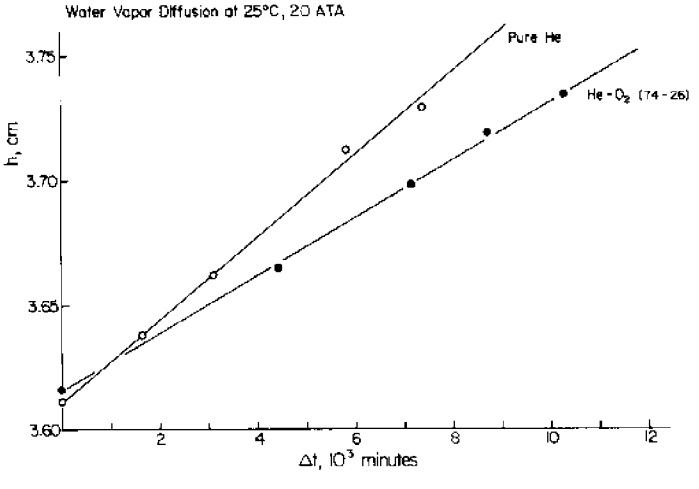

Fig. IIIA-7. h in cm vs elapsed time Δt in units of 10^3 min for binary diffusion (pure He) and ternary diffusion (He-O_2 in a 74%-26% mixture) of water vapor at 20 ATA.

of the water vapor flux out of the tube per unit cross-sectional area. Because part of the liquid water which evaporates remains behind in the tube, $^{dh}/_{dt}$ does not measure the flux exactly. However, the amount of water vapor remaining behind is several orders of magnitude less than the quantity which evaporates, and can be neglected in the present analysis. Table IIIA-2 lists ternary fluxes computed as described above, with values of the corresponding binary fluxes in water-vapor-He and ternary/binary flux ratios given for comparison. When O_2 is present at a concentration of 26% by volume, water vapor flux is reduced in the ternary system to between 60- and 70% of its value in pure helium. At 1.6% O_2 by volume there is very little effect on the water vapor fluxes. Standard deviations of the fluxes were calculated in the same fashion as described for the diffusion coefficients.

Table IIIA-2. Water vapor fluxes at 25°C for the ternary system $H_2O(g)$-He-O_2 at indicated compositions (% by volume), and the binary system $H_2O(g)$-He.

Gas System	Pressure ATA	Water Vapor Flux, $(g \cdot s^{-1} \cdot cm^{-2}) \cdot 10^6$		Ternary Flux / Binary Flux*
		ternary	binary*	
$H_2O(g)$, 74% He-26% O_2	1	1.86 ± 1.7%	2.99 ± 0.5%	0.62
	4	0.49 ± 2.1%	0.79 ± 2.4%	0.62
	20	0.19 ± 1.0%	0.28 ± 2.1%	0.68
$H_2O(g)$, 98.4% He-1.6% O_2	1	2.94 ± 1.2%	2.99 ± 0.5%	0.98
	20	0.27 ± 1.4%	0.28 ± 2.1%	0.96

Values are % SD, determined as described in text.
*Binary fluxes have been computed for the system $H_2O(g)$-He at the same pressure as the corresponding ternary flux.

Discussion

Results of the present investigation show that the diffusivity of water vapor in He varies closely as 1/P, at least up to 10 ATA. However, by the time 20 ATA has been reached, reduction in $\underline{D}H_2O,He$, has not kept pace with increasing P. In N_2, deviation from direct proportionality between $\underline{D}H_2O,N_2$, and 1/P is already apparent at 4 ATA, and grows greater with increasing pressure. At 50 ATA, $\underline{D}H_2O,He$, is 13% larger than expected (0.0188 $cm^2\ s^{-1}$ by actual measurement vs 0.836/50.88 or 0.0164 $cm^2\ s^{-1}$ predicted); $\underline{D}H_2O,N_2$, is 34% larger (0.00749 $cm^2\ s^{-1}$ measured vs 0.253/51.01 or 0.00496 $cm^2\ s^{-1}$ predicted).

Deviations of binary diffusivity from 1/P dependence at high pressure are not unique to systems containing water vapor. Durbin and Kobayashi (3) have shown a similar phenomenon in the pressure dependence of krypton-85 diffusion in krypton, argon, nitrogen, helium, and carbon dioxide. Their deviations are reported in the form of increasing values of the product (gas diffusivity × density) with increasing pressure and thus are in the same direction as those noted in the present study, though smaller in magnitude for comparable pressure changes. It is apparent that the simple inverse relation between $\underline{D}$ and P for systems containing water vapor begins to break down at quite modest pressures, and at 50 ATA, a pressure already achieved in saturation diving, can be seriously in error, at least for N_2-containing gas mixtures.

Our data on reduction in $\underline{D}H_2O,N_2$, with pressure provide a qualitative explanation for the findings of Paganelli et al. (7) on pressure-induced changes in water loss from eggs, which was mentioned previously. Those findings also showed the reduction in water loss rate with pressure to be less than expected on the basis of Chapman-Enskog theory for dilute gases.

The deviations from Chapman-Enskog theory for dilute gases observed in the present experiments are not surprising in view of the assumptions on which the theory rests: that the diffusing molecules are monatomic, spherically symmetrical, and non-polar, and undergo only binary, elastic collisions (4). In approaching problems involving diffusion of real gases at pressure, it would seem best to rely on measured values of diffusion coefficients wherever possible.

Our measurements of water flux in the ternary system H_2O-He-O_2 showed several features of interest. Introduction of 26% O_2 by volume into a water-vapor-He system reduced the water vapor flux by nearly 40% at 1 and 4 ATA, and by slightly less at 20 ATA. When the O_2 was reduced to 1.6%, on the other hand, its effect on water vapor fluxes was negligible both at 1 and 20 ATA. From our limited data, reduction of fluxes with pressure in a ternary system of given composition is seen to follow an approximate dependence on 1/P, but again, the reductions are less than predicted at 20 ATA. We have obtained results at only two O_2 concentrations; the exact nature of the dependence of water vapor flux on O_2 concentration in a ternary system awaits further investigation.

The information which we have obtained in the present investigation points to several factors which may assist us in explaining quantitatively the changes which Hong (5) has observed in human insensible water loss at increased ambient pressure.

The first is the retarding effect which pressure per se exerts on the diffusion process by increasing the numbers of molecules in the diffusion path. The fact that pressures in the range of 20-50 ATA (and above) will have proportionately less effect on gas diffusion

than lower pressures should also be considered in this connection. The molecular species of the gas in which insensible water loss occurs must also be taken into account. Finally, one must consider whether binary, ternary, or some other multi-component diffusion process is occurring.

The recent saturation dive conducted by Hong (5) and colleagues to approximately 19 ATA in 1.6% O_2-98.4% He may be used as an illustration of some of the foregoing. Preliminary analysis of the data shows that insensible water loss was reduced to 60% of the levels observed in air at 1 ATA. The most appropriate comparison from our data should be made between the binary diffusivities $\underline{D}H_2O,He$, at 20 ATA and $\underline{D}H_2O,N_2$, at 1 ATA. The ratio of these diffusivities is $0.044/0.253 = 0.17$; thus, other things being equal, the insensible loss at about 20 ATA in an approximately normoxic He-O_2 environment should be about 17% of its 1 ATA control in air. It is clear that in the saturation dive described above, "other things" were not equal. Skin temperature, ambient relative humidity, convective flow, and degree of activity can all influence insensible loss, and these factors varied significantly between the 1 ATA air and 20 ATA He-O_2 exposures. It is not surprising, then, that predictions based only on the diffusive behavior of water vapor should be inadequate to explain quantitatively the observed depression of insensible water loss. However, it should be possible to design future experiments in which non-diffusive factors are controlled, and under those circumstances we may look forward to a test of the diffusive behavior of insensible water loss.

ACKNOWLEDGMENT

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B. EFFECTS OF HELIUM ON PULMONARY GAS EXCHANGE: CONVECTION AND DIFFUSION: HUGH D. VAN LIEW

Based on the theories of others (1,2,3,10,12,14) this paper will explain how two processes, molecular diffusion and convective flow, may interact in the delivery of gas to a person's lungs. Breathing of helium-oxygen mixtures is the experimental technique to use for study of the diffusion/convective interplay because the light molecule, helium, has higher diffusivity than N_2 or O_2, and therefore imparts higher diffusivity to O_2 in the $He-O_2$ mixture.

Axial Dispersion

In 1915, Yandell Henderson and his co-workers (8) published a drawing which showed a cone-shaped spike of smoke moving through a glass tube, an early demonstration of what may occur in the lung airways if the convective flow is laminar. Henderson claimed that smoke began to issue from the tube when a volume of smoke equal to about a third or quarter of the tube's volume had been put in it. From the Poiseuille equation, one can calculate that in ideal laminar flow the front of new gas will begin to emerge when half this volume has been replaced. This means that if ideal laminar flow occurs, flow in a 500-ml tidal volume would extend as cone-shaped pencils through at least the first liter of lung volume. Although enough air to fill the airways to the twentieth generation of Weibel's 23-generation model (13) had been introduced, some gas would penetrate significantly closer to the alveolar membranes, to the twenty-second generation. Thus the axial dispersion of the gas in laminar flow will be greater than one might expect from the inspired volume.

Two processes can decrease the axial dispersion of inspired gas. Turbulence causes gas to mix intensely with nearby gas rather than sliding past it as in laminar flow, with the result that fresh gas moves forward as a cylinder with a blunt profile of composition vs. length rather than with the cone-shaped pencils or spikes of laminar flow.

Radial Diffusion

The second process that can cause a blunt composition profile is radial diffusion, an exchange of molecules between fast and slow-moving laminae. The result of this exchange is that while the flow continues, the fast-moving laminae become depleted of fresh gas and take on the composition of the stale gas that was present before the new gas entered, and conversely, the slow-moving laminae become enriched with the new gas that has moved out of the fast-moving laminae. Fig. IIIB-1 illustrates this blunting of the composition profile by radial diffusion.

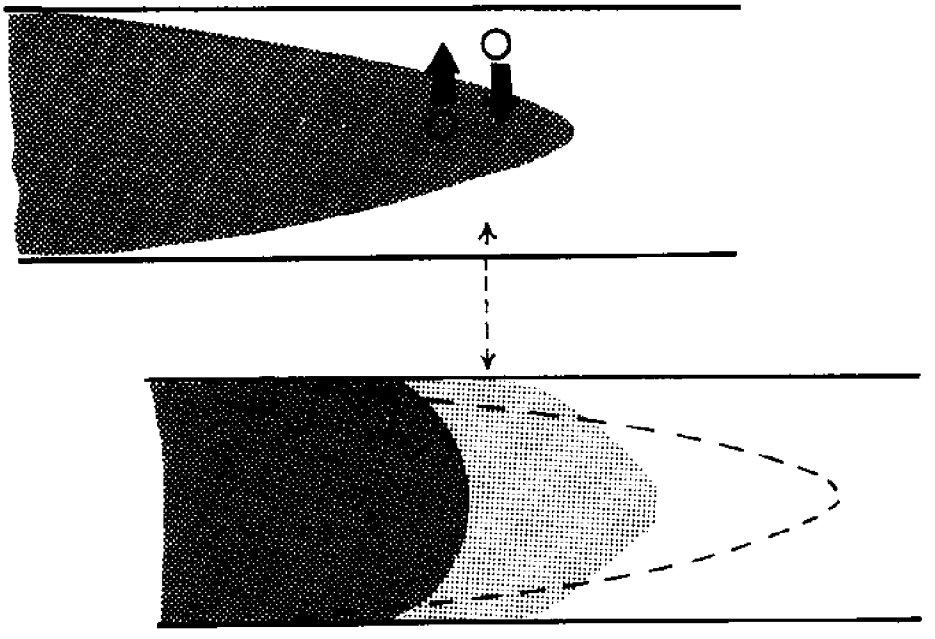

Fig. IIIB-1. Illustration of how radial diffusion blunts the profile of concentration vs. distance in laminar flow. Upper: shaded molecule diffuses out from shaded spike of fresh gas and clear molecule of stale gas diffuses in. Lower: as flow proceeds downstream, exchanges typified in upper panel have depleted fresh gas from forward end of spike. Shaded fresh gas now appears to be moving forward like a cylinder or plug which moves at average velocity of flow, not at high velocity of central fast moving laminae.

Dr. Robert Mazzone, a recent graduate of our Department in Buffalo, made several interesting observations regarding the diffusion/convective interplay while working with Drs. Farhi and Modell. Most of his work (11) will be published later, but I have Dr. Mazzone's permission to describe the model experiment shown in Fig. IIIB-2. This is a clear demonstration that radial diffusion can blunt a concentration profile. Dr. Mazzone allowed argon containing traces of other gases to flow through a plastic tube which had a mass spectrometer sampling port at the end. The tracers, helium and the dense gas SF_6, both reached the sampling point at the same time on the average, but helium showed a steeper rise in concentration, indicating a blunted profile and decreased axial disperson.

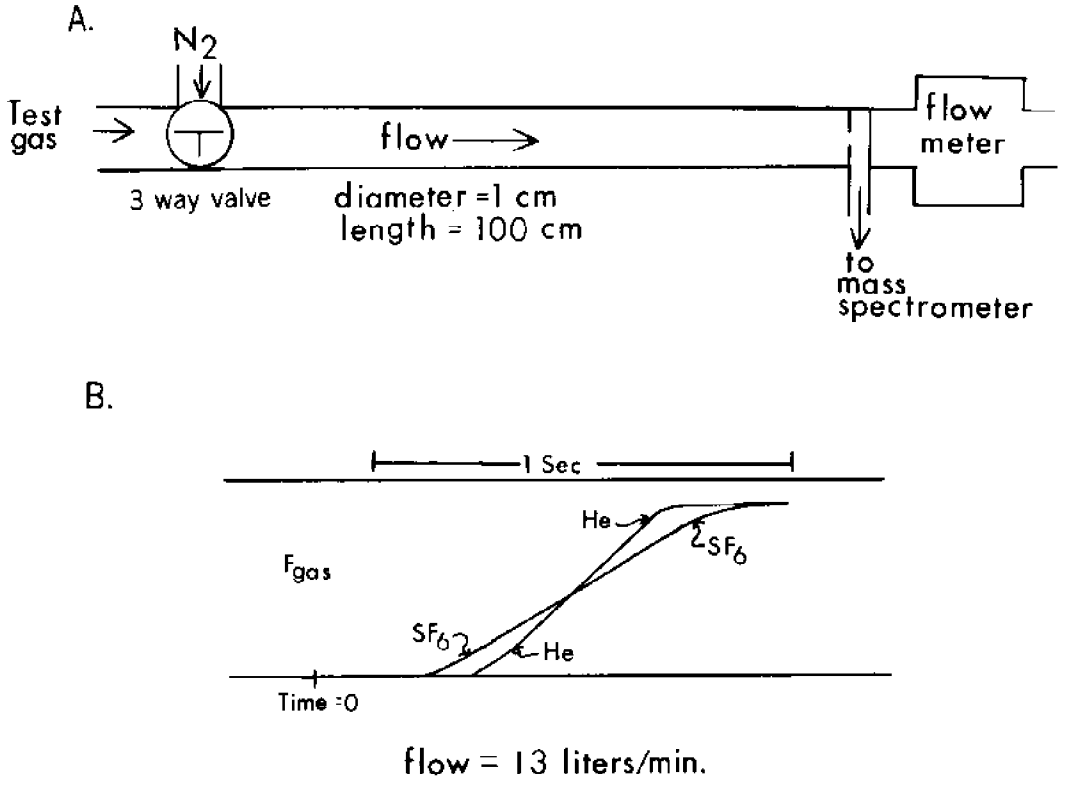

Fig. IIIB-2. Model experiment to prove that radial diffusion blunts concentration vs. distance profile of gas in laminar flow, from work of Mazzone (11). Above: preparation. Below: output of mass spectrometer.

Despite the appeal of the smoke and tracer gas demonstrations, further experimentation is needed to determine whether the phenomena of axial dispersion and radial diffusion have any relation to gas exchange in the lungs. These phenomena do not occur in turbulent flow, and even in regions where flow is laminar the airway system is more complicated than the straight tubes from which simplified descriptions have come.

Breath-Hold Experiments

Molecular diffusion is vital to transport of O_2 from air to blood; oxygen must diffuse in an axial direction to the alveolar membrane from wherever in the lung the high O_2 in a breath is delivered. However, we have seen that high diffusivity of a gas may blunt the incoming front of fresh gas, and thereby impede gas exchange.

Dr. Larry Johnson and I published data supporting this view of the good/bad nature of high diffusivity (9). Anesthetized paralyzed dogs were ventilated by a pump. Arterial PO_2 was continually monitored

with a flow-through O_2 electrode arrangement. Fig. IIIB-3 is a reproduction of a typical record, reading from left to right. Note that the PO_2 of the breathing mixture was purposely high so that complications of blood HbO_2 carriage would be avoided.

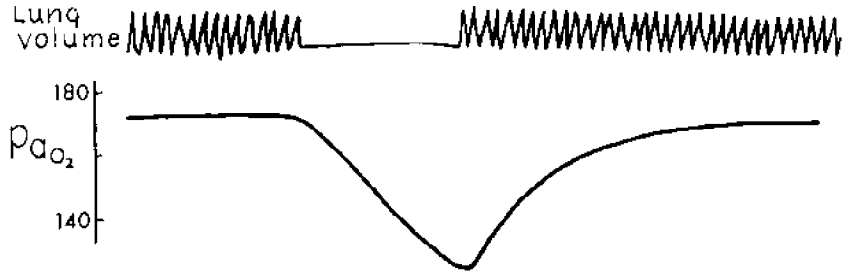

Fig. IIIB-3. Arterial PO_2 in an anesthetized paralyzed dog during a breath hold in the expiratory position, from Johnson and Van Liew (9). Upper trace indicates breaths and time of breath hold.

When the pump was stopped, PO_2 in the lung and blood fell, and when the pump was turned on again, PO_2 rose. To compare $He-O_2$ mixtures with N_2-O_2 mixtures two measurements were made, the steepness of the downslope and the rate of oxygen washin or rise. It was found that the downslope was less steep with $He-O_2$ mixtures, as if the pool of O_2 available for uptake into the blood was greater. This is interpreted as an indication that helium facilitated the diffusion of O_2 in the exchange units.

In contrast to the finding with the downslope, helium appeared to hinder O_2 transport during the washin. The rise of the PO_2 after turning on the ventilatory pump was slower with He, apparently because of blunted profiles of incoming O_2 in the airways.

Inspiratory Breath Holds

In the experiment described above, the breath hold was done at the end of expiration so the facilitative effect of helium during the breath hold was on exchange within the alveolar gas; the fresh gas front had been expired and the airways were filled with gas that was approximately the same as that within the exchange units. In a different experiment (4), Dr. Kenneth Bondi and I observed the effect of helium on interchange between the exchange units and fresh gas in the airways by studying breath holds in the inspiratory position. The experimental situation was very similar to that outlined above. Anesthetized paralyzed dogs were ventilated by pump, and a continuous sample of arterial blood was analyzed for pH as an indicator of CO_2. Figure IIIB-4 shows a typical record. When the pump was stopped in the inspiratory position, pH rose to a higher, more alkaline level than it had during previous uninterrupted breaths.

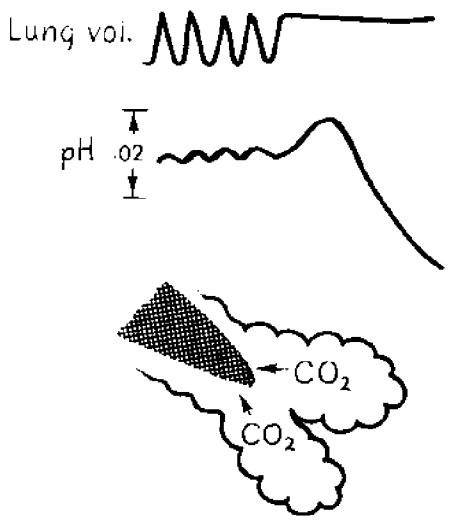

Fig. IIIB-4. Arterial pH during a breath hold in the inspiratory position, from Bondi and Van Liew (4). Hump indicates that with increased time of contact between fresh air and exchange-unit air, there is more CO_2 exchange than in ordinary preceeding breaths.

For lack of a better term, we call the pH rise a hump. Apparently some time-dependent process was facilitating gas exchange during the inspiratory breath hold. When an He-O_2 mixture was used instead of air to ventilate the dog, the hump seemed slightly bigger and a little prolonged. I am amused that we weren't more startled by this result before. Why should helium, which promotes diffusion, have so little effect on this gas exchange between fresh airway gas and the stale exchange unit gas? From the work of Engel, Macklem and associates (5,6,7), it now appears clear that the beating of the heart has such a major effect on gas mixing in the airways, and presumably between airways and exchange units also, that diffusion is relatively unimportant.

In summary, we can make three statements which relate the diffusion/convection interplay to problems of diving. a) At one atmosphere pressure, the effect of helium breathing on gas exchange is hard to predict. Enhanced radial diffusion in the airways is expected to decrease the axial dispersion of inspired gas, but improved diffusion within the exchange units may make up for the disadvantage. b) In a hyperbaric environment, molecular diffusion is decreased. Therefore, by the same reasoning as in a), gas exchange with N_2-O_2 mixtures at hyperbaric pressures will be influenced by opposing tendencies; less radial diffusion will improve axial dispersion, but poorer diffusion within the exchange units will be a hindrance. c) Gas exchange in He-O_2 mixtures

in hyperbaric environments will tend to be like exchange in N_2-O_2 mixtures at normal pressure. The decrease of diffusivity due to pressure will tend to counterbalance the naturally high diffusivity of helium.

Summary

Molecular diffusion and laminar convective flow apparently interact in the airways in such a manner that highly diffusible gases, e.g., helium, or O_2 in helium-oxygen mixtures, tend to penetrate the airways poorly. In anesthetized paralyzed dogs, O_2 and CO_2 exchanges with animals breathing He-O_2 mixtures were compared to exchanges with N_2-O_2 mixtures. The results showed that helium facilitated the gas exchange during a breath hold in the expiratory position, hindered O_2 washin into the lungs, and had little effect during a breath hold in the inspiratory position. These findings were explained by the ideas that diffusion within the exchange units was improved with helium, but that radial diffusion in the airways prevented fresh He-O_2 from penetrating deeply into the lungs, and that agitation by the heart, not diffusion, plays the major role in gas mixing between airways and exchange units.

ACKNOWLEDGMENT

This study was supported in part by National Institutes of Health Grant 5 P01 HL 14414.

Note

A paper by P. A. Kvale, J. Davis, and R. C. Schroter, "Effect of gas density and ventilatory pattern on steady-state CO uptake by the lung," Respiration Physiol. 24:385-398, 1975, provides additional experimental evidence that radial diffusion can be an important hindrance to gas exchange. Carbon monoxide uptake in man was less with low inspired volumes, less in helium than in SF_6, and less with low inspiratory flow rates. Apparently the axial penetration depended on the depth of the breath but also was less when diffusivity and time for diffusion in the flow stream were high.

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C. EFFECT OF GAS DENSITY ON O_2 UPTAKE IN MAN DURING INCREASING WORK LOADS: TERENCE O. MOORE AND D. G. WATT

Man's exposure to hyperbarism has become more frequent, and various diluent inert gases have been utilized to avoid density and/or narcotic effects. As interest in human work performance in these environments has increased, so has research on the effects of the physical characteristics of diluent gases on work performance.

As gas density increases under hyperbaric exposure, both the ventilatory and gas exchange functions of the lung are altered (3, 5,7,8). Other density factors which increase thermal conductivity of the medium have recently been reviewed by Webb (10), and are not considered here.

Opinions about oxygen delivery from lung to blood with altered gas densities have varied ranging from no change at 1 ATA vs. 4 ATA on N_2-O_2 (6), to increased O_2 uptake as density increased at 1 ATA (5,8,12).

Van Liew concludes, in a series of experiments, that while increased oxygen diffusivity should occur in a less dense carrier gas, the possibility of increased radial vs. axial diffusion may be disadvantageous to overall oxygen delivery to blood.*

The present experiment attempts to determine $\dot{V}O_2$ characteristics attributable to inspired gas densities in a situation of controlled and relatively high work output and O_2 requirement.

Methods

Experiment I. Ten subjects performed exercise on a Quinton-Monark bicycle at 300, 600, and 900 $kpm \cdot min^{-1}$ at 1 ATA on either compressed normoxic nitrogen or helium/oxygen. All trials took place in an environmental room maintained at $27^{o}C \pm 5^{o}$, and replicate trials were averaged. The breathing rig in all cases was a 2-stage, double-hose assembly (U.S. Divers, Santa Ana, California). In a given experiment, the subjects either breathed the helium or the nitrogen mixture and were not tested more than once in any week.

After resting measurements, each subject pedaled at each load for a minimum of five minutes, the last two of which were used to determine heart rate (HR), respiratory rate (RR), minute ventilation ($\dot{V}E$), O_2 consumption ($\dot{V}O_2$) and CO_2 production ($\dot{V}CO_2$). Expired and inspired gases were analyzed on a Beckman OM-11 oxygen meter and LB-1 CO_2 meter, and compared with the method of Scholander (9) in all cases.

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*See his paper in this Symposium.

Experiment II. In this case, work load was constant (discussed below) at the 900 kpm·min^{-1} level, and was maintained for 20 min. In addition, normoxic argon-O_2 was added as an inspired gas and the mix to the regulator was randomly altered (via 3-way valve) at 5-min intervals during the 20-min exercise period. Six male subjects (including 4 from Experiment I) were chosen, based upon their ability to maintain the work load for the required period of time. All were experienced bike riders, and no difficulties were encountered. An rpm level of 50 was maintained by metronome pacing.

Statistical inferences were made from paired-t tests in both experiments.

Results

Experiment I. The basic measurements with calculated tidal volume (VT) appear in Table IIIC-1. $\dot{V}E$ was not different until the highest work level was reached, where He-O_2 ventilation was significantly ($P < 0.02$) higher than for N_2-O_2 breathing. This apparently was the result of a slightly higher RR ($P < 0.02$) rather than VT, an unusual finding for rhythmic work. $\dot{V}O_2$ was depressed breathing He-O_2 ($P < 0.02$) at all levels above resting. This is shown by the ventilation equivalent curve in Fig. IIIC-1, which demonstrates the

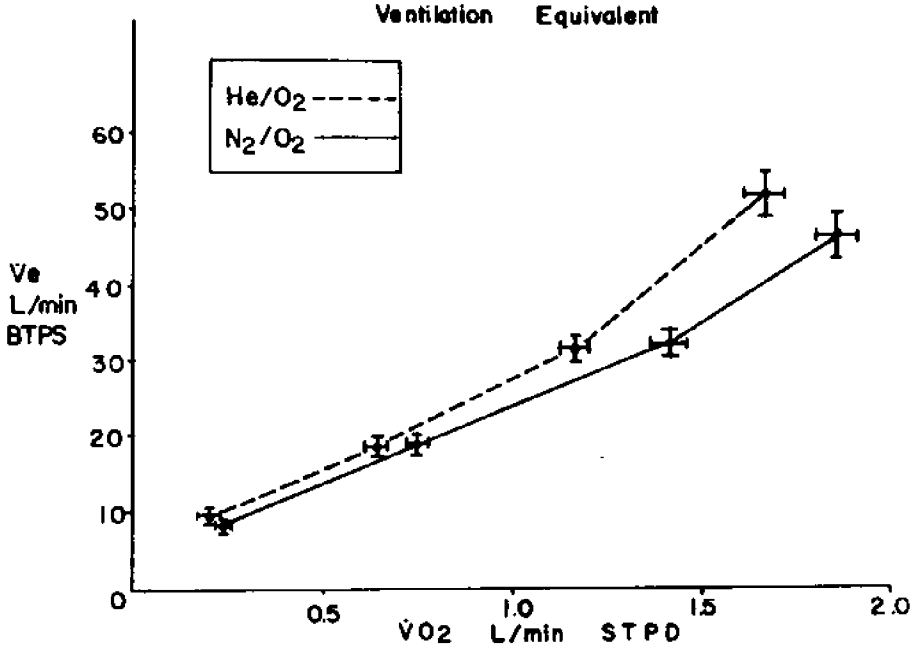

Fig. IIIC-1. Ventilation equivalent for increasing work load breathing normoxic He-O_2 or N_2-O_2 at 1 ATA.

Table IIIC-1. Ventilatory and gas exchange measurements breathing $He-O_2$ or N_2-O_2 at rest and during exercise at 300, 600, and 900 $kpm \cdot min^{-1}$

	N_2-O_2				$He-O_2$			
	Rest	300	600	900	Rest	300	600	900
$\dot{V}E$, liter/min BTPS	8.01 ± 0.29	18.97 ± 0.89	32.33 ± 1.41	46.61 ± 2.95	9.52 ± 0.43	18.58 ± 0.59	31.95 ± 1.47	52.41 ± 2.97
$\dot{V}O_2$, liter/min STPD	0.233 ± 0.013	0.749 ± 0.03	1.418 ± 0.064	1.861 ± 0.057	0.197 ± 0.026	0.633 ± 0.027	1.161 ± 0.045	1.672 ± 0.053
$\dot{V}CO_2$, liter/min STPD	0.214 ± 0.009	0.709 ± 0.030	1.390 ± 0.060	2.001 ± 0.090	0.255 ± 0.019	0.645 ± 0.023	1.270 ± 0.058	2.019 ± 0.101
Respiratory rate, breath/min	12.2 ± 1.1	15.2 ± 1.5	18.7 ± 1.9	24.4 ± 2.6	13.5 ± 1.3	16.6 ± 1.6	20.4 ± 2.1	27.6 ± 2.9
VT, liter	0.696 ± 0.053	1.350 ± 0.133	1.860 ± 0.173	2.057 ± 0.188	0.781 ± 0.091	1.210 ± 0.109	1.680 ± 0.156	2.040 ± 0.172
Heart rate, beats/min	73.1 ± 2.6	97.4 ± 3.1	123.4 ± 5.2	147.9 ± 6.7	71.5 ± 4.3	100.6 ± 3.6	127.3 ± 6.4	154.0 ± 6.9

Values are means, ± 1 SE.

relatively poor O_2 extraction during work on He-O_2. Heart rate was higher ($P < 0.01$) with He breathing at the high load.

Experiment II. The higher work load was chosen for this study because differences seen in Experiment I appeared greatest under this condition. Table IIIC-2 contains the measured values. Again,

Table IIIC-2. Ventilatory and gas exchange data at work load of 900 kpm·min^{-1} breathing normoxic He-O_2, N_2-O_2, and Ar-O_2.

	He	N_2	Ar
$\dot{V}E$, liter/min BTPS	63.5 ± 3.6	50.5 ± 3.4	54.1 ± 5.5
$\dot{V}O_2$, liter/min STPD	1.320 ± 0.090	2.215 ± 0.060	2.567 ± 0.150
$\dot{V}CO_2$, liter/min STPD	2.841 ± 0.102	2.266 ± 0.099	2.444 ± 0.085
Respiratory rate, breaths/min	23.5 ± 2.1	18 ± 1.9	19.2 ± 2.0
VT, liter	2.702 ± 0.130	2.806 ± 0.190	2.818 ± 0.180
Heart rate, beats/min	155 ± 3.8	135 ± 4.7	145 ± 7.1

Values are means, ± 1 SE

$\dot{V}E$ was highest in the He-O_2 breathing, significantly different ($P < 0.02$) from both N_2-O_2 and Ar-O_2. The latter were not different. $\dot{V}O_2$ varied directly and linearly with the density of breathing gas mix, lowest with He-O_2 and highest with Ar-O_2. $\dot{V}CO_2$ was much more variable in this experiment and was higher than that found in the previous experiment with He and N_2. Respiratory rate was again higher breathing He-O_2 ($P < 0.02$) and no difference was found between the other two mixes. VT was not altered in any case, but was higher than found in Experiment I. Heart rate was higher ($P < 0.05$) with He and not different on N_2 vs. Ar.

Discussion

Common denominators in both experiments are the elevation of ventilation on low density He and the enhanced O_2 uptake with increasing

density. Wood et al. (12) had reported that ventilation was more uniformly distributed when relatively high flow rates of dense gases were breathed, and Saltzman et al. (8) suggested that increased gas density may improve gas exchange by favorably altering ventilation/perfusion relationships. In 1969, Lanphier (5) reported that the distribution and mixing of inspired argon was more uniform in subjects at 7 ATA than at 1 ATA.

Other studies on breathing of dense gases, in man (7) and dog (6), have shown the enhanced uptake of O_2 with increasing density and work load.

An attractive hypothesis for improved O_2 uptake (1,11) is that increased density helped to abolish stratification via better convective mixing in the airways. This allows deeper penetration into lung exchange units, and results in shorter diffusion distances. While it is still debatable that the decrease in gas diffusivity can be offset by the shorter diffusion distance, Farhi (3) is of this opinion for the hyperbaric environment.

Interestingly, the He-O_2 ventilation equivalent curve found in this study coincides quantitatively with one reported by Dejours (2) for exercise under a variety of conditions. In his study, this curve bore the label "acute hypoxia."

In summary, this study demonstrates that within density ranges from 1 ATA He-O_2 (0.389 g/liter at 27°C) to an equivalent He-O_2 depth of approximately 4 ATA (1.558 g/liter) or 1 ATA Ar-O_2, high O_2 consumptions are supported by increasing density, while decreases in ventilation tend to level off. The data are consistent with the view that increased convective mixing does indeed offset decreases in diffusivity that might occur at work loads and densities seen by the working diver. If the relationship holds at higher densities, the hyperoxic environment generally provided for the habitat diver might be reduced without incurring important performance decrement.

Summary

A total of 14 fit male (n = 10) and female (n = 4) subjects performed bicycle work at 300, 600, and 900 kpm·min^{-1} at 1 ATA breathing normoxic helium-O_2 or air, and at 900 kpm·min^{-1} on normoxic argon-O_2 (n = 6). Minute ventilations ($\dot{V}E$) and O_2 uptakes ($\dot{V}O_2$) were compared at identical work loads to discern the O_2 uptake characteristics with different inert media as density varied. $\dot{V}E$ decreased slightly and $\dot{V}O_2$ increased significantly ($P < 0.01$) with increasing gas density, indicating a less efficient ventilation equivalent breathing helium-oxygen mixes. This effect was exacerbated at higher work loads. $\dot{V}O_2$ for the highest work load varied from 1.320 ± 0.09 liter/min breathing He-O_2 to 2.567 ± 0.15 liter/min breathing Ar-O_2, with N_2-O_2 being 2.215 ± 0.06 liter/min. Increased work of breathing did not appear to account for the increased $\dot{V}O_2$. It is suggested that, within the ranges measured here, a higher density inert gas carrier increases efficiency of O_2 uptake during work in man.

ACKNOWLEDGEMENT

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D. EFFECTS OF HELIUM ON THE CARDIOVASCULAR SYSTEM UNDER NORMOBARIC AND THERMONEUTRAL CONDITIONS: Y. C. LIN AND S. M. C. LUM

Helium-oxygen mixtures under normobaric and thermoneutral conditions are generally held to be pharmacologically inert. Physiological effects which have been thus far reported are largely attributed to their high thermal conductivity, (2,3,6,11,23), low specific density (1,15), and facilitation of diffusion of other gases, including water vapor (26,34,36). Several specific effects of helium gas have been reported and are summarized in Table IIID-1. Only those observations

Table IIID-1. Biological effects of normobaric thermoneutral helium gas

Effects	Investigators
Depresses resting heart rate in unanesthetized rat	Rhoades et al., 1967 Lin and Kato, 1974
in man	Hong et al., 1973 Flynn et al., 1972 Hu and Russo, 1972
Antiarrhythmic activity in anesthetized dog	Piferre et al., 1968, 1969, 1970 Raymond et al., 1972 Teltzis and Scott, 1972
Elevates ventricular fibrillatory threshold	Leon and Harris, 1973
Depresses circulating catecholamine	Raymond et al., 1972
Elevates circulating growth hormone during exercise	Raymond et al., 1974
Alters EEG	Hu and Russo, 1973 Cohn and Katzenelbogen, 1939
Alters metabolic pathways	Schatte et al., 1973 South and Cook, 1953
Depresses growth rate in mold	Schreiner et al., 1962
Alters $\dot{V}O_2$ and $\dot{V}CO_2$	Rinfret and Doebbler, 1971 South and Cook, 1953 Maio and Neville, 1966
Alters Na-K-Mg ATPase activity	Koehler and Gottlieb, 1972
Potentiates apneic bradycardia	Hong et al., 1973
Depresses embryogenesis	Weiss and Wright, 1968
Increases serum lactic dehydrogenase and serum glutamic oxalacetic transaminase	Ritter et al., 1969

made under normobaric and thermoneutral conditions are included, since pressure per se reportedly exerts its own biological effects, which are not thoroughly understood. The wide variety of effects and the contradictions among effects reported make generalization impractical.

Rinfret and Doebbler (24) reviewed earlier reports of the effects of helium substitution for nitrogen on tissue homogenates, tissue slices, eggs, organs, embryos and whole organisms, using species ranging from mold, bacteria, yeast, amphibians, and reptiles, to small mammals and man. Helium had little effect on oxygen consumption, CO_2 production, anaerobic glycolysis, and glycolysis. Schreiner's review included results of recent studies on the general biological effects of the helium-xenon series elements (30). He drew generalizations

concerning the depression effect of the helium-xenon series elements on the growth rates of filamentous fungus (Neurospora crassa). There was a linear correlation between the mycelial growth rate of Neurospora crassa and the square root of the molecular weight of the gas (29). He, Ne, N_2, Ar, Kr, and Xe, in that order, corresponded to the increasing growth rate depression of Neurospora crassa. This is also the exact order of narcotic potency for these gases in man.

The effects of helium breathing on the resting heart rate have also been studied. Repeated breathing of the air (25°C) and heliox (35°C) caused repetitive and reversible changes in the resting heart rate of unanesthetized rats. Helium breathing suppressed the resting heart rate in the rat approximately 10% more than air breathing (13). Helium breathing at 1 ATA depressed the resting heart rate in man where it was already relatively high (8). In another series of experiments, Lin and Kato (13) measured oxygen consumption and heart rate simultaneously in unanesthetized rats. The rats' heart rate was lower in heliox environments than in air environments at any given level of oxygen consumption. This relationship had been reported previously in the rat (23) and in men exercising in hyperbaric heliox environments, but the helium and pressure effects could not be separated in this experiment (27). The suppression of the resting heart rate by heliox breathing can be attributed to a combination of elevated vagal and depressed sympathetic activities, deduced from autonomic nervous blockades.

Specific effects of helium on the cardiovascular system have come to limited attention only in recent years. When anesthetized dogs with acute left circumflex coronary artery occlusion were ventilated with gas containing helium (20-80%), the incidence of ventricular fibrillation was drastically reduced (19). Fifty-four percent of the dogs with coronary ligation developed ventricular fibrillation while being ventilated with air, whereas only 0-8% of the dogs developed ventricular fibrillation when ventilated with gas mixtures containing 20-80% helium. The same group of investigators showed that gas mixtures containing 20% helium and oxygen sufficient to maintain arterial PO_2 greater than 140 mmHg were most effective in preventing ventricular fibrillation following coronary ligation (20). Based on flow measurements (20) and collateral studies (19), they postulated that the effect could be mediated by improved blood supply to the myocardium distal to the ligation. They suggested that the reduced fibrillatory incidence resulted from improved diffusion and rapid establishment of collateral circulation in the presence of helium in the respiratory medium.

Raymond et al. (21) showed that helium protects against cardiac fibrillation induced by acute coronary ischemia in a group of anesthetized dogs whose ventilation and body temperature were controlled. There were no differences in arterial PO_2, PCO_2, pH, mean arterial blood pressure, or the responsiveness of the cardiovascular system to exogenous epinephrine between the dogs ventilated with N_2-O_2 (75-25%)

and with He-O_2 (75-25%) mixtures. There were, however, two differences: 1) plasma potassium concentration was higher in the N_2 group than in the He group following coronary ligation; and 2) plasma catecholamine concentration tended to be lower in the He group than in the N_2 group.

Various mechanisms of the antiarrhythmic effect of helium gas during acute ischemia have been proposed: a) accelerated development of collateral circulation (19,20); b) depressed adrenergic activity indicated by lowered circulating catecholamine (21); c) prevention of elevated plasma potassium (21); d) increased minimal current required (ventricular fibrillation threshold) to induce fibrillation (12); and e) elevated arterial oxygen content (32).

Despite this evidence in support of the antiarrhythmic effect of helium gas during acute ischemia, other contradictory results have been reported. Holland et al. (7) could not reproduce the antiarrhythmic effect of helium respiration. Helium breathing did not alter the course of ouabain-induced arrhythmias in rats and mice, or epinephrine- and deslanoside-induced arrhythmias in cats, nor did it alter the outcome of coronary ligation in cats which developed ventricular fibrillation within 7 min following coronary ligation (16). Unpublished data from our laboratory support the results of Holland et al. (7) and Toltzis et al. (33) which showed the possibility of developing fibrillation following occlusion of left circumflex coronary artery (LCCA) to be only 12.5% (1 out of 8 dogs); Holland et al. reported a 9% possibility (2 out of 22 dogs), and only 5% (1 out of 19 dogs) using the closed-chest preparation (33). This contrasts with a 50% possibility reported by Pifarre et al. (19,20) and Raymond et al. (21). Our data also agrees with that of Holland et al. (7) that helium breathing did not reduce the frequency of arrhythmias in those dogs which did not go into fibrillation as a result of LCCA ligation. We found that arrhythmic activity subsided spontaneously one hour after the occlusion while under air ventilation. This feature makes it difficult to make conclusions regarding the efficacy of helium gas as an antiarrhythmic agent.

These apparently conflicting results may be due to the inappropriateness of models employed. There are inherent difficulties in the investigation of antiarrhythmic effects when one employs a model in which: 1) the level of arrhythmic activity is not reproducible from animal to animal; 2) arrhythmic activity varies with time in an unpredictable manner; 3) the induced arrhythmias are irreversible regardless of the antiarrhythmic agents or procedures used; and 4) the frequency of induced fibrillation following LCCA ligation is too low. Other models should be explored to test the efficacy of helium gas as an antiarrhythmic agent. Two such possibilities are: 1) 24-hr monitoring of subjects who display spontaneous arrhythmias while the inspiratory gas is altered periodically without their knowledge; and 2) a model in which major factors affecting the performance of the heart can be controlled and the only uncontrolled variable is arrhythmia.

This study was conducted according to the Langendorff technique, and used isolated perfused rat hearts to study: 1) the performance of the heart under constant preload, as affected by the presence of N_2 or He in the perfusate; 2) the recovery of the rhythm of the heart from total cessation of coronary flow for prolonged durations; and 3) the coronary flow under a constant perfusion pressure, and hence the coronary resistance of the coronary circulation as affected by the presence of N_2 or He in the perfusate.

Method and Materials

Eighteen male Wistar rats (average weight 434 $\pm$ 19 gm) were stunned by a blow to the head. The hearts and aortas were excised and placed in a 4°C Kreb-Henseleit solution (K-H solution). Each aorta was attached to a metal cannula and the coronary vessels were cleared with cold K-H solution immediately following the attachment of the cannula. The heart was then connected to the Anderson Perfusion Apparatus (Metro Scientific, Inc.). Perfusion temperature was maintained at 30 $\pm$ 0.5°C by a constant temperature circulating water bath (Haake, FK-10). A thermistor was located near the perfusate entering the heart, and the temperature was monitored using a digital telethermometer (United System Corp.). Contractile force was recorded isometrically under 0.5-g preload tension by using a Grass FT 0.03 force displacement transducer (Grass Instruments, Inc.). The electrocardiogram was obtained by a fine platinum electrode located at the apex of the heart with the other electrode attached to the metal aortic cannula. All recordings were made on a Beckman type-R recorder. Perfusion pressure was maintained at 90 cmH_2O and the coronary flow was obtained by timed collection of the effluent from the heart.

The physiological solution used in all experiments was Kreb-Henseleit solution with the following composition in mM/liter: NaCl, 118.4; KCl, 4.7; $CaCl_2$, 1.9; $MgSO_4$, 0.62; KH_2PO_4, 1.6; $NaHCO_3$, 25; and glucose, 11; all in distilled water. Three mixtures of gas were employed for saturation with the perfusate: 1) 95% O_2 and 5% CO_2 (control group); 2) 50% O_2, 45% N_2, and 5% CO_2 (N_2 group); and 3) 50% O_2, 45% He, and 5% CO_2 (He group). Each experiment made use of one heart and one gas composition only. The sequence of gas mixture used was random.

Measurements. Forty-five minutes were allowed for equilibration following the onset of perfusion, after which the following measurements were made at 15-min intervals: heart rate (f_h); coronary flow rate (Q); contractile force (F), time-to-peak tension (TTPT, measured from R-wave of EKG record to maximum force developed); maximal rates of force development (+dF/dt) and relaxation (-dF/dt); time-tension integral (TTI/beat, measured by determining the area under the time-tension curve for each heart beat with a planimeter), and a power index (TTI/min, i.e., f_h x TTI/beat). The entire experiment lasted 3 hr.

Antiarrhythmic activity. Upon completion of this experiment, the perfusate was turned off for 20-30 min and then the flow was re-established. In 8 experiments (5 from the control group, 2 from the N_2 group, and 1 from the He group), flow was turned off for 30 min. In 10 experiments (1 from the control group, 4 from the N_2 group, and 5 from the He group), the perfusion was stopped for 20 min. At the end of this predetermined period, the flow was re-established and the rhythmicity and mechanical activity of the heart were recorded.

Results

Oxygen contents of the perfusates which were equilibrated with 50% O_2 (He or N_2 mixture) were 1.5 vol%, which is roughly one half that of a 95% oxygen equilibrated solution. Therefore, our results would only be suitable for comparison with results of experimental groups which used 50% O_2 mixtures where the only difference was the species of inert gas, i.e., helium as compared to nitrogen. The 95% O_2-5% CO_2 mixture was used to compare the performance of the heart in the present study with results reported in the literature. The maximal value of developed force in the control group was 3.26 ± 0.48 g of force/g of wet ventricle (Table IIID-2), which compared favorably with the literature.

Table IIID-2. Performance of the isolated heart perfused with Krebs-Henseleit solution equilibrated with various gas mixtures

Measurements	A 95% O_2-5% CO_2	B 50% O_2- 45% He-5% CO_2	C 50% O_2- 45% N_2-5% CO_2
Ventricular wt, g	1.654 ± 0.144	1.467 ± 0.168	1.617 ± 0.040
Maximum tension (F)			
g	5.10 ± 0.48	6.80 ± 0.50	3.70 ± 0.50**
g/g ventricle	3.26 ± 0.48	4.83 ± 0.39	2.33 ± 0.34**
Heart rate, beat/min	163 ± 9	139 ± 11	134 ± 8
Coronary flow			
ml/min	5.63 ± 0.52	6.73 ± 0.50	7.18 ± 0.40
ml/min/g ventricle	3.50 ± 0.32	4.70 ± 0.27	4.59 ± 0.78
Max +dF/dt, g/s	183 ± 11	312 ± 25	134 ± 23*
Max -dF/dt, g/s	76 ± 10	88 ± 6	51 ± 7**
Time-tension integral, g·s/beat	0.84 ± 0.09	1.16 ± 0.13	0.66 ± 0.08**
Power index, g/s/min	134 ± 14	162 ± 9	88 ± 11**
Time to peak tension, ms	110 ± 10	115 ± 9	131 ± 6
Coronary flow resistance, mmHg/ml/min			
45 min perfusion	12.3 ± 1.1	10.1 ± 0.7	9.1 ± 0.5
90 min perfusion	15.0 ± 1.4	10.4 ± 0.9	10.9 ± 0.5
180 min perfusion	18.4 ± 2.8	10.8 ± 0.9	14.9 ± 1.2
Ventricular water content, %***	83.90 ± 0.06	83.23 ± 0.12	83.93 ± 0.47

Values are means ± 1 SE, for 6 rats in each group, weighing 434 ± 10 g; performance parameters of hearts listed were measured at time of maximal tension development (45 min after onset of perfusion) which corresponded to time 0 in Figs. IIID-1,2. * = $P < 0.05$, ** = $P < 0.01$, comparing groups B and C. ***Water content of ventricles, obtained from separate set of experiments with exact time course of previous experiments, from 9 rats (3 in each group) weighing an average of 471 ± 7 g.

Maximal developed tension (F_{max}) was reached 45 min after the onset of perfusion in all three groups, after which the F declined. Maximal values of performance indices are listed in Table IIID-2. Heart rate was not statistically different at this time, although the mean values of f_h in the N_2 group were lower than those in the He group ($0.2 > \underline{P} > 0.1$). Coronary flow was also not statistically different between the two groups. The hearts in the He group performed better than those in the N_2 group in terms of F_{max} ($\underline{P} < 0.05$), +df/dt ($\underline{P} < 0.01$), -dF/dt ($\underline{P} < 0.01$), TTI/beat ($\underline{P} < 0.01$), and TTI/min ($\underline{P} < 0.01$). TTI indicates the average tension exerted by the heart over the duration of the cardiac cycle which has no unit of work but is an indicator of work. Similarly, TTI/min is an indicator of power.

Performance of the hearts in all three groups declined after the maximal tension developed (Figs. IIID-1,2). The time designated as 0 was 45 min after the beginning of perfusion. Hearts in the control and He groups maintained their rates throughout the 3-hr experimental period, but the heart rate in the N_2 group declined steadily, and was lower than in the He group 90 min after the start of perfusion. Coronary flow in the He group was maintained at 4.4-4.6 ml/min/g of ventricle throughout the entire course of all 6 experiments, while that in the N_2 and control groups declined rapidly. Contractile force in the He group was consistently higher than either the control or N_2 group. Time-tension integral per minute, an index of rate of work (power) remained higher in the control and He groups than in the N_2 group at all times (Fig. IIID-1).

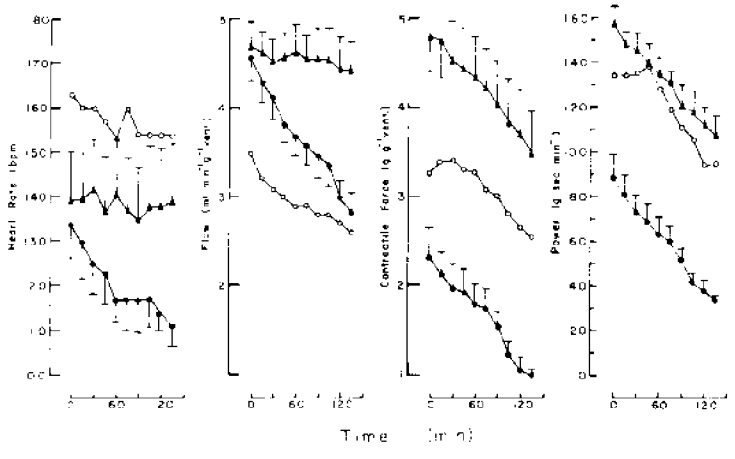

Fig. IIID-1. Heart rate, coronary flow, contractile force, and power index of isolated hearts perfused with 95% O_2-5% CO_2 (o), 50% O_2-45% He-5% CO_2 (▲), and 50% O_2-45% N_2-5% CO_2 (●), equilibrated Kreb-Henseleit solution at 30 ± 0.5°C. Time 0 is 45 min after start of perfusion. Vertical bars indicate ± 1 SE.

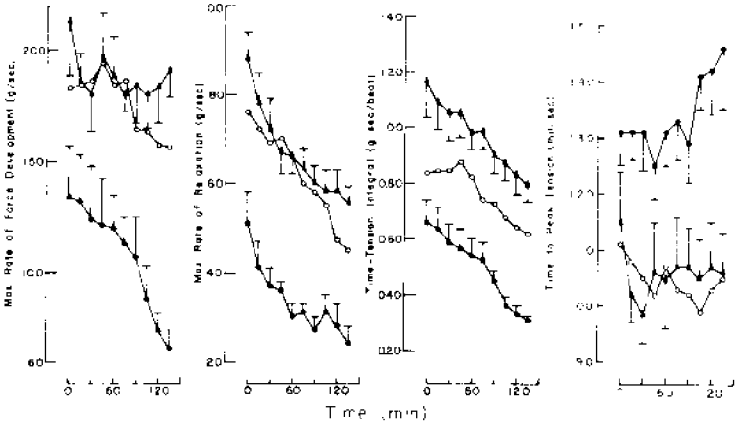

Fig. IIID-2. Maximal rates of force development and relaxation, time-tension integral, and time-to-peak tension of isolated hearts perfused with 95% O_2-5% CO_2 (o), 50% O_2-45% He-5% CO_2 (▲), and 50% O_2-45% N_2-5% CO_2 (●), equilibrated Kreb-Henseleit solution at 30 ± 0.5°C. Time 0 is 45 min after start of perfusion. Vertical bars indicate ± 1 SE.

Maximal rate of force development remained high, approximately 180 g/s in both the He and control groups throughout the experiment, but declined drastically in the N_2 group. Maximal rate of force relaxation declined in all three groups, but in the He and control groups it was consistently higher than in the N_2 group. The time-tension integral declined steadily in all three groups throughout the experimental period, with the He group exhibiting the highest and the N_2 group the lowest TTI. The time it took to reach peak tension was much greater in the N_2 group than in either the He or the control group (Fig. IIID-2).

Coronary flow resistance was calculated by dividing perfusion pressure (66 mmHg) by flow rate in ml/min. At time 0 (45 min after the isolation of the heart), resistance was 12.3, 10.1, and 9.1 mmHg/ml/min for the control, He, and N_2 groups, respectively. Two hours later, resistance was 29% and 54% higher in the control and N_2 groups than their respective time 0 values, while resistance in the He group remained virtually unchanged (Table IIID-2).

With re-establishment of the coronary flow after 20 min of total cessation, the control heart and 3 of the 5 hearts in the He group displayed a rhythmic electrocardiogram and contractile activity. The rest of the hearts (2 from the He group and all 4 from the N_2 group) went into fibrillation within 2 min after the flow was restored. All hearts went into fibrillation following the re-establishment of coronary flow after 30 min of total cessation of flow.

Discussion

Interest in the biological effects of helium gas has been renewed by the discovery, (18) confirmation, (19,20,21,32) and denial (7,16) of antiarrhythmic and/or antifibrillatory effects in the anesthetized dog breathing a gas mixture containing proportions of helium ranging from 20-80%. Pifarre and his associates proposed that the observed beneficial effect of helium in the coronary-artery-ligated anesthetized dog may be related to the increased collateral circulation using Schlesinger mass and coronary sinus blood flow measurements (19,20). However, Raymond et al. (21) could not reproduce the increased collateral circulation by Schlesinger mass and india ink injections They suggested that the alteration in autonomic nervous activities may be linked to the antiarrhythmic activities of helium. Lin and Kato (13) reported that respiration of an 80% helium-20% oxygen mixture depressed the heart rate (f_h) of unanesthetized rats, the f_h in rats breathing a helium-oxygen mixture being lower than in rats breathing air at any given rate of oxygen consumption. These experimental observations suggest that helium has a direct effect on the myocardium and/or the vasculature in the myocardium. Whether other He effects reported in the literature (Table IIID-1) have any relation to the observed improved performance and rhythmicity of the heart remains to be seen. Our findings indicate that:

1) Helium gas in the perfusate improves the mechanical performance of the isolated perfused rat heart, as indicated by a higher rate of force development (+dF/dt) and relaxation (-dF/dt), higher maximal force, higher indices of work (TTI/beat) and of power (TTI/min), and faster attainment of peak tension during systole. With a constant preload, the single most sensitive indicator of contractility is max dF/dt (17). The contractility of hearts in the He group was equal to that in the control group, while hearts in the N_2 group were depressed (Fig. IIID-2). The mechanism by which helium gas improves myocardial contractility as compared to N_2 gas remains unresolved. But we know that at time 0, (Table IIID-2, Fig. IIID-1,2) improved contractility in the He group was not due to the difference in coronary flow rate or the frequency of contractions. Pifarre et al. (19) reported that 2 hr after occlusion of the coronary artery, the color of the infarcted area was noticeably pinker in the He group than in the N_2 group. They speculated that oxygen diffused better in the presence of He. There is still no evidence that diffusion of O_2 in solution is facilitated in the presence of the gas, although there is evidence that O_2 diffusion is facilitated in the gas phase (26,34). Alternatively, the facilitated diffusion may be secondary to the opening of collateral anastomoses which they observed directly using Schlesinger mass. They also concluded that coronary collateral anastomoses developed faster when helium was administered. The capillary endothelium may also be altered to facilitate O_2 diffusion. Ritter et al. (25) reported that helium breathing causes a significant increase in serum lactic dehydrogenase and serum glutamic oxalacetic transaminase, a phenomenon associated with leaky membranes, implicating involvement of the membrane mechanism.

2) Improved contractility of hearts in the He group over those in the N_2 group for a prolonged period after isolation may be related to the higher coronary flow which was found in the He group, indicating a greater availability of oxygen in this group (Fig. IIID-1). Resistance to coronary flow rose steadily in the N_2 group. By the end of the experiment, values of resistance in the N_2 group increased by more than 50% over those at time 0, while resistance in the He group was unchanged. Pifarre et al. (20) had reported that the coronary flow of dogs breathing a helium-oxygen mixture returned to or surpassed the pre-occlusion level. Coronary flow level in the N_2 group also recovered, but to a lesser degree. They attributed these results to the effect of He gas on increases in coronary collateral circulation. Our results support this conclusion. The mechanism by which helium gas opens up the previously closed capillary is unknown. An alternative to this is that edema may develop in the hearts of the N_2 group during the course of each 3-hr run, compressing the cardiac capillaries and increasing resistance to flow (Table IIID-2). Helium may prevent the formation of edema. However, in experiments conducted under conditions similar to the ones we have described, no significant differences in ventricular water content (% wet wt) were observed among the hearts perfused with the three gas mixtures. This does not preclude the possibility of a difference in intravascular volume among the three groups.

3) The antifibrillatory activity of helium gas was demonstrated by the prevention of fibrillation in 60% of the He group hearts when coronary flow was re-established after the 20-min cessation period. All hearts which had been perfused with the N_2-saturated solution went into fibrillation after the flow was re-established after 20-min cessation. In the 30-min flow-stop experiments, all hearts ended in fibrillation within 2 min of restoration of flow regardless of what inert gases were present in the perfusate. However, it would be erroneous to conclude that He gas exerts no antifibrillatory effect in this type of experiment. An irreversible model (such as 30 min of zero flow, severe ouabain toxicity, and cardiac ischemia of major proportion), regardless of whatever agents or procedures are used, is not a valid model to indicate a negative result with certainty (see Introduction).

Summary

The biological effects of helium on the isolated male Wistar rat heart was studied using the Langendorff technique. In each heart experiment, the Kreb-Henseleit perfusate was saturated with one of three gas mixtures at 30 $\pm$ 0.5°C: 1) 95% O_2-5% CO_2; 2) 50% O_2-45% He-5% CO_2; and 3) 50% O_2-45% N_2-5% CO_2. Heart rate (f_h), coronary flow rate (Q), contractile force (F), time-to-peak tension (TTPT), time-tension integral (TTI/beat and TTI/min), and the maximum rates of force development (+dF/dt max) and relaxation (-dF/dt max) were measured or calculated. Contractile performance of hearts perfused with the helium mixture was equivalent to that of hearts perfused

with the 95% O_2-5% CO_2 mixture, and the hearts exhibited activities superior to nitrogen-mixture-perfused hearts in terms of f_h, Q, F, TTPT, TTI, and $\pm$ dF/dt max during the entire 3-hr period of perfusion. Hearts maintained on the helium-saturated solution had significantly higher coronary flow rates than hearts perfused with the nitrogen equilibrated mixture, indicating a greater availability of oxygen.

Helium therefore exerts a direct effect on the coronary vasculature, reducing its resistance to flow and improving the delivery of oxygen to the myocardium; this accounts for the improved myocardial performance. All hearts (4/4) perfused with the nitrogen equilibrated solution went into fibrillation within 2 min following restoration of coronary flow after 20 min of total cessation, whereas only 40% (2/5) of the hearts perfused with the helium-equilibrated solution went into fibrillation. Antiarrhythmic activity of helium gas is implicated.

ACKNOWLEDGMENT

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CHAIRMAN'S SUMMARY: HERMANN RAHN

One common thread emerges from these various papers: the effects of a helium atmosphere on physical and biological processes, and the possible implication of these when man is exposed to a helium atmosphere at pressures greater than one atmosphere.

Paganelli's presentation sought to define the role of the diffusion of water vapor in man's insensible water loss when exposed to high pressures in a helium atmosphere. If the diffusion process is an important factor, there should be significantly reduced insensible water loss, because according to kinetic theory the binary diffusion coefficient for water vapor is inversely proportional to the absolute pressure. For the first time Paganelli measured the diffusion coefficient in helium at various pressures up to 50 atm and found that the inverse law begins to deviate from the ideal above 4 atm. When oxygen is present in the helium atmosphere, binary diffusion laws no longer apply and the deviation from kinetic theory is considerably greater.

By using the increased diffusion coefficient of oxygen in helium over that in nitrogen, Van Liew made continuous measurements of arterial PO_2 in the dog to show that the oxygen delivery to the alveolus was reduced due to axial diffusion in the airways, but enhanced once oxygen was in the alveolar space. According to this interpretation an increase in pressure and consequent reduction in the diffusion coefficient would reduce axial diffusion loss of oxygen. Such a role for axial diffusion within the airway upon oxygen delivery to the alveolar space is of particular interest in looking at the problems of oxygen delivery during exercise when helium is breathed.

Moore tested 10 subjects breathing air and heliox at various work loads. At the 900-kpm load during a 20-minute stint, subjects were switched at five-minute intervals from one to the other mixture, including normoxic argon gas. While breathing helium their ventilation and heart rates were slightly higher, as were their CO_2 outputs, compared to those breathing air. However, the oxygen uptake was significantly reduced with helium, 1.3 liter/min compared to 2.2 liter/min on air and 2.6 liter/min on a normoxic argon mixture. Since we presumably have steady states throughout the 20-minute work span, it is difficult to account for the large reduction in oxygen consumption associated with a respiratory quotient slightly over 2.0 during the helium exposure.

Lin's presentation deals with another most interesting helium effect, namely, the presence of helium versus nitrogen gas in the perfusion fluid supplied to the isolated rat heart, employing the Langendorff technique. In one case the perfusion fluid was saturated with a 45% He, 50% O_2, 5% CO_2 gas mixture; in the other case the 45% He was exchanged for N_2. Hearts perfused with the helium-saturated solution had significantly higher coronary flow rates and performed better as judged by contractile force, time-to-peak tension, and maximum rate of force development than did hearts perfused with the N_2 gas. These observations

suggest that there is a direct effect of helium on the coronary vasculature. Whether this can be explained by an unknown pharmacological action or is somehow associated with an increased oxygen diffusion within the aqueous phase is difficult to judge, because classical concepts offer little basis for either explanation.

SESSION IV: SATURATION DIVING

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A. ENERGY BALANCE: PAUL WEBB

This paper concerns energy balance in the nutritional rather than the thermal sense, and describes the caloric exchange of men during saturation diving. A striking observation has been made that men lose weight during these dives despite adequate-to-high food intake; this was noticed during the first human saturation dive, Genesis E (G. Bond, personal communication). I have written on this topic before (5,6). I have now been able to find descriptions of 15 saturation dives where body weight was measured, and in all but one, loss of weight occurred despite food intakes ranging from 2500 to 6000 kcal/day. The one exception which showed neither loss nor gain of weight was a relatively shallow, 200-foot equivalent dry dive for seven days.

Why do men living for days under hyperbaric conditions lose weight? Does the environment cause a large increase in metabolic expenditure, as signalled by increased oxygen consumption rate ($\dot{V}O_2$)? A number of measurements of resting $\dot{V}O_2$ have shown only a small increase, e.g., Raymond et al. (1) and Salzano et al. (2), but a more complete measurement of energy expenditure throughout a day's activities might reveal the explanation. Do men fail to digest and absorb the food they eat during saturation dives? Only a complete caloric balance study would answer this question. A start was made in the 1971 dive to 1200 feet equivalent at the University of Pennsylvania, where we used conventional dietary techniques to measure food calories eaten. An intake of just over 3500 kcal/day was accompanied by a weight loss of 4 kg over 17 days, but there was little, if any, increase in resting or exercise $\dot{V}O_2$. This will be a report of a more complete experiment, a thorough balance study made during the 580-foot equivalent saturation dive called Hana Kai II, in Hawaii during March of 1975.

Loss of Body Weight in Saturation Diving. Let's look at the record of weight loss in saturation diving. Fig. IVA-1 shows weight loss as a function of the exposure in 15 dives, including Hana Kai II. Weight loss ranges from 0.0 to 4.0 kg. Pressure exposure is shown as the multiple of the days at depth times the gauge pressure (not ATA) of the dive in atmospheres. Thus, exposure 1 ATA is at 0 gauge pressure, and the product has to be 0.0 as well. That is, indefinite exposure to sea-level pressure does not, per se, relate to weight loss. A regression line is shown on the figure for 13 of the points. Two dives, separated on the figure by encircled points, had a significant cold pressure, which must have increased the caloric drain, and hence increased weight loss significantly.

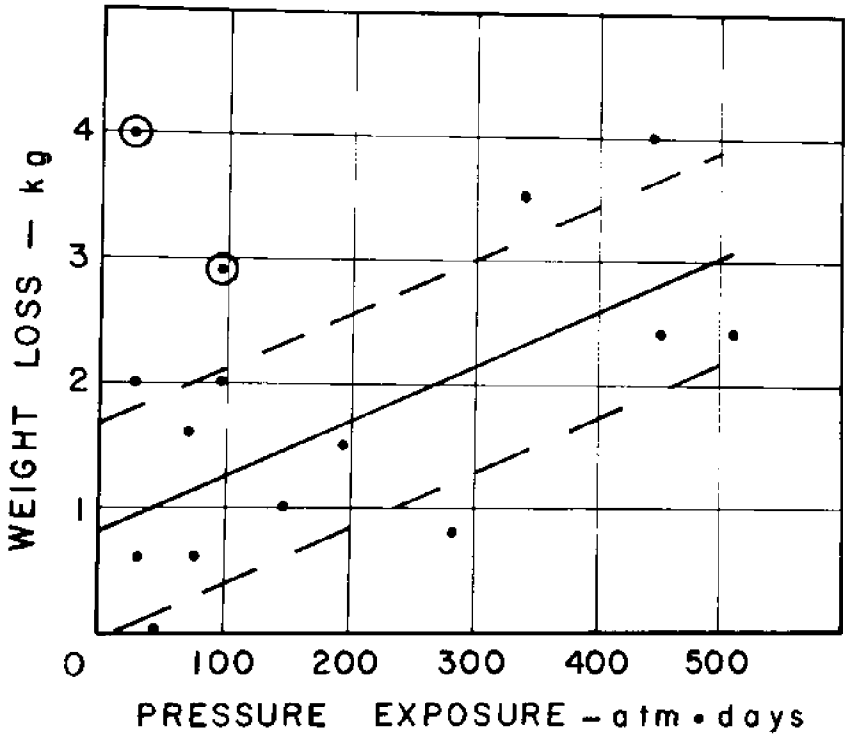

Fig. IVA-1. Weight loss of men during saturation dives as a function of pressure exposure; days at depth times gauge pressure in atmospheres. Regression line relates 13 of the points; SE is shown with dashed lines. Two encircled points are from saturation dives which produced distinct cold exposure.

The continuous nature of the loss of weight is illustrated in Fig. IVA-2 by the weight losses recorded in two dives where the subjects were weighed during the dive. The 1971 University of Pennsylvania dive was deeper and caused more weight loss. The University of Hawaii dive is the recent Hana Kai II; it shows an early loss of about 1.0 kg, regained during decompression. However, although the average body weights in Hana Kai II were the same after the dive as before, the average total body water had increased by 0.8 kg; there had thus been a loss of substance to that degree.

Methodology in Hana Kai II

The March 1975 saturation dive at the University of Hawaii, identified as Hana Kai II, placed five men at a pressure equivalent to 580 fsw, or 18.6 ATA, for 17 days. The atmosphere was mainly helium, with an oxygen partial pressure of about 210 mmHg, some nitrogen, and small quantities of CO_2 and water vapor. The temperature was regulated at a warm comfort level of 31°C, except for three days when

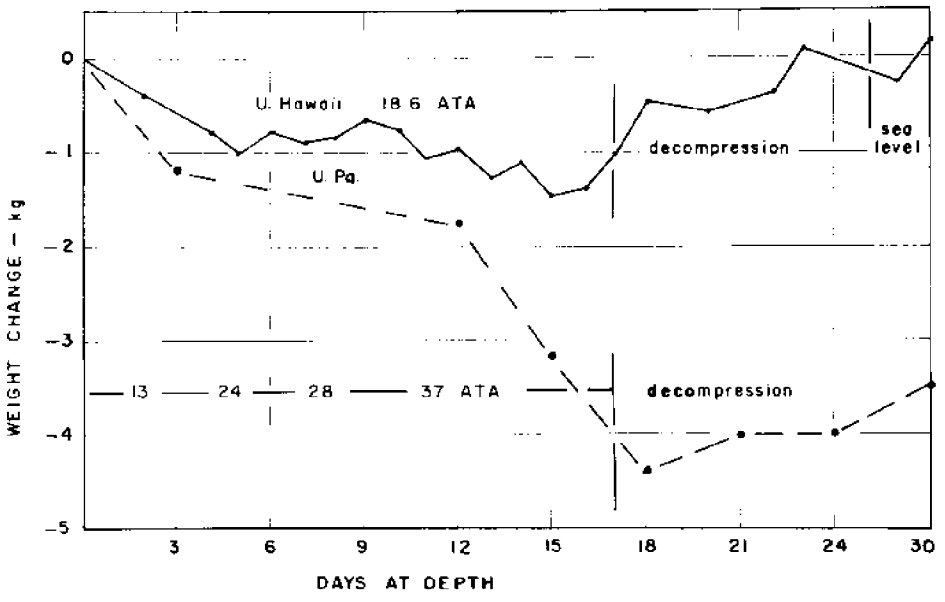

Fig. IVA-2. Average weight losses for five men during University of Hawaii's Hana Kai II project at 18.6 ATA, and average weight losses for four men at the University of Pennsylvania during a progressive dive ending with 6 days at 37 ATA (1200-foot equivalent).

it was kept at 27°C to produce an intentional cold exposure. The subjects, all members of the Department of Physiology of the university, carried out a full day's schedule of experimental and housekeeping activities. Exercise was limited to occasional submaximal bouts and two maximal efforts on a bicycle ergometer.

The entire experiment involved 30 days in the hyperbaric habitat with rigorous control of activity, food intake, and waste collection. The general outline of the experimental conditions was:

Dive days 1 - 3	Sea-level control
Dive day 4	Pressurization
Dive days 5 - 16	18.6 ATA, 31°C
Dive days 17-19	18.6 ATA, 27°C
Dive day 20	18.6 ATA, 31°C
Dive days 21 - 27	Decompression
Dive days 28-30	Sea-level control

Thus, the pressure exposure was 282 atmosphere-days, which omits compression and decompression days, since they were omitted in the other dive data used in compiling Fig. IVA-1.

The specific measurements for the energy balance study included body weight and composition, total body water, calories in food, calories in waste, and energy expenditure.

Body weight was measured each morning between 0600 and 0700 hours, before the subject had eaten but after voiding of urine.

Body composition, to give a value for the weight of body fat, was calculated from measurements of body density from underwater weighing. This was done the day before and the day after the experiment. To make the estimate more precise, total body water was measured on dive days 1 and 30. Also, as qualitative indicators of changes in the subcutaneous fat layer, skinfolds at nine sites and the circumferences of the chest and abdomen were measured daily.

Total body water was measured by deuterium oxide dilution, using a procedure like that of Wang et al. (3). In addition to the predive and postdive measurements on days 1 and 30, total body water was measured at mid-dive on day 13.

Calories in food were determined by oxygen bomb calorimetry. Each man's intake of each food item was known from the weight of food sent into the chamber, less his plate waste. Calorimetry of food items is still in progress at the Naval Medical Research Institute (NMRI), but we have data for the six days of sea-level control, the three days of 18.6 ATA at 27°C, and six of the twelve days of 18.6 ATA at 31°C.

Calories in waste were determined from collections of all urine and all feces. Urine calories are not yet available from NMRI, but all the fecal calories are available for calculation. A preliminary estimate of urine calories was used in the analysis to follow.

Energy expenditure was calculated from measured oxygen consumption (and CO_2 production) using a specially developed apparatus for long-term monitoring. It was an open-circuit method similar to the ventilated-hood technique, and employed a lightweight plastic facepiece and calibrated blower, similar to those of the metabolic rate monitor of Webb and Troutman (7). A fixed volume rate of 80 liters/min of chamber gas was drawn over the wearer's face, from which he breathed in and out. Continuous samples of the chamber gas and mixed exhaled and chamber gas from above and below the man were led outside the chamber and into sensitive differential gas analyzers for measuring the changes in PO_2 and PCO_2. From these data and the gas flow, continuous values for $\dot{V}O_2$ and $\dot{V}CO_2$ were computed electronically. These values were totalized electronically for sequential 10-minute periods, and the 10-minute totals printed out. Four of

the five subjects took turns wearing this apparatus for 24 hours a day throughout the experiment, except for the days of pressurization and decompression. During the 21 days the apparatus was used, 1500 ten-minute periods were measured, for a 61% coverage of the possible time available. These extensive data were used to calculate the energy expenditure in kcal/day, using the simplified method of Weir (4) to convert $\dot{V}O_2$ totals.

Energy Balance Calculations. From the data available at this time, we have calculated the energy terms for the three major experimental periods: six days of sea-level control; 12 days at 18.6 ATA, warm (31°C); and 3 days at 18.6 ATA, cold (27°C). Average values for the five men are shown in Table IVA-1.

Table IVA-1. Average daily energy values in major dive periods for 5 subjects

	Energy expenditure, kcal	Food intake, kcal	Fecal loss, kcal	Urine loss, kcal	Body Fat expended (stored), kcal
Sea-level control	2170	3586	-220	-125	(-40)
18.6 ATA, warm	2507	3341	-205	-125	392
18.6 ATA, cold	2972	3827	-208	-125	520

By convention, one expects the net intake to match the energy expenditure in periods lasting this long, and for this many subjects. In our data, it does not. Net intake is the calories in food, less the waste in urine and feces, plus (or minus) the fuel drawn from (or added to) fat stores. The inequality is shown graphically in Fig. IVA-3.

A number of things are evident from Fig. IVA-3 and Table IVA-1. First, food intake (allowed on a free-choice, unlimited basis) was high throughout the experiment, even during the sea-level control periods. We cannot ascribe this to hyperbaric conditions alone. Second, the energy expenditure rose with the hyperbaric condition alone, by about 15%, and rose again from the combination of 18.6 ATA and cold. But under none of the experimental conditions did energy expenditure match the high food intake. At the same time, the five men, on the average, lost a little weight over the 30 days. In fact, three men lost body fat, one stayed the same, and one gained a little. Third, the absorption of food was a normal 90% of food

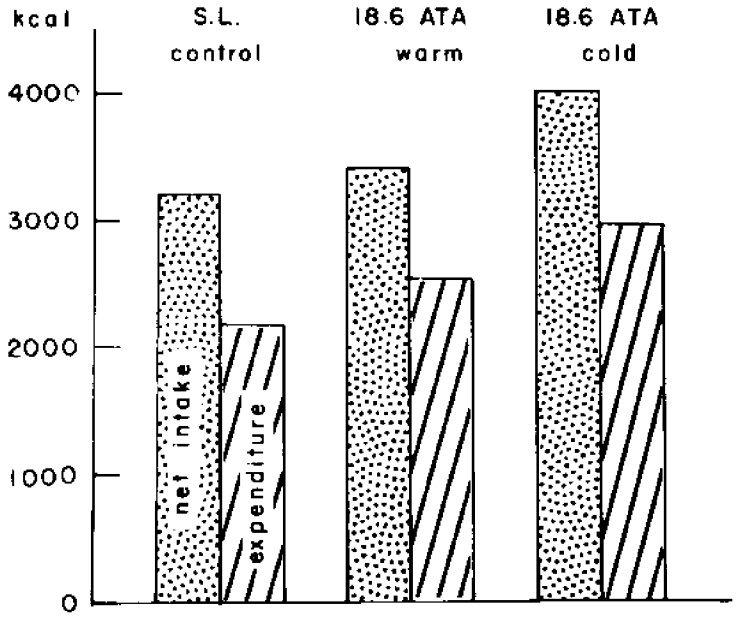

Fig. IVA-3. Average daily net intakes (food minus urinary and fecal waste, plus or minus fuel from fat stores) and energy expediitures for five men during major periods of Hana Kai II. There were 6 days of sea-level (S.L.) control, 12 of 18.6 ATA at 31°C (warm), and 3 of 18.6 ATA at 27°C (cold).

intake, under all conditions of the experiment. Fourth, the mismatch between net intake and expenditure is constant and striking. This warrants further discussion.

In all three major conditions of the experiment there appeared an excess of intake over expenditure of roughly 1000 kcal/man each day. Such an excess of intake should have caused storage of body fat. If our preliminary calculations are correct (we are still missing data on nitrogen balance, and thus we cannot use R.Q. corrections and we cannot estimate tissue loss), and further, if the 19 days of caloric balance data represent the whole 30 days, then the amount of fat storage which should have occurred can be estimated. One thousand kcal per day for 30 days is a storage of 30,000 kcal/kg/man, which means, assuming adipose tissue contains about 7000 kcal/kg, that each man should have gained about 4.3 kg. We know they did not.

It would appear that there is something wrong with the conventional assumption that intake and expenditure, as usually measured, should balance out over many days for several subjects. The situation here is something like luxus consumption, in which an excessively

high intake, in relation to the subject's activity, does not cause as much weight gain as expected. There has never, in my opinion, been an adequate experimental explanation of this, although investigators have postulated that a high-energy expenditure must accompany an excess intake.

A similar but opposite situation is beginning to appear in some as yet unpublished work in my own laboratory. In these studies, direct calorimetry and energy expenditure do not provide 24-hour matches, yet there is no body heat storage. When men undereat in relation to their energy expenditure, the mismatch of that expenditure is higher than the sum of all the measured losses of heat and work, which, one step removed, is like the bar graphs of Fig. IVA-3 in reverse.

We are beginning to think that entropy plays a significant role in metabolism, and that this unmeasurable quantity, entropy, is the missing 1000 kcal/man each day in the Hana Kai II data. More extensive and more rigorous experiments will be needed in the laboratory to resolve this. Perhaps by the time another saturation dive is planned to include an energy balance study, we will be able to devise an appropriate experimental design to test the entropy hypothesis in hyperbaric conditions.

Summary

During saturation diving, men lose weight despite an adequate-to-high intake of food. A complete caloric balance study was undertaken to define the energetics of hyperbaric living. During the Hana Kai II saturation dive to 18.6 ATA for 16 days, we measured daily body weight on five men, their total daily food intake, and we collected all urine and feces. Change in body fat was estimated from body density and total body water before and after the dive. Calories in food and waste were determined from oxygen bomb calorimetry. In addition, energy expenditure was calculated from 24-hr monitoring of oxygen consumption on four of the men. Results showed that food intake was high, about 3500 kcal/day; food absorption was a normal 90% of intake, and energy expenditure, while elevated by 15% over sea-level control, was about 1000 kcal/man per day less than net food intake. The five men lost an average of 0.8 kg of body fat in the 30 days of the experiment. The discrepancy between intake and expenditure is thought to be due to entropy.

ACKNOWLEDGMENT

I express gratitude to Dr. S. K. Hong for allowing me to participate in and to modify his Hana Kai II project, and my admiration for the five subjects, and for the many other members of the University of Hawaii's Physiology Department, who worked so hard to make the experiment a success. Finally, without the enthusiastic

participation of Dr. V. Frattali and his laboratory at the Naval Medical Research Institute, the energy balance data would not have been done.

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B. CARDIOVASCULAR FUNCTIONS DURING SATURATION DIVING: RICHARD M. SMITH

In 1936 Shilling, Hawkins, and Hansen (32) described a reduction in pulse rate with increased barometric pressure which they attributed to the increased pressure of oxygen. Since then most human studies in the hyperbaric field have confirmed the existence of a "hyperbaric bradycardia" (15), but the underlying mechanism(s) and significance of this response remain obscure.

Effect of Hyperoxia. Since hyperbaric atmospheres are generally hyperoxic (0.3 to 0.4 ATA O_2), the original suggestion (32) of an effect of high PO_2 would seem to have merit, particularly when combined with exercise, because hyperoxia is known to reduce heart rate (HR) for a given submaximal work load both at 1 ATA (4) and at increased ambient pressure (3). However, HR during tests of $\dot{V}O_{2max}$ at 580 ft He-O_2 was significantly lower by 8 beats/min during both hyperoxia and normoxia.* Thus, a hyperoxic effect may not be present at all levels of work. Moreover, resting HR in men at increased pressures is generally decreased by about 15% (15) while, by contrast, breathing pure oxygen at 1 ATA generally produces little if any decrease in the resting HR (6).

Fagraeus (9) has addressed this question of the effect of high PO_2 and succeeded in showing an oxygen-dependent reduction of HR at depth which he ascribed largely to a direct cardiac effect with a smaller component mediated via the vagus nerve. However, an important component of the bradycardia was unaffected by PO_2 or atropine and this component could be reduced but not abolished by propranolol. Thus, it appears that while there may be a hyperoxic effect on HR it cannot be the sole explanation for the hyperbaric bradycardia, particularly at rest and during maximal exercise.

Pressure and Increased Gas Density. Strong evidence against hyperoxia as a sole cause of hyperbaric bradycardia was recently obtained by Flynn, Berghage, and Coil (8) who used a variety of pressures and normoxic gas mixtures of various densities to demonstrate the separate contributions of gas density and pressure to normoxic hyperbaric bradycardia. During exercise, pressure and density already appeared to exert their maximum effect at about 10 ATA. At depths greater than 300 fsw the exercise bradycardia appeared to be independent of both pressure and gas density. The apparently independent effects of gas density and pressure could not be explained, although the possibility of a

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*R. H. Dressendorfer, S. K. Hong, J. F. Morlock, J. Pegg, B. Respicio, R. M. Smith, and C. Yelverton. Hana Kai II: a 17-day dry saturation dive at 18.6 ATA. V. Maximal oxygen uptake. Undersea Biomed. Res. (in preparation).

helium effect on the pressure component was mentioned.* Flynn et al. further suggested that the effect of gas density was mediated by the associated increase in airway resistance. They proposed that an increased tidal volume (VT) at depth may activate thoracic stretch receptors, leading to bradycardia. The exact nature of this reflex was not discussed.

Albano (1) has suggested that abdominal gas compression at depth caused a reduction of intra-abdominal pressure with abdominal pooling of blood and activation of intra-abdominal stretch receptors. The mechanism by which this hypothesized response causes bradycardia is not clear.

Cold. In addition to demonstrated effects of hyperoxia, gas density, and perhaps pressure or inert gas on heart rate at depth, Moore and Hong and associates (27,16) have suggested that a subtle cold stress may be present in hyperbaric heliox environments (35) which could contribute to the observed bradycardia. Cold, especially on the face, is known to elicit both bradycardia and peripheral vasoconstriction; the former but not the latter is blocked by atropine (14). Though not always present, an increase in resting blood pressure (BP) at depth has been reported in several dives (24, unpublished observations). Moore et al. (27) observed an attenuation of bradycardia at a depth of 500 feet when the ambient temperature was raised from 27.8° to 29°C. This experiment is not decisive, however, since no control for time at depth was performed and the bradycardia might have disappeared without a temperature change. Several studies show that bradycardia is attenuated and eventually disappears as a function of time at depth (5,15,24, unpublished observations). This theory holds that the cold-induced increase in total peripheral resistance (TPR) might lower the HR via the carotid sinus or perhaps indirectly via central pooling of blood (11) and subsequent increase in cardiac preload. A major difficulty with this theory is that exposure to a cold 1-ATA environment may evoke an increase in TPR without changing cardiac index or HR (21), and habitat temperatures are often comfortably warm, particularly during compression, when the bradycardia is pronounced.

Finally, Hong et al. (16) reported an increased bradycardial response to face immersion in 30°C water in the hyperbaric environment which was equivalent to the response seen in much colder water (5-15°C) in 1 ATA air. Based on this evidence, the authors suggested that facial cold receptors may respond differently to a given thermal stimulus in a hyperbaric heliox environment.

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*The effects of helium on the cardiovascular system are discussed elsewhere in this symposium by Y. C. Lin and will not be pursued here.

Multiple Factors. The existing literature indicates that hyperbaric bradycardia is complex; many factors contribute to bradycardia at depth, and no single factor adequately explains it. Moreover, the various factors involved probably affect HR for different reasons and differently during various activities. The different effects of hyperoxia at rest and exercise are a case in point. Another example is presented by Hong et al. (16), who found that resting HR was not affected by pressure upon decompression, but the 60-s breath-hold HR was lower at depth. This disputed result (18) has recently been confirmed in four subjects during decompression from 580 ft (unpublished observations). In addition, changes with time further complicate the problem. For example, the reported attenuation and even disappearance of bradycardia with time at depth may indicate an adaptation to saturation diving. Clearly, our present knowledge of the significance and causes of hyperbaric bradycardia is incomplete and will be improved by further research.

The purpose of this paper is to present evidence which suggests that altered respiration at depth may be responsible for the density-dependent component of hyperbaric bradycardia. We propose that a more negative intrathoracic pressure with increasing gas density leads to an increased thoracic blood volume which contributes not only to the bradycardia seen at depth, but also to increased stroke volume and diuresis upon compression, which results in possible dehydration. In addition, lack of evidence for an important role of cold in hyperbaric bradycardia is presented.

Methods

Data were obtained from four subjects during a 17-day dry saturation dive to 580 ft in the three-chambered habitat Aegir. Complete methods and results from this 30-day experiment, code named Hana Kai II, will be published subsequently.

Thoracic impedance (Z_0) between the base of the neck and the xiphosternal joint was measured with a 4-electrode impedance plethysmograph, described by Kubicek et al. (19). Except for compression day, the electrodes were removed after each measurement. Changes in the distance between pick-up electrodes affect Z_0, and were compensated for by dividing Z_0 by the distance (length in cm) between the electrodes. Cardiac stroke volume (SV) was calculated by the method of Kubicek, except that the electrical resistivity of blood used was 135 ohm cm. The method proposed by Kubicek et al. to calculate SV was calibrated using a modified single-breath nitrous oxide method (20). Agreement between the two methods was excellent in the four divers when they were seated erect in a body plethysmograph. Linear regression of N_2O stroke

volume (N_2OSV) versus impedance stroke volume (ZSV) yielded the following relationship, $\pm$ SD

$$ZSV = 0.98\ (\pm\ 2.2)\ N_2OSV + 3.1\ (\pm\ 23.3);\ r = 0.95.$$

Associated with very low HR's in two subjects were stroke volumes exceeding 180 ml/beat, causing the mean SV's reported here to be unusually large.

Thoracic impedance at fixed lung gas volume was used to estimate changes in thoracic conductive volume following the method of Schaefer et al. (31), except that blood resistivity was again taken to be 135 ohm cm. Impedance data were obtained between 1100 and 1230 hours, according to the following schedule: dive days (DD) 2,3 (predive 1-ATA control); DD4 (compression), DD5 (first day at 580 ft), DD15,16 (580 ft, comfortable temperature (Ta = 30-31°C)), DD19 (580 ft, cold (Ta = 27-28°C)), DD29,30 (postdive 1-ATA control).

Results

Thoracic Impedance. Average results of the thoracic impedance measured at three lung volumes in three postures are shown in Fig. IVB-1. The most important observation is a fall in Z_0 (top) on DD4 during compression irrespective of posture or lung volume (values on abcissa (DD4) are approximate depths in feet). Since the electrodes remained on the chest during DD4, the distance between the pick-up electrodes did not change, hence the Z_0/L curve (middle) faithfully follows the Z_0 curve. If the equation for calculating thoracic conductive volume (TCV) has merit, an extra 150 ml of blood was present in the thorax during compression when subjects were seated erect at 350 ft. The increase while lying supine was nearly twice this volume. On the first day at 580 ft (DD5), the seated TCV generally decreased to below control values, while the supine TCV appeared to return to the predive value. However, there was a tendency for Z_0/L to remain lower than the predive value. Thus, I suspect that the TCV was still increased on DD5 despite the diuresis.

After 11 days at 580 ft, Z_0 was sharply increased, but Z_0/L changed little between DD5 and DD16. Yet calculated TCV is maximal on DD16, probably because of the influence of an increased L on calculated TCV (Fig. IVB-1). I believe this weakness in the equation for calculating TCV places all relative changes in TCV in doubt, except those occurring during compression where L was not changed. However, Z_0/L at fixed lung volume is probably a reliable index of changes in thoracic blood volume (19,29) even when the circumferential band electrodes are not positioned in exactly the same place on repetitive measurements. There was a tendency for Z_0/L to increase in the cold (DD19) indicating a reduction in thoracic blood volume. This cold stress was sufficient to cause shivering with an increase in $\dot{V}O_2$ of about

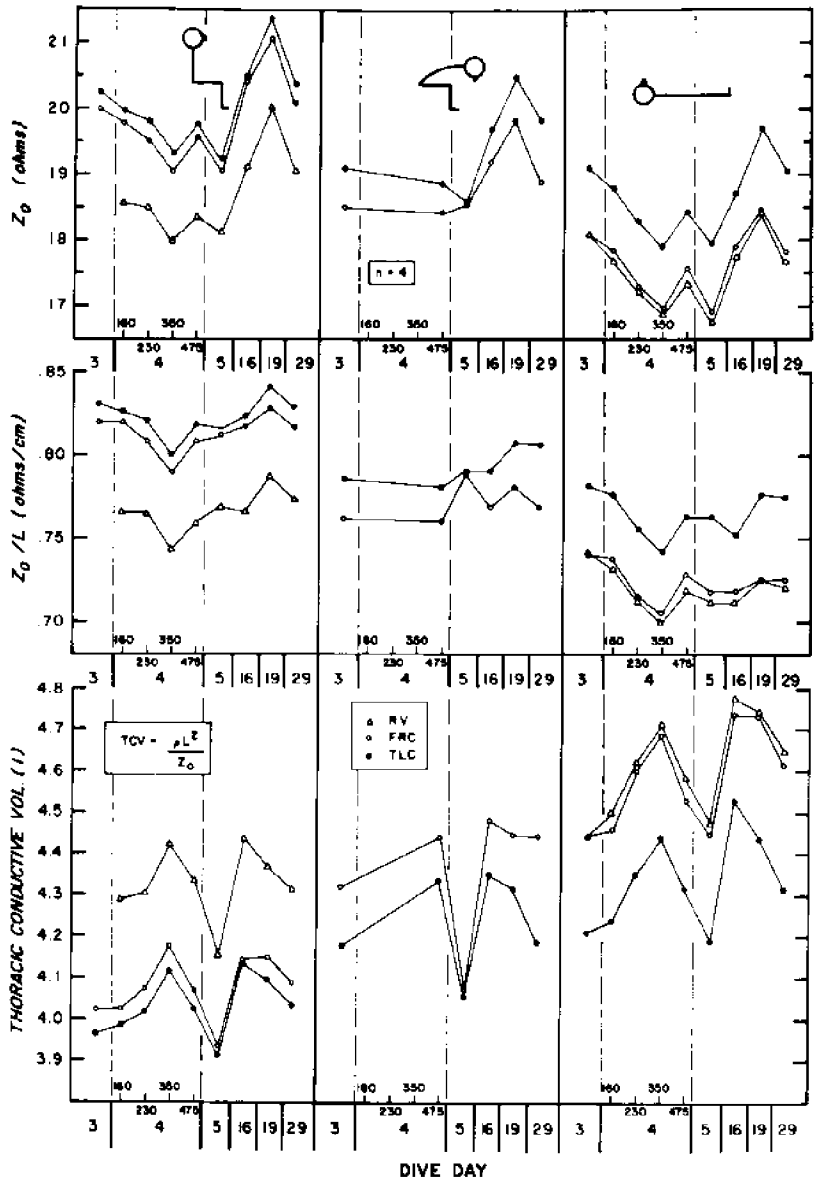


Fig. IVB-1. Thoracic impedance and its derivatives as a function of dive day. Where measured, 3 postures and 3 lung volumes are shown: (Δ) RV = residual volume; (○) FRC = functional residual capacity; (●) TLC = total lung capacity. Depth in ft on day 4 is also shown, and corresponds sequentially to the following approximate hours of day: 0930, 1130, 1430, and 1730.

100 ml/min (STPD). Postdive (DD29) Z_0/L returned to predive values when subjects were seated erect but tended to be lower than predive values when they were in the supine position, particularly at low lung volumes (Fig. IVB-1), possibly indicating an increased postdive thoracic blood capacitance.

Figure IVB-2 shows that as TCV peaked during compression at 350 ft, urine flow also was increasing markedly. It is tempting to speculate that the increased urine flow tended to restore the TCV towards normal (10,12,33), possibly leading to relative dehydration elsewhere in the body. Signs of mild systemic dehydration were evident during the first 3 days at pressure (15). Increased urine flow persisted overnight (DD4) and by mid-morning of the first day at 580 ft, the TCV was apparently restored to normal (Fig. IVB-2),

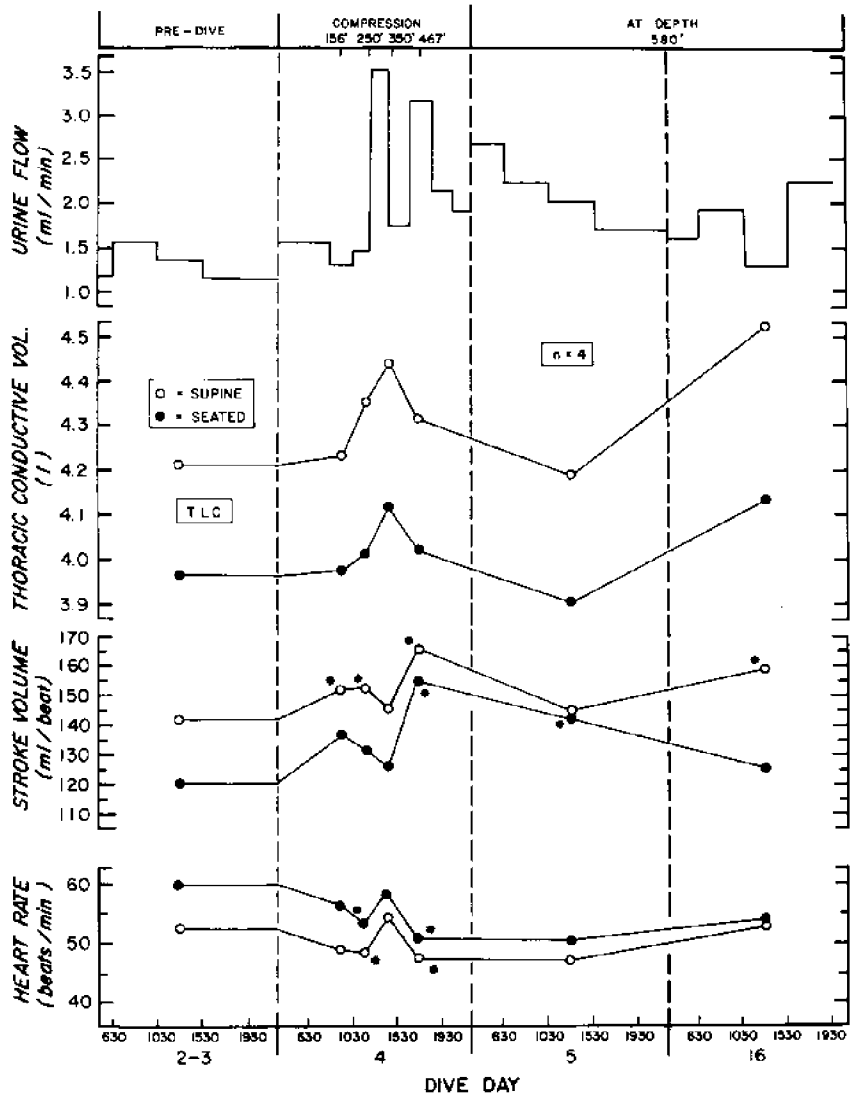


Fig. IVB-2. Time course of selected parameters during compression to 580 ft. Thoracic conductive volume measured at full inspiration (TLC); heart rate and stroke volume measured at normal end-expiratory level.

but Fig. IVB-1 shows that Z_0/L was still lower than the predive value. During 11 days at pressure the TCV seemed to increase again (Z_0/L data from supine subjects agree with this interpretation) indicating that a high thoracic blood volume may be a persistent phenomenon at depth. The supine HR had returned to normal by DD16, but seated HR was still depressed after 11 days at 580 ft. In general, the cardiac SV changed inversely with HR and was significantly increased at depth. Overall, the SV increase was larger than the HR decrease so that cardiac output (CO) was slightly increased at depth (5).

Effects of Cold. Interestingly, steady-state cold stress (DD17-19) caused an increase in HR with little change or a decrease in TCV (Fig. IVB-1) suggesting that the bradycardia and expanded thoracic blood volume at depth were not the result of cold-induced peripheral vasoconstriction.

The possibility of altered thermoreception (16) in He-O_2 was tested in the following experiment (Table IVB-1). We obtained face

Table IVB-1. Effects of changing ambient temperature (Ta, °C) in 1 ATA air and 18.6 ATA He-O_2 on maximal change in HR due to face immersion (FI) in 27°C water, compared to 60-s sham FI.

HR_{max}, beats/min	Air (1 ATA)		HeO_2 (18.6 ATA)	
	T_{face}	Ta	T_{face}	Ta
0	30.6	20.4	30.6	27.8
1	31.6	22.6	31.7	29.2
2	32.6	24.7	32.8	30.5
3	33.6	26.6	33.8	31.8

temperature (T_{face}) and the maximal difference in HR between sham face immersion (FI) and 27°C FI after 4 hr in air temperatures from 15° to 28.5°C, and compared these with values obtained at 18.6 ATA He-O_2. Results show a close agreement between Δ HR_{max} and T_{face} at 1 and 18.6 ATA, in spite of much different Ta's in the two conditions. Since Δ HR_{max} with 27°C FI is similar at sea level and at depth for a given face temperature, I conclude that for these conditions the thermoreceptors are behaving similarly.

Finally, habitat Ta was uncomfortably warm during compression (DD4) when bradycardia was present and Z_0/L was falling. Thus, it appears that the increase in TCV and decrease in HR seen in this study were not related to cold or to altered thermoreception.

Discussion

Evaluation of Impedance Data. Conclusions depend directly on the ability of the impedance plethysmograph to detect changes in thoracic blood volume adequately. No other instrument or technique scans the same anatomical area, which makes direct calibration difficult or impossible. Nevertheless, changes of Z_0 with posture and lung volume (Fig. IVB-1) indicate that the impedance signal contains information. Sjoestrand (33) estimated that about 500 ml of blood shifted to the thorax when subjects are supine compared to standing erect. Thus, a calculated blood shift of 300 ml seated to lying seems to be of the correct order of magnitude and lends credence to the calculated shift of about 150 to 250 ml during compression when L did not change.

Pomerantz et al. (29) using Kubicek's machine, demonstrated a linear relationship between the amount of pleural effusion removed by thoracentesis and an increase in Z_0 in 3 patients, with a slope of 1 ohm/200 ml of effusion removed. Their figure shows that a 50-ml change was detectable over the 0-1300 ml range. These results and other as yet unpublished observations lead me to believe that impedance plethysmography is probably the most sensitive technique available for detecting changes in thoracic fluid volume. However, caution should be exercised in interpreting the absolute thoracic conductive volumes because this derived parameter is strongly influenced by L, as mentioned, and also the electrical resistivity of blood which was not measured in the present study. Additionally, at low lung volumes the diaphragm moves into the measurement field causing Z_0 to decrease. In this case the TCV is not a true reflection of the thoracic blood volume.

Finally, since the entire chest between the recording impedance electrodes is scanned, it is impossible to locate the precise intrathoracic site of the increased TCV. The limited data available (33) suggest that an increase in thoracic blood volume is shared by the lungs and heart in a 3:1 ratio, respectively. It would be interesting to obtain measurements of cardiac chamber dimensions at depth.

Altered Respiration at Depth. The cardiovascular and body fluid consequences of altered respiration have been topics of importance for some time (10,12,33). However, despite good evidence for an altered breathing pattern in the hyperbaric environment, the cardiovascular sequalae of slower and deeper respiration at depth have not been investigated.

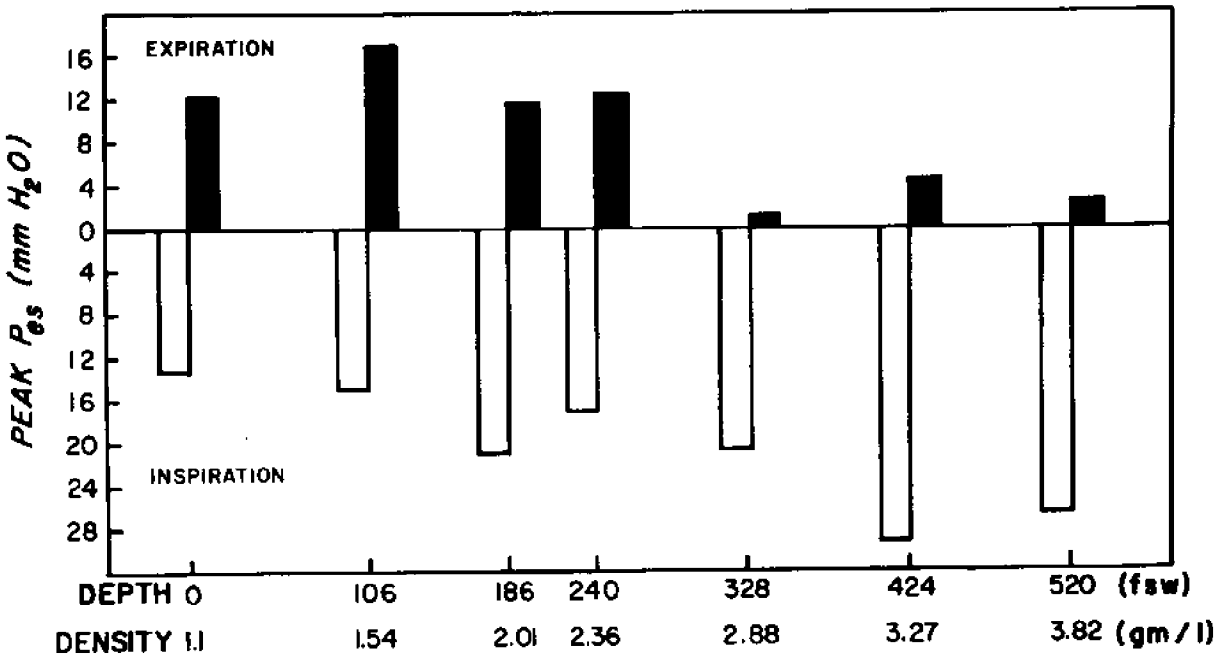
McIllroy et al. (26) showed that the typical adaptation to an increased non-elastic breathing resistance included both a reduction in respiratory frequency (f_r) and an increase in the end-expiratory (FRC) lung volume. A given subject may not show both responses, however, since there is an inverse relationship between the decrease in f_r and the increase in FRC. Two groups have now demonstrated a 300-400 ml increase in FRC at pressure (relative gas density about 4) (36, unpublished observations).

Maio and Farhi (23) have shown that a relative gas density of 4.0 caused an increased airway resistance leading to a three-fold increase in the work of breathing and a reduction in maximal voluntary ventilation (MVV) of about 40%. Zechman, Hall, and Hull (37) found that the addition of a flow resistance (applied equally to inspiration and expiration) which reduced MVV by 40% caused an increase in the resting FRC in addition to the tidal volume (VT), confirming the results of McIllroy et al. This increase in the resting FRC and tidal lung volumes must increase the passive recoil pressure of the lungs, allowing expiration to remain largely a passive process. Finally, added flow resistance (37) hardly changed the time required to reach peak inspiratory flow while peak expiratory flow, in addition to being lower in magnitude than inspiratory flow rate, was delayed slightly in time (37). This adaptive pattern of ventilation at depth increases $\dot{V}A$ relative to $\dot{V}E$, minimizes the work of breathing (28), and may prevent a further increase in expiratory flow resistance due to dynamic airway compression which occurs at depth when expiration is active (13).

Intrathoracic Pressure and Blood Volume. Data above show that most of the increased work of breathing at depth occurs during inspiration, suggesting greater peak inspiratory than expiratory pressures. Esophageal pressure (P_{es}) has often been measured at depth to calculate the work of breathing, but unfortunately the values for P_{es} are not generally reported. I measured P_{es} in one subject during decompression from 580 ft (Fig. IVB-3) and found that peak inspiratory pressures increased with increasing gas density, while the peak excitatory pressure simultaneously decreased.

I reason that a more negative intrapleural pressure at depth should augment venous return, leading to an increased thoracic blood volume with larger cardiac end-diastolic and stroke volumes. Thus, the density-dependent component of hyperbaric bradycardia is viewed as secondary to an increased cardiac preload induced by the altered breathing pattern and larger lung volumes at depth. Indeed, since thoracic blood volume is known to increase with increasing lung volume, an increased cardiac preload may be responsible for the inverse relationship between lung volume and HR previously reported (3).

Sinus Arrhythmia. One might hypothesize that slowing the respiratory rhythm induces bradycardia via sinus arrhythmia (25). As mentioned, when both inspiratory and expiratory flow resistance are



Spontaneous peak inspiratory and expiratory esophageal pressures in 1 subject during decompression. Each value is average change from end-tidal (FRC) level during a 5-min observation.

similarly increased, the time to reach peak inspiratory flow is hardly changed, while the time for expiratory flow is slightly lengthened (37). Since cardio-acceleration generally accompanies inspiration and cardio-deceleration accompanies expiration at normal breathing frequencies (2), the added flow resistance due to increased gas density (23) alone could conceivably produce a lower average HR because of changes in the centrally induced sinus arrhythmia, particularly since lower respiratory frequencies at depth seem to potentiate this phenomenon (1,18). According to data of Angelone and Coulter (2), this fluctuation in HR is easily of a magnitude (up to 25 beats/min) to account for the 15% reduction in resting HR typically seen early in saturation dives (15). It has been suggested that this mechanism partly accounts for the apneic bradycardia seen in sea lions, where apnea accounts for 84% of the respiratory cycle (22).

Hypothesis. A literature survey of the effects of an added resistive load on respiration suggests an increased cardiac preload may be responsible for the gas-density component of hyperbaric bradycardia. In addition, a possible sinus arrhythmia effect may produce bradycardia directly, with a secondary effect on cardiac preload and stroke volume. The situation is further complicated by the possible role of increased transmural pressure during inspiration at the

level of the aortic baroreceptor, and it may prove difficult to separate these effects in humans. Separation of gas density effects from pressure or helium effects will require 1-ATA studies using breathing gases of varying density.

Whether an increased cardiac preload is the primary cause of bradycardia or a secondary effect of it, the results presented here show that at pressure the thoracic blood volume is increased and that this is associated with bradycardia, increased stroke volume, increased urine flow (Fig. IVB-2), and possibly an increase in blood pressure.

It should be restated that many factors may affect cardiovascular parameters at depth besides gas density. Bradycardia at depth appears to be extremely complex as discussed above. In addition, suppression of insensible water loss could contribute to the effects discussed here.* However, there does not seem to be enough time for the suppression of insensible water loss to account for the rapid shift of blood to the thorax during compression. Thus, it is tempting to speculate that altered respiration at depth contributes not only to hyperbaric bradycardia and increased TCV and SV, but to the early diuresis as well. This effect may be mediated by high- (12) or low-pressure (10) intrathoracic stretch receptors, and may lead to a mild dehydration with reduced plasma volume and other long-term consequences for body fluid homeostasis under pressure (17).

Summary

A literature review of cardiovascular functions during saturation diving suggested that the often reported hyperbaric bradycardia is complex, with possible 1) hyperoxic, 2) pressure (or inert gas), 3) gas density, and, possibly, 4) cold effects. Preliminary data from a 580-ft dry saturation dive relevant to the gas density and cold effects are discussed. It appears that cold is not a major contributor to bradycardia at depth. However, a literature survey of the effects of altered respiration due to increased gas density at pressure suggested two mechanisms by which increased gas density may contribute to hyperbaric bradycardia: 1) slightly prolonged expiration may augment the normal sinus arrhythmia and 2) larger lung volumes at depth may lead to a more negative intrathoracic pressure with increased venous return. Esophageal pressure (P_{es}) measurements in one subject during decompression from 580 ft confirmed that P_{es} is more negative at depth. Thoracic impedance plethysmography in four subjects undergoing compression to 580 ft confirmed an increase in thoracic blood volume at depth, which was associated with an increased stroke volume, bradycardia, an increased urine flow, and a mild dehydration. It is suggested that these

*See S. K. Hong's paper in this symposium.

cardiovascular and body fluid alterations at pressure are primarily the result of the altered respiratory pattern due to breathing a dense gas.

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C. BODY FLUID BALANCE DURING SATURATION DIVING: SUK KI HONG

Maintaining a constant body fluid volume and composition is one of the major requirements to support divers' activities during a prolonged dive, as in the case of saturation diving. However, in one of the earlier dry saturation diving experiments conducted by Hamilton et al. (7), a significant increase in urine flow was observed at a depth of 650 ft. A similar hyperbaric diuresis was noted in many subsequent dives to various depths (see below). If such diuresis is sustained during a long dive, severe dehydration could develop which will not only affect divers' performance but also endanger their safety. This presentation will attempt a comprehensive review of the current state of the art concerning the mechanisms and the consequence of hyperbaric diuresis.

Hyperbaric Diuresis as a Phenomenon

Daily urine flows observed in various dives are summarized in Fig. IVC-1. Open bars represent urine flow before the dive and

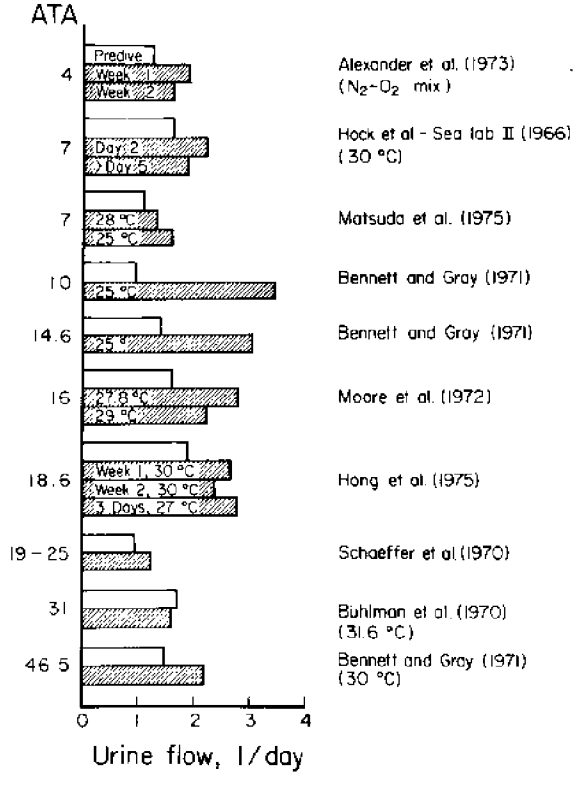

Fig. IVC-1. Summary of daily urine flows (liter/day) observed in different dives

shaded bars during the dive. Three important conclusions can be drawn from these data. First, urine flow increased under pressure in all dives except in the case of a dive conducted by Buehlmann et al. (4). On the average, the urine flow increased from the pre-dive level of approximately 1,000-1,500 to 1,500-3,000 ml/day during the dive, independent of the depth. Second, a careful inspection of the figure indicates that magnitude of diuresis is related to ambient temperature. For instance, the greatest diuresis was seen in dives to 10 and 14.6 ATA conducted by Bennett and Gray (2); the ambient temperature in these dives was 25°C, which is considerably lower than the comfortable level at the above pressures (21). Moreover, in studies conducted by Hong et al. (unpublished data), Matsuda et al. (13) and Moore et al. (14), a greater diuresis was observed when the ambient temperature was lowered by several degrees without changing the pressure. Third, it is important to note that the diuresis was maintained throughout the dive. For instance, the diuresis lasted throughout the 14-17 day dive period in dives conducted by Alexander et al. (1) and Hong et al. (unpublished data). However, the data also indicate a slight attenuation of diuresis as the dive progressed.

Such a diuresis observed under pressure gradually disappears during decompression and the urine flow returns to the pre-dive level upon returning to the surface. The time course of the appearance of this diuresis has not been carefully investigated in most dives. Matsuda et al. (13) noted that urine flow increased within a few hours after the start of compression to 200 ft. To get a better resolution on the time-course of the increase in urine flow, we collected urine every 2 hr during the compression phase of our recent dive to 580 ft, and found that urine flow abruptly increased nearly threefold 6 hr after the start of compression. These findings indicate that the mechanisms for this hyperbaric diuresis are set in motion as soon as a compression starts.

Whatever may be responsible for this diuresis, one can readily rule out greater fluid intake as a cause, since the total water intake (drinks plus water contained in food) did not significantly increase during the dive (13, unpublished data of Hong et al.). Fluid intake in the form of drinks also remained almost unchanged during the dive (2,19).

Body Fluid Volume

As stated above, a hyperbaric diuresis is sustained during the dive in the absence of any increase in water intake. Therefore, one would predict a significant dehydration to develop during a prolonged dive. However, two studies in which the body fluid volume was measured indicate the absence of such a dehydration (Fig. IVC-2). Assuming insignificant changes in daily water intake,

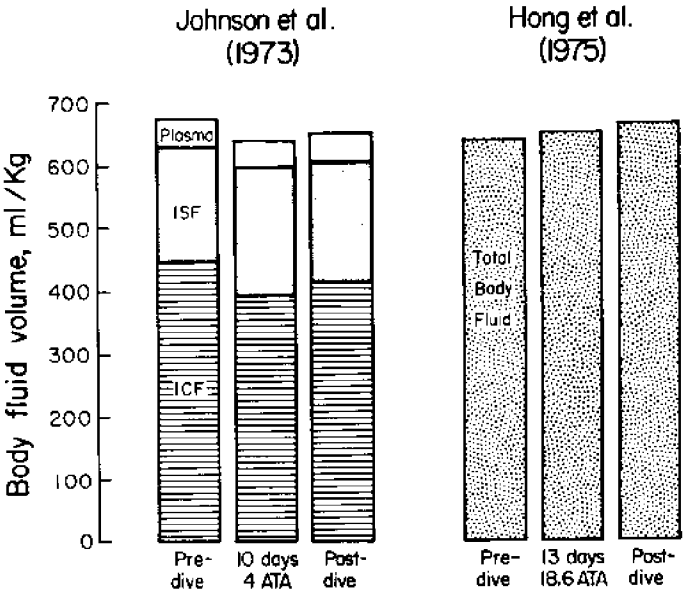

Fig. IVC-2. Body fluid volume data obtained from 2 dives

one would have expected in the study by Johnson et al. (9) a reduction in the total body water volume of 5.6 liters (or 70 ml/kg) during 10 days at 4 ATA, in contrast to the actual reduction of only 35 ml/kg. In our own dive, no change in the total body water volume was observed during 2 wk at 18.6 ATA, while a reduction of 9.3 liters (or 130 ml/kg) would have been predicted.

These results strongly suggest that the overall body fluid balance appears to be maintained in the presence of a persistent diuresis during a prolonged dry dive, as long as there is no cold stress. However, the data of Johnson et al. (9) suggest that there may be some redistribution of body water among three major body fluid compartments. At 4 ATA, the extracellular fluid volume slightly increased while the intracellular fluid volume decreased. Moreover, the plasma volume decreased at depth. The physiological significance of these results is not clear at present, and it is important to verify the above findings in future studies.

Hematocrit Ratio and Plasma and Urine Chemistry

Many studies included measurements of the hematocrit ratio and the chemical composition of plasma and urine. Although the results are by no means clear-cut in many cases, I tried to be as objective as possible in extracting the trend shown in Table IVC-1.

The changes in the hematocrit ratio were variable: it increased in some studies and decreased in others. Apparently, these variable results are not related to cold stress, since opposite changes are

Table IVC-1. Summary of changes in the hematocrit ratio, plasma chemistry, and urinary excretion of solutes observed during various saturation diving experiments.

Pressure, ATA	Hematocrit ratio	Plasma Concentration					Urinary Excretion						Reference
		Osm	Na	K	Creatinine	Protein	Osm	Na	K	Ca	P	Creatinine	
4.0	+	+	+	+	0	+	+	+	+	+	+	+	(1)
7.0	0		+	0				0	+	-	+	0	(8)
7.0		0	0	0	0	0	0	0	0				(13)
10.0								+	+	+	+		(2)
14.6								+	+	+	+		(2)
16.0	+	-	-	0	0	+	0	0	0			-	(14)
18.6	+	-	-	-	0	+	+	0	+	-	+	0	Unpublished data of Hong et al.
19.0-25.0	0		0	0				+	0	0	+		(19)
31.0	-	0	-	-	0			-	+	-	+	0	(4)
46.5								-	+	-	+		(2)

+ = increase; 0 = no change; - = decrease

seen even in the absence of cold stress (4, unpublished data of Hong et al.). However, the increase in the hematocrit ratio observed in 3 out of 5 studies is consistent with a reduction in the plasma volume, as reported by Johnson et al. (9).

Plasma concentrations of total osmotic particles, Na, and K also show a large variation among different studies. On the other hand, the plasma protein concentration tends to increase, if there is any change at all, which suggests a loss of some plasma water. This may be related to changes in the hematocrit ratio and plasma volume, discussed above.

Urinary excretions of total osmotic particles, Na, and Ca show no consistent trend. On the other hand, the majority of studies shows a consistent increase in the urinary excretion of K and P. Whether the latter findings indicate a faster turnover of intracellular constituents or not is yet to be determined.

The plasma concentration of endogenous creatinine seems to stay remarkably constant while urinary excretion shows variable results. In other words, the endogenous creatinine clearance, which is a measure of the glomerular filtration rate (GFR), seems to change randomly.

Renal Tubular Functions

Fractional excretions of the filtered total osmotic particles, Na, and K are calculated from the values of the plasma clearance of these substances relative to the endogenous creatinine clearance. Only four studies provide the necessary information for this calculation (Table IVC-2). In general, fractional excretions of the filtered osmotic particles, Na, and K tend to increase, if there is any change at all, which suggests an inhibition of the tubular reabsorption of these filtered substances.

All studies which measured the urine osmolality generally showed that it decreased during the dive but always remained hypertonic. Therefore, the free water clearance, i.e., the urine flow minus the osmolal clearance, was still negative. Table IVC-2 also shows that the changes in this negative free water clearance ($T^c_{H_2O}$) are quite variable.

In other words, the magnitude of increase in urine flow during a dive is comparable to that in osmolal clearance. This indicates that inhibition of tubular reabsorption of solutes is associated with a corresponding inhibition of water reabsorption.

Mechanism(s) of Hyperbaric Diuresis

According to the modern theory of urine formation, an increase in GFR and/or an inhibition of the tubular reabsorption of water

Table IVC-2. Changes in fractional excretion of the filtered total osmotic particles, Na, and K, and negative free water clearance ($T^c_{H_2O}$) during various saturation diving experiments.

Pressure, ATA	Fractional excretion of filtered total osmotic substances	Na	K	$T^c_{H_2O}$	References
4	0	0	-	+	(1)
7	+	+	+	0	(13)
16	+	+	+		(14)
18.6	0	0	+	-	Unpublished data of Hong et al.

+ = increase; 0 = no change; - = decrease

induce a diuresis. As discussed earlier, the GFR as estimated by the plasma clearance of endogenous creatinine seems to vary randomly during the dive. Therefore, one may rule out an increase in GFR as a primary mechanism for hyperbaric diuresis.

The tubular reabsorption of water can be inhibited either directly, by lowering the level of circulating antidiuretic hormone (ADH), or indirectly, by inhibiting the tubular reabsorption of osmotic particles, especially Na. The latter is determined by the level of circulating aldosterone and so-called "third factors(s)." The exact nature of the third factor(s) is still unknown, but it is held responsible for a natriuresis observed during an expansion of the extracellular fluid (ECF) volume. A pituitary hormone, prolactin, induces both antidiuresis and antinatriuresis (3), and inhibition of this hormone is considered to be a candidate for a third factor (Solomon, personal communication). On the other hand, a dilution of plasma protein concentration, usually associated with the ECF volume expansion with saline, leads to a reduction of the oncotic pressure of the peritubular capillary blood, which in turn inhibits the reabsorption of Na and water from the proximal tubule through some unknown mechanism (see, for example, 10). In fact, many renal physiologists strongly suspect that such a change in physical force for transcapillary exchange may be another third factor (physical factor theory). At any rate, it is most likely that hyperbaric diuresis is primarily associated with alterations of some or all of the mechanisms outlined above. As discussed in the preceding section, the data in the literature indeed suggest an inhibition of the tubular reabsorption of the filtered osmotic particles and water (Table IVC-2).

Several theories have been advanced in the past to account for hyperbaric diuresis. Bennett and Gray (2) proposed that hyperbaric

diuresis is basically a cold diuresis. Inhibition of the ADH system in cold is well established (20). As discussed earlier (see the first section of this paper), the degree of hyperbaric diuresis is inversely proportional to the chamber temperature at a given depth (Fig. IVC-1), lending support to the above theory. However, the fact that a significant diuresis is present even at high chamber temperatures strongly argues against the "cold stress theory." Moreover, a typical cold diuresis is not accompanied by an increase in osmolal clearance and is self-limiting because of ensuing dehydration. In contrast, a hyperbaric diuresis is accompanied by an increase in osmolal clearance and is moreover sustained for as long as 14-17 days. These considerations lead to the conclusion that cold stress, if present, may play a role, but is certainly not the primary mechanism for hyperbaric diuresis.

During rapid compression the partial pressure of inert gases in the blood should exceed that in the poorly perfused tissues, thereby producing osmotic gradients (11). Such osmotic gas gradients would cause water shifts from poorly perfused tissues to the blood and better perfused tissues. If so, one might expect an increase in the ECF volume which would lead to a diuresis through various mechanisms discussed above (e.g., inhibition of ADH and aldosterone and activation of third factors). In fact, Schaefer et al. (19) proposed this as a primary mechanism for hyperbaric diuresis. It should be pointed out, however, that the above osmotic gas gradients will eventually disappear once the body is fully saturated with inert gases. Therefore, this "gas osmosis theory" may account for the diuresis observed during the early phase of saturation diving, but cannot explain the diuresis which is either present during decompression or sustained for two weeks or longer at depths. The fact that the magnitude of diuresis is greater during the first week as compared to the second week (1, unpublished data of Hong et al.) may be explained by this theory. However, it is very difficult to consider this as the primary mechanism for hyperbaric diuresis.

During compression the density of breathing gas increases progressively, thus altering the mechanics of respiration. One of the consequences of breathing a denser gas is to increase the negative intrathoracic pressure as measured by the esophageal pressure (22, unpublished data of Smith et al.). On the basis of this finding, one would expect an increase in venous return as well as in thoracic blood volume. In our recent dive, we found that thoracic impedance at a fixed lung volume decreased progressively in all four subjects during compression (unpublished data of Smith et al.), strongly implying an increase in the thoracic blood volume. If we accept the above interpretation, it is possible to explain the diuresis by involving an inhibition of both ADH and aldosterone mediated by the stimulation of volume receptors located in the left atrium. It is generally accepted that the stimulation of left atrial receptors leads to suppressions of both ADH (see, for

example, 6) and the renin-aldosterone system (5). A critical test for this "density theory" will be to see if diuresis develops when breathing a denser gas mixture at 1 ATA. To my knowledge, this has not been done so far.

Finally, I would like to consider the possible effect of pressure per se. If we look carefully at the composition of chamber gases used in different dives, we are struck by the fact that the partial pressure of O_2, N_2, and He varied greatly. For instance, PO_2 varied from 0.2 to 0.56 ATA (1,4), PN_2 from 1.2 to 3.8 ATA (1,13), and PHe from 0 to 46 ATA (1,2). Despite such wide variations in chamber gas composition, a diuresis similar in magnitude is observed in all dives (Fig. IVC-1). It may be that the pressure per se somehow interferes with the overall regulation of body fluid volume. Such a pressure effect should be dependent upon the level of the pressure, but this pressure-dependence may be offset by other effects of various component gas species.

One important effect of pressure on body fluid volume regulation is the suppression of water vapor diffusion from the skin to the environment. According to the Chapman-Enskog theory, there is an inverse relationship between the binary diffusion coefficient of a gas and the ambient pressure (18). The same theory also predicts that the binary diffusion coefficient of a gas is greater as the molecular weight of the ambient gas decreases. Paganelli and Kurata (15) have recently tested this theory in our laboratory for the binary diffusion of water vapor into air, He, and heliox over a pressure range of 1-50 ATA, and noted that the theory holds for pressures below 20 ATA, above which a slight but significant deviation was observed. According to their observation, water vapor diffusion into He is approximately three times greater than that into air at a given pressure level. In other words, the rate of water vapor diffusion into He at 3 ATA is comparable to that into air at 1 ATA, and at pressures greater than 3 ATA the rate of water vapor diffusion into He is below that into air at 1 ATA.

If the above theory and observations are extrapolated to the physiological system, one could easily visualize disturbances in the body fluid volume regulation in hyperbaric environments. One would expect a reduction in the insensible water loss at air pressures greater than 1 ATA or at heliox pressures greater than 3 ATA. Such a reduction in the insensible water loss from the skin would then account for hyperbaric diuresis. As pointed out earlier, the total body water volume does not change even when the diuresis lasts for 14-17 days (Fig. IVC-2), lending strong support to this theory. If the increment in urine flow represents the decrement of insensible water loss at pressure, there is no reason to expect a change in the total body water volume. Slight increases in the hematocrit ratio and plasma protein concentration associated with a reduction in the plasma volume noted in many dives are not

consistent with this theory; it is possible that these changes reflect the combined effect of other factors not directly related to the insensible water loss.

In a dive conducted by Matsuda et al. (13), the diuresis was sustained for 7 days but there was no evidence for dehydration, indicating that the body water balance was well maintained. Based on this premise, I calculated the magnitude of insensible water loss at 1 ATA air and 7 ATA heliox environments (Fig. IVC-3).

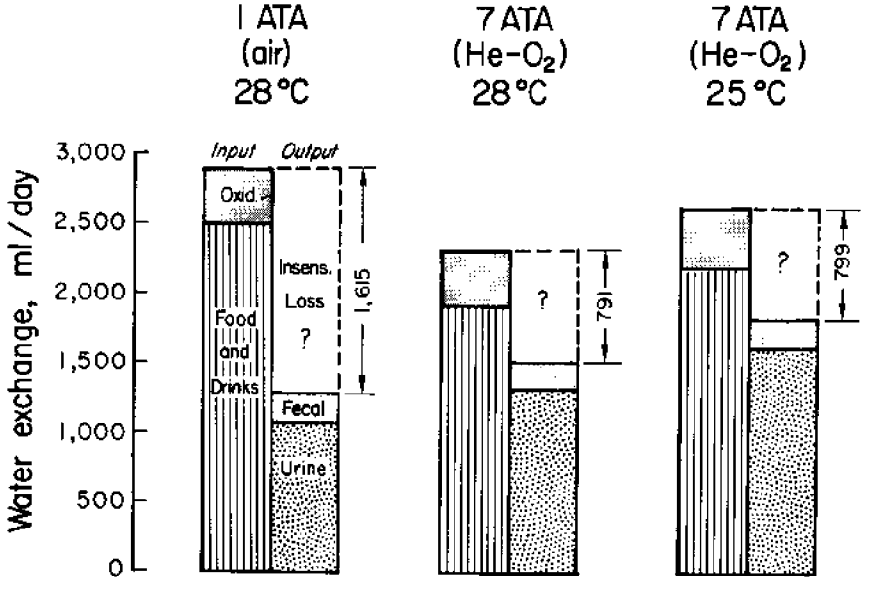

Fig. IVC-3. Reconstruction of daily body water exchanges during a dive to 7 ATA conducted by Matsuda et al. (13)

The insensible water loss is estimated to be approximately 1,600 ml/day at 1 ATA air and decreased to 800 ml/day at 7 ATA, representing a 50% reduction. In at least three dives, the insensible water loss was measured and the results are shown in Fig. IVC-4. Since the insensible water loss is also affected by humidity and skin temperature, the results are not as clear-cut as expected. Nevertheless, there is a clear tendency for the insensible water loss to decrease as the pressure increases.

On the basis of considerations given above, I propose that the primary mechanism for sustained hyperbaric diuresis is the suppression of the insensible water loss from the skin. Associated with this primary mechanism are several secondary mechanisms (such as cold stress, the osmotic effect of inert gases, and the effect of breathing denser a gas mixture) that alter the basic pattern of diuresis.

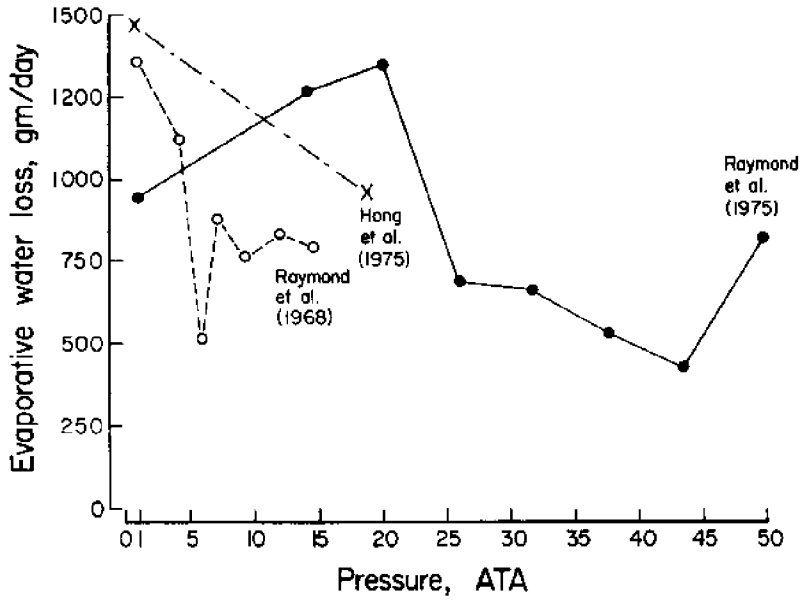

Fig. IVC-4. Daily evaporative water loss as a function of pressure. Two curves for Raymond et al. are calculated from their data on evaporative heat loss (16,17).

Endocrine Changes

Whatever the primary and secondary mechanisms are for hyperbaric diuresis, one might expect to find certain clues from changes in the blood and/or urine levels of hormones that are known to control the tubular reabsorption of osmotic particles and water. Table IVC-3 summarizes the findings in the literature. As in the case of other data, the measurements are not only scarce, but the results are also highly variable; it is thus virtually impossible to draw any definite conclusions. Actually, it is not surprising to see such variability, because divers are exposed to multiple stresses.

All theories (cold stress, gas osmosis, density, water vapor diffusion) proposed by various investigators predict an increase in either thoracic blood volume, ECF volume, or total body water volume, all of which would at least transiently inhibit the ADH system and/or renin-angiotensin-aldosterone, and which could activate the third factors. Table IVC-3 shows that urinary ADH decreased in two dives but remained unchanged in the other. On the other hand, renin and aldosterone in blood and urine either increased or remained unchanged. Serum prolactin decreased in one dive but not in another. This means either that the theories are wrong, or that changes in thoracic blood volume, ECF volume or total body water

Table IVC-3. Changes in endocrine functions during various saturation diving experiments.

Pressure, ATA	Blood Level				Urinary Excretion			References
	Renin	Aldosterone	ADH	Others	Aldosterone	ADH	Others	
4.0	+			- T_3 - T_4 + insulin 0 cortisol	0	-	+ epineph. + NE + cortisol	(12)
7.0	0			0 prolactin	0	0	+ epineph. 0 NE	(13)
10.0, 14.6, 46.5							+ 17-OH corticosteroid	(2)
18.6	0	+		- prolactin	+	-		Unpublished data of Hong et al.
31.0							+ epineph. - NE	(4)

+ = increase; 0 = no change; - = decrease

volume as predicted above may be too small to elicit consistent changes in the above hormones. Another critical point is that the hormone levels in blood and urine may undergo cyclic changes during a dive, and it may be meaningless to determine them intermittently. At any rate, far more comprehensive studies are needed in the future to identify possible endocrine changes that may reflect the mechanism of hyperbaric diuresis.

Table IVC-3 also shows changes in the level of thyroid hormone, insulin, catecholamines, and other adrenal critical hormones during various dives. Some of these changes may alter the body water balance, thereby further complicating the basic issue. Although no data are available, it is tempting to speculate a stimulation of the parathyroid gland. As pointed out earlier, in the section on hematocrit ratio and in Table IVC-1, a phosphaturia is observed in all dives in which the measurement was made. This is associated with a reduction in Ca excretion in 4 out of 8 dives.

Summary

A diuresis develops during compression and is sustained throughout the dive. This diuresis is not accompanied by a corresponding increase in fluid intake, and yet total body water volume appears to be maintained. This diuresis is independent of changes in the glomerular filtration rate and appears to be due to an inhibition of tubular water and solute reabsorption. It is proposed that the primary mechanism for this hyperbaric diuresis is the suppression of the insensible water loss from the skin. Associated with this primary mechanism are several secondary mechanisms, such as cold stress, the osmotic gas effect, and the effect of breathing a denser gas, that alter the basic pattern of diuresis.

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CHAIRMAN'S SUMMARY: PETER B. BENNETT

I do not intend to spend very much time reiterating what has been better said by the speakers; it has proved difficult to summarize papers, particularly when I only received about 30% of them prior to the meeting! Instead, after a few brief comments, I would like to identify a practical problem of great significance for saturation diving, if saturation is to remain an accepted method of diving.

Dr. Miller has effectively described the SCORE and other air-excursion programs, and it is clear that this method of diving is probably restricted to depths of less than 250 feet, with the saturation stage at 60 or 90 feet. To carry this stage further, or maybe even at this depth, will result in exposing men to long durations of high nitrogen partial pressures, and we know little of how this will affect the body. It is pertinent, however, that there is at present considerable concern among anesthesiologists about the effects of exposure to low doses of anesthetics over a prolonged period on the function of the liver and other organs.

In terms of the degree of narcosis at 250 feet, Dr. Miller has noted that the men themselves maintained that they were fit and able to carry out their tasks. Nevertheless, this is a formidable level of nitrogen partial pressure, and I do not believe that the diver himself is able to make judgments as to his own susceptibility. Indeed, during the dry studies at Duke, the individual who said he was the least narcotic at 300 feet showed the most performance decrement.

Whether adaptation to narcosis occurs on making excursions from a saturation stage is not clear. Certainly there is no lessening of narcosis on compression to greater depths after saturation at 60-ft air.

If the greater nitrogen partial pressure at 90 ft will have any better effect remains to be proved and there is much work to be done on air saturation diving, including the determination of safe decompression practices.

The question of diving bradycardia as addressed by Dr. Smith is for me as puzzling as ever. Although altered respiration at depth may contribute to hyperbaric bradycardia, the complex interaction of other variables such as cold, oxygen, and inert gas narcosis are confusing and suggest a complex mechanism affected by changes of many variables. My and Dr. Fagraeus' recent finding of no bradycardia during oxygen-helium dives at 500 and 600 ft during arm exercise, and the fact that other Duke studies by Dr. Salzano show an adaptive process, merely serve to indicate that much more work is required on this interesting physiological

phenomenon, preferably on isolated heart preparations. However, the bradycardia is not of pressing practical importance to man in the sea.

Hyperbaric diuresis as described by Dr. Hong is similar, but is of significant physiological interest. Apparently this occurs in the absence of any increase in water intake but without dehydration, perhaps because of a reduction of insensible water loss. Measurements of fluid balance are difficult at the best of times--during a complex saturation dive they are difficult indeed, which may account for the wide variability in results of electrolyte and enzyme changes during such dives. Controls are singularly lacking and it would be interesting to know what would happen to the same divers in the same situation but without exposure to increased pressure. The nature and water level of food is also often ignored in such studies, yet much water can be obtained through food and many electrolyte changes could be due to the diet.

Dr. Hong argues that since chamber temperature is relatively high, usually around 31°C for helium dives, there is no cold diuresis, but this argument ignores the special thermal characteristics of helium and that a 1/2°C shift may cause a diver to shiver. Also, with many such saturation dives a 1/2°C fall in core temperature has been reported. Again, more research is required before we can decide which of the many variables is responsible for the diuresis.

Dr. Webb has given an excellent, if nonconclusive, discourse on the energy balances of nutrition versus metabolism during helium saturation diving, in trying to explain the origins of weight loss. Since time is short, I want now to take a little time to identify what is for me the most important aspect for the future of saturation diving, and one which has not been discussed here.

This is that due to HPNS, and possibly to aseptic necrosis of bone, compression now needs to have stages like decompression. For very great depths of 1600 ft or greater these appear to require at least 12 hours. French workers have used 49-hr stages and 10 days to reach 2001 ft, yet the men were still not normal. With 10 days required to get back to the surface, the economics of such diving make it neither practical nor safe. The oxygen levels and possible Chouteau effect, which allows only a tiny range of allowable oxygen with oxygen toxicity occurring on one side and Chouteau hopoxia on the other, are other critical areas.

There is another less obvious but still important aspect of saturation diving, which at first glance may seem naive, but to me is not. I refer to boredom. The pressing boredom experienced by divers during long saturation dives badly needs attention, as do the frequent occurrence of nightmares during sleep.

There is no time to consider the whole range of serious physiological disorders in saturation diving; most of those present are certainly familiar with them anyway. However, I would like to say this in closing. The shortage of research funds and qualified researchers and laboratories makes it imperative that we concentrate our interests on the major problems which affect the diver's efficient performance, and only later investigate the physiologically interesting but inessential and less practical problems. If we do not, "man in the sea" may be replaced by "machine in the sea". This then is a plea to those basic researchers doing very excellent work to relate their findings as soon as possible to the applied workers trying to solve important practical problems. We shall hear more of these practical problems tomorrow--in that sense this session has been a bridge between basic and applied approaches to man in the sea.

SESSION V: DECOMPRESSION SICKNESS

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A. THE PHYSICS OF BUBBLE FORMATION: DAVID E. YOUNT

The first recorded case of decompression sickness was described by the British physicist and chemist, Robert Boyle, in 1670. Boyle had just invented a vacuum pump, and he soon tried it out on a number of subjects, both animate and inanimate. He observed, for example, that fresh milk and blood boiled vigorously when the gas surrounding them was evacuated with his pneumatic engine. In another experiment, Boyle subjected a viper to the same test and found that it did not die immediately from oxygen starvation, but was instead "furiously tortured". Eventually the snake developed a conspicuous bubble in the "waterish humour of one of its eyes". On the basis of his various observations, Boyle attributed the viper's agony to the formation of bubbles in the "blood, juyces, and soft parts of the body".

While Boyle probably never imagined that humans would intentionally subject themselves to the rigors of decompression, his observations remain accurate and timely. Decompression sickness occurs after a reduction in ambient pressure. It is therefore associated with such modern-day activities as diving, working in pressurized tunnels and caissons, and flying at high altitudes in unpressurized aircraft. The disease results from bubble formation in the blood or tissues, as Boyle noted, and many different parts of an animal can be affected. In fact, bubble formation is a general property of decompressed aqueous media, such as milk, blood, and animal tissue. Such generality suggests a common origin in basic physics and chemistry, and it is therefore appropriate to begin a session on decompression sickness at this fundamental level.

Tensile Strength and Supersaturation Pressure. The tensile strength of a liquid (4,34) can be determined by subjecting it to a negative pressure, as shown in Figs. VA-1, A and B. If the pressure is sufficiently large, the sample will fracture or cavitate into vapor and liquid phases, as indicated in Fig. VA-1, C. Measured tensile strengths vary widely, depending upon such details as the purity of the liquid, the type of container, the temperature, and so on. For example, the presence of gas nuclei in the sample can alter the result by two or three orders of magnitude.

Cavitation can also occur because of gas dissolved in the liquid at higher than ambient pressure. Supersaturation can be achieved by subjecting the sample to a pressure schedule such as that shown in Fig. VA-2. The initial gas pressure is p_o, the final pressure is p_f, and the maximal pressure is p_m. Some minutes (t_m) after the maximum pressure p_m is reached, the pressure is lowered and held for

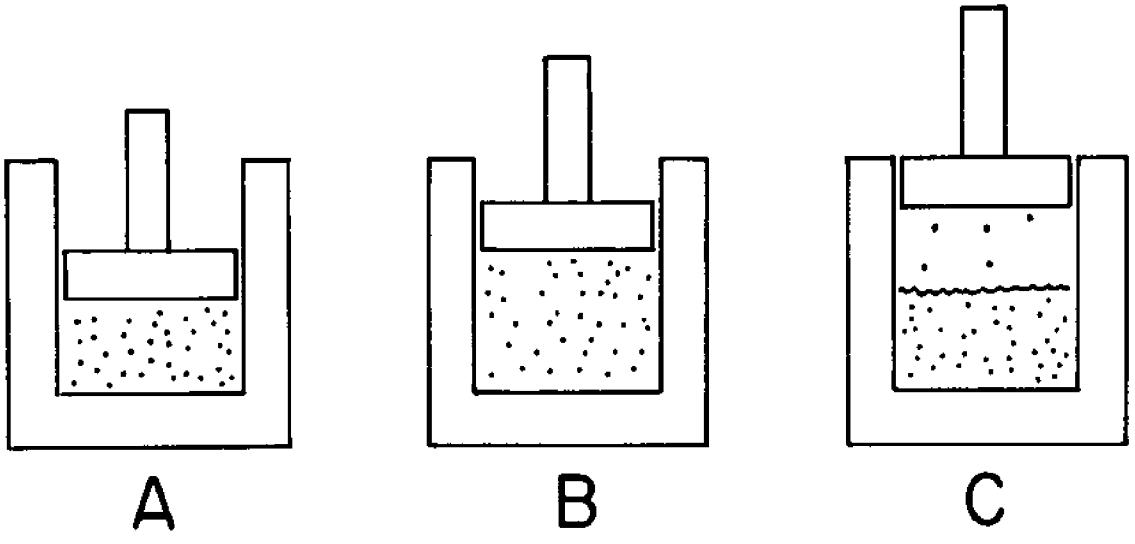

Fig. VA-1. A: liquid at ambient pressure; B: application of negative pressure, causing slight expansion; C: fracturing of sample into liquid and vapor phases.

several hours (t_s) at p_s until saturation of the sample by the surrounding gas is complete. The supersaturation pressure is defined as the difference in the gas tension in the sample and the ambient pressure p_{amb}. For complete saturation followed by rapid decompression, this gives

$$p_{ss} = \tau - p_{amb} = p_s - p_f \qquad (1)$$

By "rapid", we mean sufficiently fast that diffusion of gas into or out of the sample can be neglected. A "slow" process would be one in which diffusion plays a major role and in which the gas tension τ in the sample remains in quasi-equilibrium with the ambient pressure p_{amb} outside.

A negative, external pressure is applied in studying the tensile strength of a liquid, whereas the supersaturation pressure p_{ss} is positive and mainly internal--internal in the sense that the gas tension τ in the sample is greater than the external ambient pressure p_{amb}. The tensile pressure pulls the liquid apart, while the supersaturation pressure bursts it open. Further, the tensile strength of a pure liquid depends upon vaporization, while bubble growth in a supersaturated sample depends largely upon diffusion of the dissolved gas. In spite of these distinctions, the two concepts are closely related and have a common basis in nucleation, the process by which a vapor or gas phase originates within a liquid.

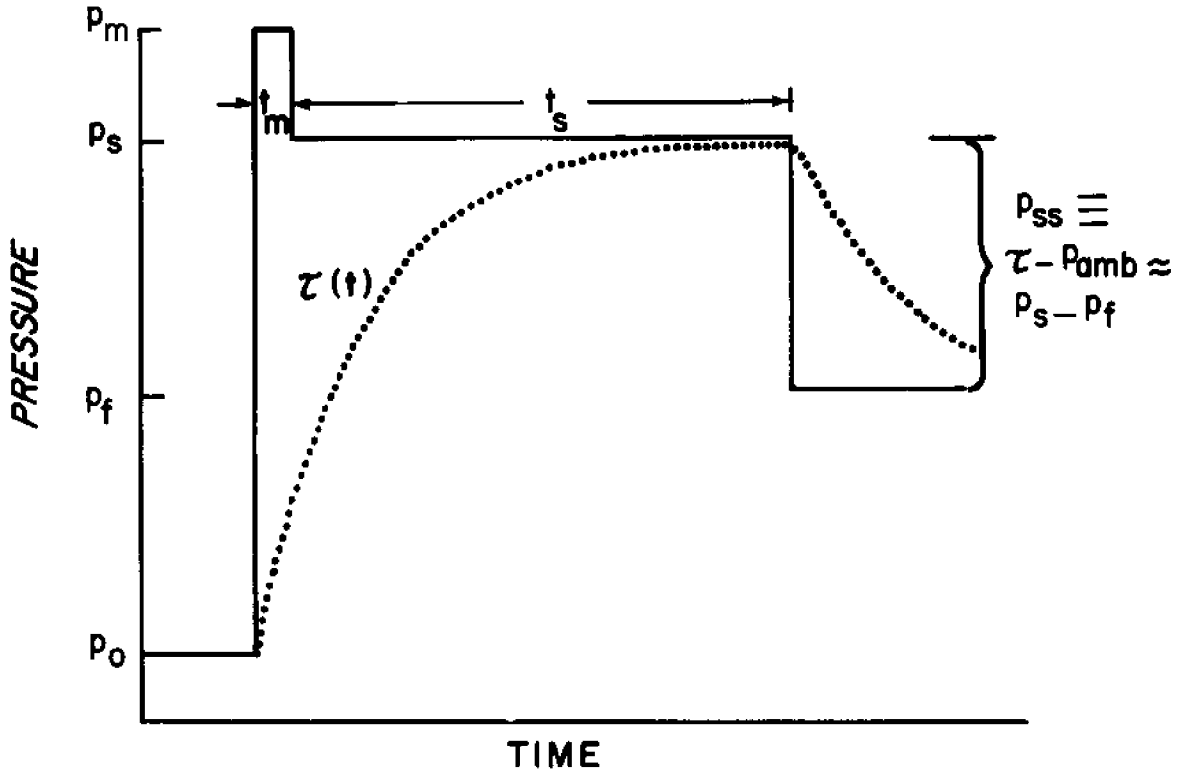

Fig. VA-2. Pressure schedule used by Hawaii group to achieve supersaturation of gelatin. Initial gas pressure is p_o, final pressure is p_f, and maximum pressure is p_m. Some minutes (t_m) after maximum pressure p_m is reached, pressure is lowered and held for several hours (t_s) at p_s until saturation of sample by surrounding gas is complete. Gas tension $\tau(t)$ approaches p_s with a time constant that is longer than t_m and much shorter than t_s. Initial spike to p_m is varied to test hydrostatic crushing of pre-existing gas nuclei.

Critical Radius. The most important feature of bubble formation and growth is that it is a threshold phenomenon: once the threshold pressure, negative or positive, is exceeded, the vapor or gas phase grows spontaneously until the pressure is relieved and the system is again in equilibrium. This point can be demonstrated by considering a spherical gas phase of radius r embedded in a supersaturated liquid. Such a cavity will expand or contract by diffusion according to the equation (17,23)

$$dr/dt \propto [\tau - p_b] \qquad (2a)$$

where p_b is the pressure inside the cavity

$$p_b = p_{amb} + (2\gamma/r) \tag{2b}$$

Substitution gives

$$dr/dt \propto [\tau - p_{amb} - (2\gamma/r)] \tag{2c}$$

where γ is the surface tension and where the vapor pressure and gravitational hydrostatic pressure have been neglected. A more explicit expression for the rate of change of the radius dr/dt is derived in the Appenix.

Evidently, the radius of a gas phase increases or decreases according to whether the quantity in brackets in Eq. 2c is positive or negative. Furthermore, the process is self-sustaining: if r increases, the quantity in brackets also increases, and the radius grows even faster. Conversely, if the quantity in brackets is negative, the radius decreases in a self-sustaining manner. These considerations lead to the notion of critical radius, i.e., that radius which makes the bracketed quantity equal zero

$$r_c = 2\gamma/(\tau - p_{amb}) \tag{3a}$$

We reserve the term "nucleus" for gas or solid phases having characteristic radii less than r_c, and we use the word "bubble" in referring to gas phases of characteristic radius larger than r_c.

Equation 3a can be applied to the decompression of a saturated liquid as follows. Immediately prior to a rapid decompression, the sample is at equilibrium at the saturation pressure p_s (i.e., $\tau = p_s$), as shown in Fig. VA-2. Immediately after a rapid decompression from p_s to p_f, the ambient pressure is p_f ($p_{amb} = p_f$). The critical radius and supersaturation pressure are then related by

$$r_c = 2\gamma/(p_s - p_f) = 2\gamma/p_{ss} \tag{3b}$$

$$p_{ss} = p_s - p_f = 2\gamma/r_c \tag{3c}$$

To every supersaturation pressure p_{ss} there corresponds a critical radius r_c such that any gas phase in the sample with characteristic radius larger than r_c will grow into a macroscopic bubble.

Homogeneous Nucleation. How can a gas nucleus originate in a liquid free of impurities? At the most primitive level, the random motion of the molecules in the liquid can, from time to time, deplete a given region and leave behind a space. This process, known as homogeneous nucleation, was first proposed by Becker and Doering (7,15, 16) and is described in well-known texts (18,30). The instantaneous local probability for the formation of such a cavity is calculated

from the Boltzmann factor

$$w \propto \exp(-E/kT) \tag{4}$$

where k is the Boltzmann constant, T is the absolute temperature, and E is the change in free energy associated with the process being considered.

The Boltzmann factor has been used to calculate such diverse quantities as the equilibrium concentration of the ortho and para states of liquid hydrogen, the temperature of the sun, the probability of forming pions at 400 billion electron volts, and the composition of the universe 0.1 second after the big bang. In the present application, we require only the temperature T and the formation energy for a spherical volume of radius r in which the liquid has been replaced by vapor or gas

$$E = - [(p_b - p_{amb})4\pi r^3/3] + [\gamma 4\pi r^2] \tag{5}$$

where all of the symbols are as previously defined. The negative term represents the energy lost by the system in the form of work done by the pressure differential $(p_b - p_{amb})$ in creating the spherical volume $4\pi r^3/3$, and the positive term is the energy gained by the system in the form of work done against the surface tension in creating the spherical surface $4\pi r^2$. The chemical potential is the same for liquid and vapor states of a given substance in thermal equilibrium, and it subtracts out when the change in free energy is calculated.

The radius for instantaneous mechanical equilibrium can be found by setting the derivative dE/dr equal to zero, the condition for a minimum of the energy E

$$dE/dr = - [(p_b - p_{amb})4\pi r^2] + [\gamma 8\pi r] = 0 \tag{6}$$

Alternatively, one can set the outward (positive) volume force equal to the inward (negative) surface force

$$(p_b - p_{amb})4\pi r^2 = \gamma 8\pi r \tag{7}$$

The equilibrium radius in either case is

$$r = 2\gamma/(p_b - p_{amb}) \tag{8}$$

By means of Eq. 8, the change in free energy can now be written

$$E = 16\pi\gamma^3/3(p_b - p_{amb})^2 \tag{9a}$$

$$= 4\pi r^2\gamma/3 \tag{9b}$$

while the corresponding instantaneous, local probabilities are

$$w \propto \exp[-16\pi\gamma^3/3kT(p_b - p_{amb})^2] \tag{10a}$$

$$\propto \exp[-4\pi r^2\gamma/3kT] \tag{10b}$$

The radius, and thus the formation energy, of any cavity must lie between zero and infinity. The proportionality constant for Eq. 10b can then be found by requiring that the integral from zero to infinity over the energy eigenstates must equal one. The result is (43)

$$w \simeq (1/kT)\ \exp(-4\pi r^2\gamma/3kT) \tag{11}$$

Finally, the instantaneous, local probability W for the occurrence of a vapor or gas phase of radius larger than the critical radius can be found by integrating Eq. 11 over the energy eigenstates from the critical energy, $4\pi r_c^2\gamma/3$, to infinity (43)

$$W \simeq \exp(-4\pi r_c^2\gamma/3kT) \tag{12}$$

To get some feeling for Eq. 12, we apply it to water at 27°C $\simeq$ 300°K (γ = 72 dynes/cm) (41). For a critical radius of r_c = 10 Å, the result is $W \simeq 1.4 \times 10^{-32}$. This is the probability of finding a cavity larger than 10 Å in radius at any given point in the liquid and at any given instant in time. This is a small probability, but there are many points and many instants. For example, the number of spherical volumes of 10 Å radius in 1 cm^3 of water is

$$n = 1\ cm^3/[4\pi(10^{-7}\ cm)^3/3] \tag{13a}$$

$$= 2.4 \times 10^{20}\ volumes/cm^3 \tag{13b}$$

How long is an instant? The duration of a vapor or gas phase may be estimated from the time required by a water molecule to traverse a cavity of 20 Å diameter traveling at the speed of sound in water, of order 10^5 cm/s

$$t \sim 2 \times 10^{-7}\ cm/(10^5 cm/s) \tag{14a}$$

$$\sim 2 \times 10^{-12}\ s \tag{14b}$$

Thus the number of cavities of 10 Å radius or larger formed per second per cm^3 of water is of the order

$$N \sim 0.5 \times 10^{12} \times 2.4 \times 10^{20} \times 1.4 \times 10^{-32} \tag{15a}$$

$$\sim 1.7\ cavity/s/cm^3 \tag{15b}$$

A critical radius of 10 Å corresponds via Eq. 3c to a tensile pressure or supersaturation pressure of about 1440 atm. In principle, if one applies a pressure of this magnitude to a pure water sample of 1-cm^3 volume at 300°K, it will fracture or cavitate within about 1 s. If the above calculation is repeated for r_c = 9 Å, the probability for cavitation is increased by six orders of magnitude, while for r_c = 11 Å, it is decreased by a similar factor. The calculation is equally sensitive to small changes in tensile or supersaturation pressure. The critical radius for homogeneous nucleation is therefore about 10 Å and the tensile strength or critical pressure about 1440 atm. This result is dominated by the Boltzmann factor and is relatively independent of the other assumptions in the calculation.

The minimum supersaturation pressure required to induce bubble formation in humans is about 0.6 atm (21), while the threshold for distilled water is about 0.8 atm (42). The difference between these values and the threshold for homogeneous nucleation is impressive and underscores the importance of understanding any other forms of nucleation that may be involved.

Inhomogeneous Nucleation. The theory of homogeneous nucleation works remarkably well for certain liquids, such as ether and n-hexane (4,5) in which nearly perfect denucleation has been achieved. The highest tensile strength actually observed experimentally for water is 277 atm (5,11), while the highest supersaturation pressure sustained without cavitation is 270 atm, found with helium gas (26). The close similarity of these values may be fortuitous, yet both are about a factor of 5 below the predictions of homogeneous nucleation theory. This is generally taken as evidence that impurities, such as motes, are still present even in the highly denucleated water used in obtaining such limits on de novo formation of bubbles (3).

Plesset (34) has developed a model for inhomogeneous nucleation in which impurities are approximated by solid hydrophobic spheres of radius r_o, as shown in Fig. VA-3, A. The Boltzmann factor is then used to calculate the probability that a vapor or gas phase will develop within a concentric shell extending from r_o to a larger radius r, as indicated in Fig. VA-3, B. The formation energy, analogous to Eq. 9b, is

$$E = [4\pi r^2 \gamma_{LV}/3] + [4\pi r_o^2(\gamma_{VS} - \gamma_{LS})/3] \qquad (16)$$

where γ_{LV}, γ_{VS}, and γ_{LS} are the surface tensions associated with the liquid-vapor, vapor-solid, and liquid-solid interfaces, respectively.

Equation 16 expresses the dependence of the formation energy, and thus the formation probability, upon the properties of the vapor-liquid-solid interface. At equilibrium, the surface tensions pulling parallel to their respective surfaces have a net component

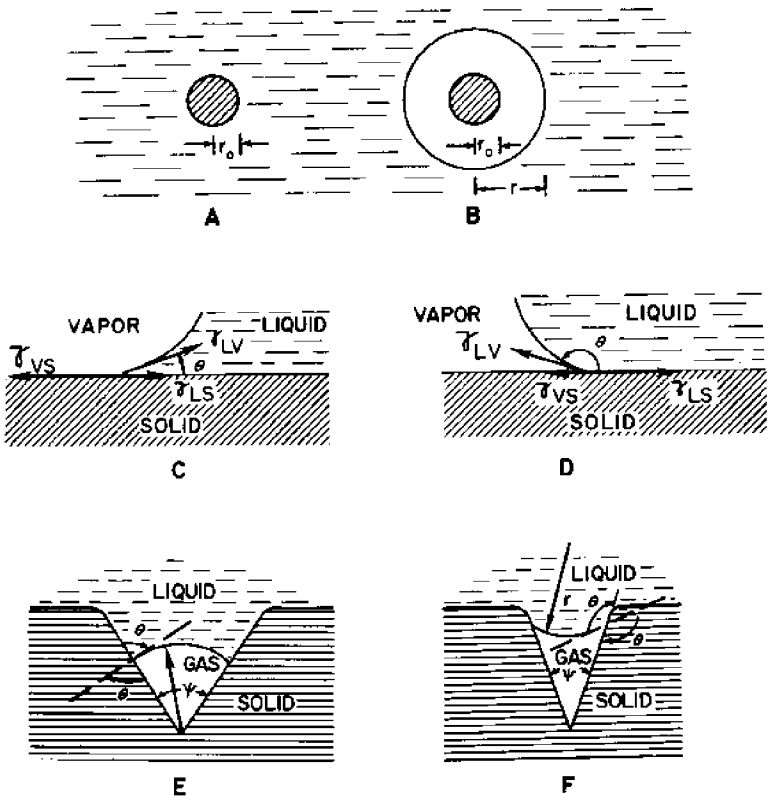

Fig. VA-3. A, B: Plesset model. Nucleation center consists of a solid hydrophobic sphere of radius r_o, and probability is calculated for a vapor phase to develop within a concentric shell extending from r_o to a larger radius r. C, D: solid-liquid-vapor interfaces for hydrophilic (C) and hydrophobic (D) solids. E, F: Harvey model. A gas phase characterized by a positive radius of curvature (E) collapses by diffusion, while one having a negative radius (F) grows by diffusion until a stable configuration is reached.

along the solid surface of zero

$$\gamma_{LV} \cos \theta = \gamma_{VS} - \gamma_{LS} \tag{17a}$$

as shown in Fig. VA-3, C and D. A solid is wetted by the liquid when

the contact angle Θ, measured through the liquid, is acute, that is, when

$$\gamma_{VS} - \gamma_{LS} > 0 \tag{17b}$$

In this case (Fig. VA-3, C), the second term in Eq. 16 is positive, and the probability of forming a bubble of radius r is decreased by the presence of a solid impurity. This is expected since a solid satisfying Eq. 17b has a greater adhesion for the liquid than the liquid has for itself.

For a hydrophobic or non-wetting solid satisfying the condition

$$\gamma_{VS} - \gamma_{LS} < 0 \tag{17c}$$

the second term in Eq. 16 is negative, and the formation energy is decreased, thereby increasing the formation probability via Eq. 4. In the limiting case, $\Theta = \pi$ ($\cos \Theta = -1$), the formation energy becomes

$$E = (4\pi r^2 \gamma_{LV}/3)[1 - (r_o/r)^2], \tag{18}$$

which is quite small for radii r near the impurity radius r_o. Thus the presence of hydrophobic motes implies a formation probability near unity for nuclei of radii comparable with those of the non-wetting solid impurities.

Experimental attempts to seed liquids with solid impurities have met with very little success. For example, Bateman and Lang (6) tried charcoal, blood corpuscles, dialyzed colloidal ferric hydroxide, ivory black, and sodium bicarbonate. Only the last two had any effect, and the results were inconclusive. Richardson (35), on the other hand, found that salt grains can carry nuclei into solution, but since the salt dissolves completely, the residual nuclei must be gaseous rather than solid. Further, Richardson's technique--absorption of ultrasonics--is specifically sensitive to gas phases. More recently, Tom Kunkle and I have attempted to nucleate gelatin with polystyrene spheres of 0.117 μ and 0.397 μ (29). Our inability to generate bubbles on hydrophobic spheres caused us to reexamine Plesset's model in some detail (43).

We believe that the observed absence of spontaneously generated gas phases around hydrophobic, solid spheres is due to the rapidity with which the formation energy increases, once the radius r exceeds the mote radius r_o. While Plesset (34) does not discuss possible limitations in the shell thickness

$$\Delta r \equiv r - r_o \tag{19}$$

we believe that this quantity must exceed typical intermolecular distances if the concept of a vapor or gas phase is to have any physical significance. In water, this implies

$$r \gtrsim 3\ \text{Å} + r_o \tag{20}$$

In Fig. VA-4, we have plotted (-E/kT) versus the mote radius r_o for various values of Δr, where E is the formation energy in water

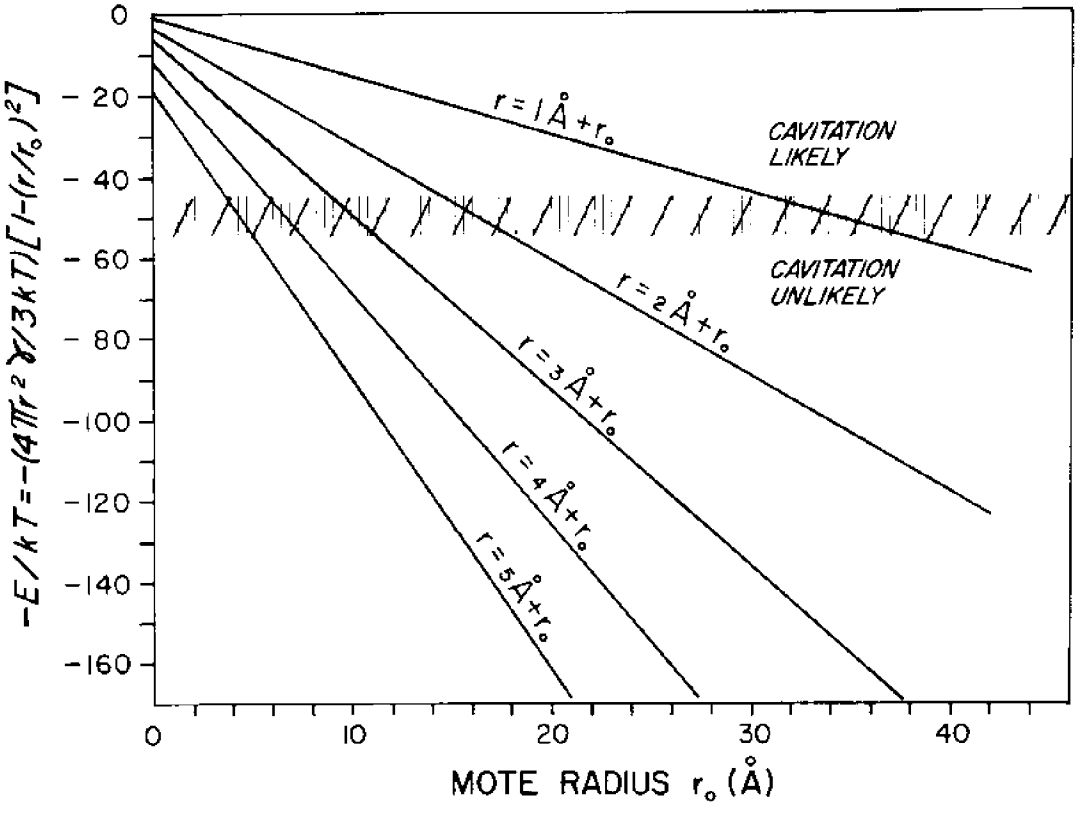

Fig. VA-4. Plot of (-E/kT) at 300°K versus mote radius r_o. Calculations are shown for several values of $\Delta r = r - r_o$ in the neighborhood of 3 Å, approximately average spacing between molecules in water. Gas nucleation is extremely unlikely unless $|-E/kT| \lesssim 50$.

given by Eq. 18, and T is taken to be 300°K. For characteristic times of order 10^{-12} s (Eq. 14b) and for mote densities of less than $10^6/cm^3$, nucleation is extremely unlikely unless

$$|-E/kT| \lesssim 50 \tag{21}$$

If $\Delta r = 3$ Å, for example, then motes larger than $r_o \approx 10$ Å are not effective. A mote of $r_o = 10$ Å radius would occasionally generate a cavity of up to $r \approx 13$ Å radius, corresponding to a tensile strength or critical pressure of about 1100 atm (Eq. 3c), but this is still three orders of magnitude larger than the threshold for bubble formation in humans.

Harvey Model. Harvey (22,24) has proposed a nucleation model consisting of a gas phase trapped in a cone of apex angle Ψ. For a broad cavity, shown in Fig. VA-3, E, the contact angle satisfies the condition

$$\Theta < 90° + (\Psi/2) \tag{22a}$$

The radius of curvature of the liquid-gas interface is positive in this case, implying (Eq. 8) a gas pressure p_b that is greater than the ambient pressure p_{amb}. Neglecting vapor pressure and assuming that the gas tension τ in the liquid is equal to the ambient pressure p_{amb}, there is a net diffusion (Eq. 2c) of gas out of the gas phase and into the liquid. The gas phase shown in Fig. VA-3, E, is unstable and eventually disappears.

The gas phase shown in Fig. VA-3, F, on the other hand, satisfies the condition (22,24)

$$\Theta > 90° + (\Psi/2) \tag{22b}$$

It therefore has a negative radius of curvature and a negative pressure differential $\tau - p_b$, and it will grow by diffusion (Eq. 2a) until a stable condition is reached, stability being defined by (42)

$$\Theta = 90° + (\Psi/2) \tag{22c}$$

i.e., by a flat liquid-gas interface.

There is usually a considerable range between the advancing and receding contact angles. Within this range, the point of contact can remain fixed, and the contact angle can vary until Eq. 22c is satisfied. Vapor pressure can easily be taken into account. For example, if the vapor pressure of the liquid is higher inside the cavity than it is above the liquid, and if $\tau = p_{amb} = p_b$, then the stable interface is slightly convex ($r > 0$). Similarly, in living animals the inherent unsaturation resulting from oxygen metabolism (8,14,27) implies that τ is less than p_{amb} and that the stable liquid-gas interface is slightly concave ($r < 0$).

A crucial feature of this model is that gas phases can originate spontaneously by homogeneous nucleation near the apex of a cone.

Providing only a few molecules are involved, the formation energy E is small, and the formation probability (Eq. 4) rather large. Once started, growth can occur via the Harvey mechanism, i.e., via Eq. 2c when Eq. 22b is also satisfied. Since the radius of the liquid-gas interface is negative in this case, this process succeeds even when the gas tension is less than the ambient pressure, i.e., when there is no supersaturation.

The Harvey model demonstrates in principle that gas phases can be produced and stabilized within liquids and that they can grow to sufficient size (e.g., 1 μ) to account for the precocious onset of bubble formation in animals. Albano (2) has argued that the interstitial spaces between cell walls can provide the negative surfaces required to sustain spontaneous growth, and he has amassed an impressive body of histological evidence in support of this view. We note, however, that the Harvey model cannot account for the low threshold for bubble formation in distilled water (42), and that other mechanisms for nucleation, stabilization, and growth must be involved. Such mechanisms would be characteristic of aqueous media generally and could be important in vivo even if other processes also occur.

Reynold's Cavitation. Large tensile pressures can originate in liquids subjected to a sudden change in velocity. This occurs, for example, in the vicinity of moving torpedos, marine propellers, and hydraulic turbines. The well-known case of the "Reynolds tube" (Venturi nozzle) is shown in Fig. VA-5, A. For fluids flowing in a horizontal plane, the effect of gravity can be neglected, and the pressure differential is given by Bernoulli's equation

$$p_2 - p_1 = (\rho v_1^2/2) - (\rho v_2^2/2) \tag{23}$$

where ρ is the density of the liquid, p_1 and p_2 are the absolute pressure, and v_1 and v_2 are the velocities in regions 1 and 2, respectively.

In practice, flow rates of $v_1 \gtrsim 10$ m/s are required to induce cavitation in water (10). The corresponding pressure differential can be estimated by setting $\rho = 1$ gm/cm^3 and by assuming that v_2 is negligible. The result is

$$p_2 - p_1 \gtrsim 0.5 \text{ atm} \tag{24}$$

As expected, this is quite similar to the minimum supersaturation pressures (noted earlier) which induce bubble formation in humans (about 0.6 atm) and in distilled water (about 0.8 atm). Cavitation is possible at this level only because of nuclei already present in aqueous media generally. This underscores the essential point that Reynold's cavitation is simply another means of exerting a differential pressure. It is not a method for creating nuclei.

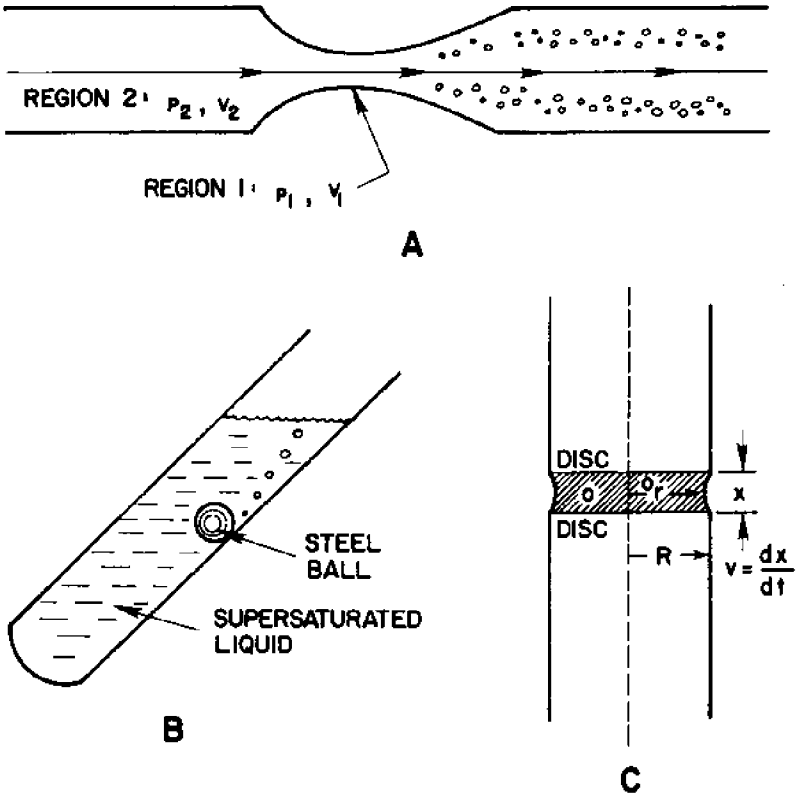

Fig. VA-5. A: Reynolds tube (Venturi nozzle). Increased velocity in Region 1 implies a decreased pressure, as compared to Region 2. B: tribonucleation of gas phases can occur when steel ball rolls along inside wall of test tube containing supersaturated water. C: Cottrell model for calculating tribonucleation pressure. Cavitation can occur when two discs are drawn rapidly apart.

Variations in blood velocity in animals are normally much less than 10 m/s and are well below the transition from laminar to turbulent flow. Harvey et al. (22) have found that velocity variations of at least 30 times the threshold for turbulence are required to produce cavities in liquids. It appears, therefore, that kinetic cavitation is not a major factor in decompression sickness, although Albano (2) has described pathological cases in which blood vessel injuries were directly related to specific decompression symptoms. Even in these instances, the pressure differentials resulting from changes in blood velocity must have been small compared to homogeneous nucleation thresholds. In this

sense, Reynold's cavitation was merely the last step in a process dependent mainly upon a high supersaturation pressure and an abundance of pre-existing nuclei.

Tribonucleation. Tribonucleation (12,13,25,28) means literally "nucleation by rubbing". For example, bubbles can be generated by rubbing two solids together under supersaturated water. Bubbles can also be produced by allowing a steel ball to roll along the inside wall of a test tube containing supersaturated water, as shown in Fig. VA-5, B (25,28). Such bubbles develop because a negative tensile pressure is created when two solid surfaces within a liquid are pulled rapidly apart. Evidently, tribonucleation is a misnomer: like Reynold's cavitation, it is a means of exerting a differential pressure and not a method for creating nuclei.

Cottrell (13) has calculated the tribonucleation pressure p_t between two coaxial discs of radius R separated by a distance x that increases with a velocity $v = dx/dt$. The geometry is shown in Fig. VA-5, C, and the result is

$$p_t = - 3\eta v(R^2 - r^2)/x^3 \qquad (25)$$

where η is the viscosity, the r is the radial distance from the common axis of the discs to some point between the discs. The tribonucleation pressure p_t and supersaturation pressure $(\tau - p_{amb})$ from Eq. 3a can be combined into the single equation

$$\tau - p_{amb} - p_t = 2\gamma/r_c \qquad (26)$$

Since p_t is negative, the two pressures work together in lowering the critical radius for nuclei to grow into bubbles.

Campbell (12) has carried out extensive calculations of both p_t and $(\tau - p_{amb})$ and has concluded that homogeneous nucleation may be feasible in water by this mechanism. This point can be illustrated by substituting into Eq. 25 the following parameter values: $\eta = 0.01$ gm/cm-s, $r = 0$, $R = 0.1$ cm, $v = 0.1$ cm/s, and $x = 0.27$ μ. The result is $p_t = - 1440$ atm, approximately the threshold for homogeneous nucleation. The values of R and v are modest, and the disc separation is many molecular diameters.

Experimentally the situation is complicated by the difficulty in denucleating water samples and solid surfaces. Ikels (28) was able to generate bubbles in olive oil and glycerol-water solutions with a steel ball rolling at speeds in the range from 0.7 to 3.5 cm/s and with various gases at supersaturation pressures of less than 1 atm. With denucleated water saturated with N_2 gas at 1 atm, however, no bubbles were observed even at ambient pressures below the vapor pressure ($p_{ss} \sim 1$ atm) and with speeds of up to 3.5 cm/s. Clearly the threshold for homogeneous nucleation in water was not

reached in these experiments. Nevertheless, tribonucleation could become important when the supersaturation pressure is large or when there are already abundant nuclei present. In particular, Ikels (28) has suggested that the articulating surfaces of animal joints could cause bubble formation in the synovial fluid by this mechanism.

Nucleation by Ionizing Radiation. Homogeneous nucleation, inhomogeneous nucleation, and the Harvey model all depend upon the random motion of molecules to form vapor or gas cavities. Similarly, Reynold's cavitation and tribonucleation are methods of applying tensile pressures: they result in bubble formation only if nuclei are provided by some other process, such as random motion. In sharp contrast, ionizing radiation is capable of generating gas nuclei of sufficient size ($\sim$ 1 μ) to account for the precocious onset of bubble formation in aqueous media generally (9,31,37,38).

Essentially, ionizing radiation creates nuclei by supplying the necessary formation energy in a volume comparable with the nuclear dimensions. The kinetic energy E of a primary charged particle is dissipated in most cases mainly by numerous soft collisions with atomic electrons. The recoil electrons are called δ rays, and their energies are usually less than 1 keV, although much higher energies are possible in rare cases. Most δ rays therefore stop within a few microns of the path of the primary via additional soft collisions that leave behind atoms in excited states, ions, and more δ rays.

It is a crucial feature of the ionization process that at low primary kinetic energies $E = mv^2/2$, the rate of energy loss along a primary path x varies as

$$dE/dx \propto 1/E \propto 2/mv^2 \quad \text{(low-energy limit)} \tag{27a}$$

where m is the mass of the primary and v the velocity; while at high energies, the ionization rate is minimal and approximately constant

$$dE/dx \simeq \text{constant} \quad \text{(high-energy limit)} \tag{27b}$$

This means, for example, that the energy density is relatively high in the vicinity of a stopping δ ray and relatively low along the path of a cosmic-ray muon.

In a bubble chamber (20) filled with superheated liquid hydrogen, the critical radius is about 40 Å (36) and the threshold energy about 500 eV (36). The rate of energy deposition required to form a bubble is thus of order

$$dE/dx \sim 500 \text{ eV}/80 \text{ Å} \tag{27c}$$

$$\sim 6 \times 10^8 \text{ eV/cm} \tag{27d}$$

Such ionization densities cannot be reached by high-energy particles per se since their average dE/dx is about 3×10^5 eV/cm in hydrogen. However, minimum ionizing particles can generate about 15 δ rays per cm with stopping energy densities above this threshold. This results in about 15 bubbles per cm being formed along the primary track.

A similar calculation for water (37) predicts that ionization energy densities of order

$$dE/dx \sim 5 \times 10^{10} \text{ eV/cm} \tag{27e}$$

are required to form cavities of 1-μ radius. The ultrasonic-cavitation experiments of Sette and Wanderlingh (37) indicate that oxygen nuclei, recoiling from collisions of cosmic-ray neutrons with water molecules, are responsible for the formation of gas nuclei in water. Because of their large mass in comparison with δ rays, recoil oxygen nuclei can reach quite substantial ionization levels, up to $6\text{-}7 \times 10^9$ eV/cm, near the ends of their short trajectories. Evidently, however, this is insufficient by itself to explain the observed cavitation, and a more complex mechanism must be involved.

Sette and Wanderlingh have noted that the density of δ rays with energy above 100 eV can reach about 5×10^6 per cm near the end of a recoil oxygen trajectory. The high density, in turn, permits the δ-ray micronuclei to coalesce, forming cylindrical cavities that may be 10 μ long and may initially have radii of about 20 Å. The temperature within the cavity is about 600°K, and it contains O_2 and H_2 gas from radiolysis, as well as water vapor, excited atoms, ions, and free electrons (37). The cavity is thus both super-saturated and superheated, and this permits it to grow and reorganize itself in a more stable configuration. That this actually happens is evidenced by the experimental observation (37) that gas nuclei formed in this way are stable for periods on the order of hours.

The marginal nature of the process just described is suggested by the experimental observation (37) that recoil hydrogen nuclei (protons) are not able to generate stable gas phases in water, although the δ-ray density is only a factor of 10 lower than for recoil oxygen nuclei. On the other hand, recoil carbon nuclei are able to produce cavities in pentane and acetone by this mechanism (31). Finally, it should be noted that the equilibrium density of gas nuclei at cavitation threshold due to cosmic rays was only about 25 per liter in the experiments of Sette and Wanderlingh and that the number was not much higher when a $Po^{210}Be^{13}$ source was used.

Tissue Elasticity. In the series of experiments on bubble formation in gelatin carried out at the University of Hawaii (29,39, 40,42), a gelatin mixture was chosen that barely permits gelation

at room temperature. In this case, elasticity is negligible, and samples closely approximate distilled water in the sense that surface tension is the controlling factor. The opposite extreme has been studied by Gent and Tompkins (19) in their work on nucleation and growth of bubbles in elastomers, i.e., foam rubber. The difference in pressure inside and outside the gas phase is given by (19)

$$P_b - P_{amb} = [(5/2) - 2(r_o/r) - (1/2)(r_o/r)^4]G + 2\gamma/r \qquad (28)$$

where G is the shear modulus, and r_o is the radius of the cavity at the time of cross linking. Clearly the term in brackets results from the elasticity and is zero when $r = r_o$ (unstretched spring). This is also the equilibrium radius when the surface tension is zero.

The feature of Eq. 28 which we wish to emphasize is that there is an equilibrium radius even when the surface tension is not zero. In this case the tendency of the surface tension to collapse the gas phase is resisted by the elasticity term, which is negative when r is less than r_o and thus tends to restore the cavity to its original shape. Here we note that at least 99.9% of the bubbles produced by decompressing gelatin can be eliminated by hydrostatically crushing the gas nuclei that originate them. In gelatin, where the elasticity is negligible, the crushing process is irreversible. The studies of Aggazzotti and Ligabue (1,2) on the compressibility of dog tissue suggest that gas phases are also present in this medium, but in this case the crushing is partially reversible--presumably because of the significant elasticity. Typical restoration times are of the order of minutes or hours.

Discussion

The central theme of this review has been that bubble formation is a general property of decompressed aqueous media, including humans. Furthermore, the process is initiated primarily by pre-existing gas nuclei. There are then two fundamental questions: 1) How are gas nuclei created? 2) How are they stabilized? In my opinion, we do not yet have a satisfactory answer to either question. The Harvey model, in which gas phases originate and grow spontaneously within hydrophobic cracks, may be capable of explaining bubble formation in animal tissue, but it is difficult to understand how this mechanism can operate in distilled water. In the latter case, creation may result from ionizing radiation by cosmic rays, whereas stabilization may be due to the chemical properties of water, such as hydrogen bonding and the ability of clathrate structures to capture gas molecules (32,33).

Appendix: Rate of Change of Bubble Radius dr/dt

An essential feature of this review, stated by Eq. 2a, is that the rate of change of bubble radius dr/dt is proportional to $(\tau - p_b)$, the difference between the gas tension in the tissue and the pressure in the gas phase. A derivation of this relationship is given here. The derivation is similar to that of W. B. Wright (private communication, 1972) and yields an equation due originally to Harvey et al. (23).

The derivation begins with the following assumptions:

1) A single ideal gas is involved.
2) The gas phase is at rest with respect to the solvent.
3) Gravity can be neglected.
4) The gas phase is spherical
5) The gas phase is in quasi-equilibrium to that to which Eq. 2b applies.
6) The diffusion gradient is confined to a thin, uniform shell of thickness Δr at the boundry of the gas phase.

The ideal gas law can be expressed as

$$p_b V = nRT \tag{A1}$$

where p_b is the absolute pressure, n is the number of moles, T is the absolute temperature in the gas phase, and R is the universal gas constant. For spherical volumes V of radius r, Eq. A1 can be rewritten

$$p_b(4\pi r^3/3) = nRT \tag{A2}$$

which gives

$$n = 4p_b\pi r^3/3RT \tag{A3}$$

The gas pressure p_b is given by Eq. 2b

$$p_b = p_{amb} + (2\gamma/r) \tag{2b}$$

Substitution into Eq. A3 then yields

$$n = (4\pi r^3 p_{amb}/3RT) + (8\pi r^2\gamma/3RT) \tag{A4}$$

Differentiation with respect to time t gives

$$dn/dt = (4\pi r^2/RT)(dr/dt)[p_{amb} + (4\gamma/3r)] \tag{A5}$$

The flux dn/dt into the gas phase is also given by

$$dn/dt = DS\nabla c \tag{A6}$$

$$\simeq D4\pi r^2(c_o - c_i)/\Delta r \tag{A7}$$

where D is the diffusion coefficient, S is the spherical surface area $4\pi r^2$, ∇c is the gradient of the gas concentration, which is approximated here by the difference in concentration at the outer (c_o) and inner (c_i) boundries of the diffusion shell, divided by the shell thickness Δr. The concentrations can be expressed in terms of the solubility "a" of the gas and by the gas tension τ and gas pressure p_b by Henry's law

$$c_o = a\tau \tag{A8}$$

$$c_i = ap_b \tag{A9}$$

which gives

$$dn/dt = (aD/\Delta r)4\pi r^2(\tau - p_b) \tag{A10}$$

Substitution of p_b from Eq. 2b yields

$$dn/dt = (aD/\Delta r)4\pi r^2[\tau - p_{amb} - (2\gamma/r)] \tag{A11}$$

By setting the two expressions (Eqs. A5, A10) for the flux dn/dt equal and solving for the rate of change of radius, one obtains

$$dr/dt = (RTaD/\Delta r)[\tau - p_{amb} - (2\gamma/r)]/[p_{amb} + (4\gamma/3r)] \tag{A12}$$

$$= (RTaD/\Delta r)[(\tau - p_{amb})/p_{amb}][r - \{2\gamma/(\tau - p_{amb})\}]/[r + (4\gamma/3p_{amb})] \tag{A13}$$

which is essentially the equation given by Harvey et al.

Equation A12 leads immediately to our Eq. 2c, and it gives Eq. 2a in one step via Eq. 2b. Epstein and Plesset (17) have derived and solved the diffusion equation without invoking assumptions 5 and 6 above. Their result differs somewhat from that of Harvey et al., but it agrees in this crucial respect, namely, that both differential equations contain the driving factor

$$F_1 = [\tau - p_b] = [\tau - p_{amb} - (2\gamma/r)] \tag{A14}$$

of Eq. A12 or the analogous factor

$$F_2 = [r - \{2\gamma/(\tau - p_{amb})\}] \tag{A15}$$

of Eq. A13.

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B. THE PHYSICS OF BUBBLE FORMATION: IMPLICATIONS FOR IMPROVEMENT OF DECOMPRESSION METHODS: D. E. YOUNT, R. H. STRAUSS, E. L. BECKMAN, AND J. A. MOORE

Previous decompression tables for humans have been based on unsupported assumptions and trial and error. Most of the diving schedules in use today were developed by such methods, mainly because the underlying processes of nucleation, stabilization, and growth of gas phases in blood or tissue were poorly understood. To the population at risk, the cost of empiric investigation has been high, and to some extent it is still being paid, in terms of diving diseases such as osteonecrosis, damaged-brain syndrome, and spinal cord damage.

The recent development of the gelatin model (13,15,16,21) has permitted many of the physical factors connected with the causes of decompression sickness to be evaluated. It has been found (21), for example, that pre-existing gas nuclei account for at least 99.9% of the bubbles formed by decompressing gelatin and that about 93% of these are already present in the water used in preparing the gel. Similarly, the Haldane-ratio principle (4) is not applicable to gelatin.

To a first approximation, the number of bubbles formed for a given gas is a function only of the supersaturation pressure and underlying radial distribution of gas nuclei. The principle of zero-supersaturation decompression (3,7,11) is also inapplicable and does not permit optimal decompression rates, since these underlying distributions of nuclear size often allow relatively large supersaturation pressures to be withstood without bubble formation. Crushing of gas nuclei has been studied quantitatively in gelatin and it has been shown that the number of bubbles can be greatly reduced by rapid compression or by pressure spikes at the beginning of a dive schedule. As a result of these experiments, it is now possible to compute mathematically optimal decompression schedules that are safer and faster for gelatin at a given depth and duration than the U.S. Navy tables (21).

Gelatin has in common with living systems the fact that both are aqueous media rich in proteins. On the other hand, there are obvious differences, and the outcome of the above comparisons of optimum and U.S. Navy schedules is perhaps to be expected. The real significance of the gelatin work is that there is at least one system in nature that can now be safely decompressed on the basis of the physical laws and phenomena reviewed in the paper by Yount (20).

Methods for calculating optimal schedules for gelatin are discussed below, and experimental comparisons with other tables are summarized. In Section III, our first attempts to prepare decompression tables for humans *a priori* are described, and the results compared with other well-known procedures.

Optimal Decompression of Gelatin

The criterion that must be satisfied if a physiologically insignificant number of bubbles is to form during the decompression of gelatin is (21)

$$p_{ss} \equiv (\tau - p_{amb}) \leq 2\gamma/r_c^{max} \tag{1}$$

where p_{ss} is the supersaturation pressure, τ is the gas tension in the sample, p_{amb} is the ambient hydrostatic pressure, γ is the surface tension, and r_c^{max} is the maximum critical radius at which a significant number of nuclei are present. In effect, the supersaturation pressure p_{ss} causes gas phases of radius larger than r_c^{max} to grow into bubbles: if none are present, no bubbles will result. Equation 1 must be satisfied at all points within the sample, and a range of time constants (calculable from the gelatin depth) must be taken into account.

Zero-supersaturation decompression may be regarded as a special case of Eq. 1 that is applicable when the right-hand side is zero, i.e., when the maximum critical radius is arbitrarily large. Since the ambient pressure p_{amb} is then always greater than or equal to the gas tension τ, zero-supersaturation decompression of gelatin is precluded by definition.

Implementation of Eq. 1 requires a knowledge of the two parameters γ and r_c^{max} for the sample and pressure history being studied. Alternatively, one can measure the maximum safe supersaturation pressure directly

$$p_{ss}^{*} = 2\gamma/r_c^{max} \tag{2}$$

for various test conditions. We shall adopt the second approach here, noting that (21)

$$\gamma_{gel} = \gamma_{sol} = (51 \pm 5) \text{ dynes /cm} \tag{3}$$

so that r_c^{max} can always be calculated from

$$r_c^{max} = [2(51 \pm 5) \text{ dynes/cm}]/p_{ss}^{*} \tag{4}$$

The relevant data are shown in Fig. VB-1, where p_{ss}^{*} is plotted as a function of the crushing pressure $(p_m - p_o)$. The test schedule was that of Fig. VA-2 in the article by Yount (20) with a mixing pressure of $p_o = 0$ psig. For a sample saturated at the maximum pressure ($p_s = p_m$) and decompressed rapidly to a final pressure p_f, the supersaturation pressure is

$$p_{ss} \equiv (\tau - p_{amb}) = (p_s - p_f) \tag{5}$$

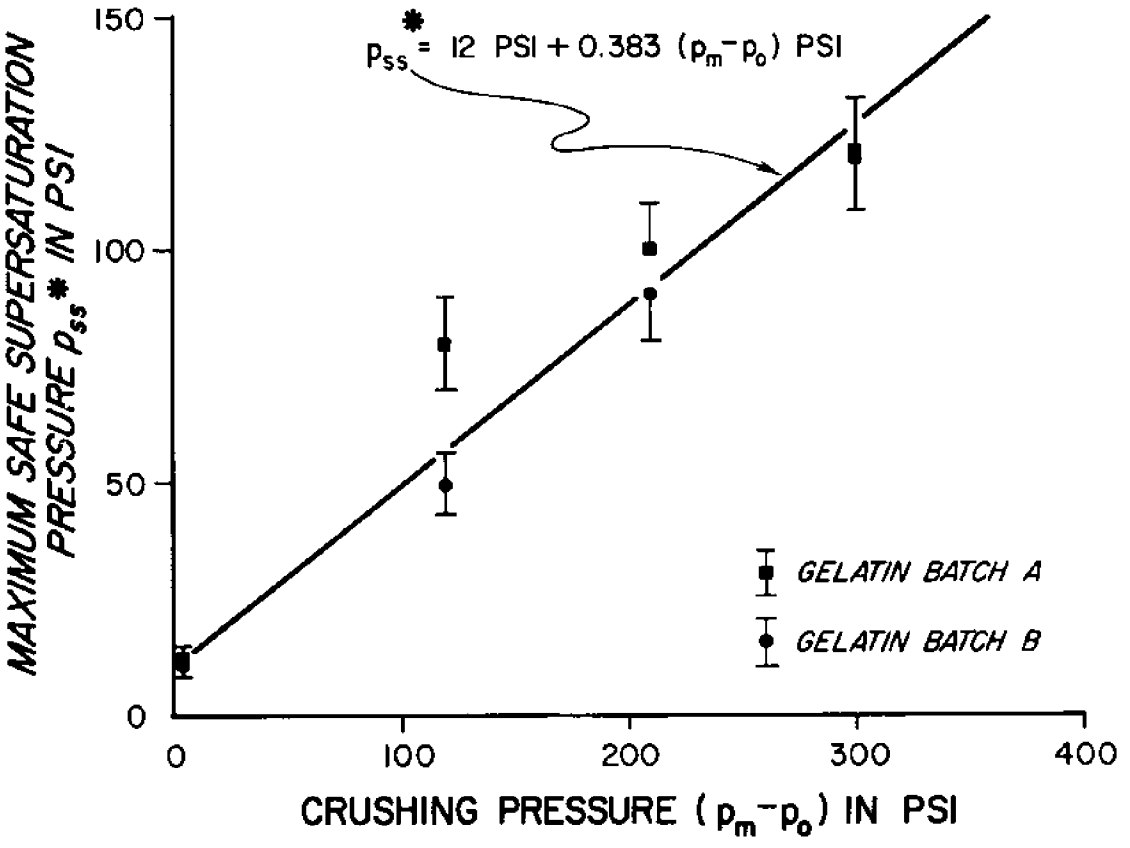

Fig. VB-1. Plot of maximum safe supersaturation pressure p_{ss}^{*} in psi versus crushing pressure $(p_m - p_o)$ in psi for 2 gelatin batches mixed at 0 psig. A reasonable fit to the data is given by linear relation $p_{ss}^{*} = 12 \text{ psi} + 0.383 \ (p_m - p_o) \text{ psi}$.

We arbitrarily define the maximum safe supersaturation pressure p_{ss}^{*} to be the threshold for producing one bubble per sample and, conceptually, decrease p_f until one bubble is seen. In practice, it is more accurate to plot the bubble count versus p_{ss}, (as in (21)), and extrapolate to the one-bubble intercept p_{ss}^{*}. The data in Fig. VB-1 can be summarized by the relation

$$p_{ss}^{*} = 12 \text{ psi} + 0.383 \ (p_m - p_o) \text{ psi (gelatin)} \qquad (6)$$

which has a χ^2 per degree of freedom of about 1.0.

We mentioned in the introduction that the number of bubbles formed in gelatin is a function only of the supersaturation pressure for a given underlying radial distribution of gas nuclei.

The underlying distributions are independent of the mixing pressure so that, for example, batches mixed in air at p_o = 100 psig can be safely decompressed to about 88 psig, and batches mixed in air at p_o = 0 psig can be safely decompressed to -12 psig (21). It is not yet known whether gelatin batches mixed at different pressures p_o are equally sensitive to the crushing pressure (p_m - p_o) when this quantity is different from zero.

Following Ref. 21, we now apply Eq. 1 to the special case of a fully saturated gelatin sample. The shortest decompression time that is consistent with negligible bubble formation (optimal schedule) is obtained by assuming the equals sign

$$(\tau - p_{amb}) = 2\gamma/r_c^{max} = p_{ss}^* \tag{7}$$

The slowest "tissue type" occurs at the bottom of the saturated sample and is characterized by a single time constant σ^*. The diffusion equation (7),

$$d\tau/dt = [p_{amb}(t) - \tau(t)]/\sigma^* \tag{8}$$

reduces to

$$d\tau/dt = [-2\gamma/r_c^{max}]/\sigma^* = - p_{ss}^*/\sigma^* \tag{9}$$

The right-hand side of Eq. 7 is independent of time so that differentiation gives

$$dp_{amb}/dt = d\tau/dt = - 2\gamma/\sigma^* r_c^{max} = - p_{ss}^*/\sigma^* \tag{10}$$

The optimum decompression schedule for a saturated gelatin sample is thus remarkably simple. It consists of only two phases

1) a rapid initial ascent to

$$p_{amb}^* = \tau^* - (2\gamma/r_c^{max}) \tag{11}$$

where τ^* is the gas tension immediately prior to decompression, and

2) a linear decompression that begins at the end of the first step and decreases with constant slope given by Eq. 10 until the final pressure p_f is reached.

The variation of the gas tension τ at the bottom of a saturated gelatin sample is also remarkably simple during optimal decompression. Beginning at τ^*, the gas tension follows another straight-line parallel to $p_{amb}(t)$ and displaced above it by $2\gamma/r_c^{max} = p_{ss}^*$. After $p_{amb}(t)$ reaches p_f, the decay of the gas tension is exponential, and τ approaches p_f asymptotically.

In Fig. VB-2, three decompression schedules are compared for a 40-minute dive to a water depth of p_s = 100 ft = 44 psig = 4 ATA. The first is the standard U.S. Navy table (18). The second, designated "empirical", is of the optimal form with first pull and constant slope determined by trial and error.

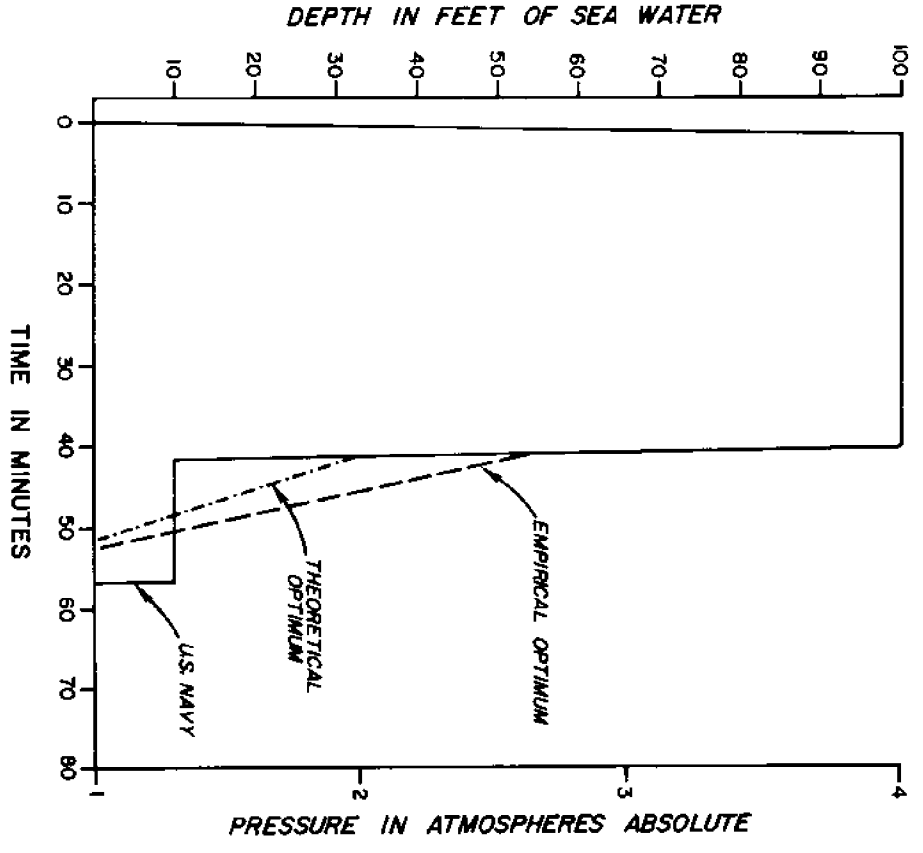

Fig. VB-2. Comparison of empirical optimum, theoretical optimum, and U.S. Navy decompression schedules for a 40-min dive to water depth of 100 ft. U.S. Navy table required 17 min to surface and yielded (12.9 ± 1.0) bubbles per sample; empirical optimum schedule required 12 min and yielded (0.42 ± 0.19) bubbles per sample; and the theoretical optimum schedule required 11 min and yielded (0.00 ± 0.17) bubbles per sample.

The third schedule shown in Fig. VB-2, labeled "theoretical", was calculated as follows. First, the crushing pressure

$$(p_m - p_o) = 4 \text{ ATA} - 1 \text{ ATA} = 3 \text{ atm} = 44 \text{ psi} \qquad (12)$$

was determined. Second, the crushing pressure was substituted into Eq. 6 to obtain the maximal safe supersaturation pressure

$$p^*_{ss} = 12 \text{ psi} + 0.383 \times 44 \text{ psi} = 29 \text{ psi} = 2.0 \text{ atm} \qquad (13)$$

which is also the magnitude of the first pull for a saturated sample. Next, the time constant for diffusion at the bottom of the 2-mm-deep sample was found (21) using the measured diffusion constant for nitrogen in a gel of 1-2% agar in water (2)

$$\underline{D} = 11 \times 10^{-4} \text{ cm}^2/\text{min} \qquad (14)$$

and the calculations of Crank (6) for an infinite plane slab of uniform thickness. This gave a time constant at the bottom of the sample of (21)

$$\sigma^* = 18.5 \text{ min} \qquad (15)$$

corresponding to about 88% saturation at this location. The slope was then determined from Eq. 10

$$dp_{amb}/dt = - p^*_{ss}/\sigma^* = 2.0 \text{ atm}/18.5 \text{ min} = 0.11 \text{ atm/min} \qquad (16)$$

For comparison we note that the first ascent for the empirical schedule was 1.3 atm and the slope 0.15 atm/min.

The U.S. Navy table required 17 minutes to surface and yielded (12.9 ± 1.0) bubbles per sample, averaged over 12 samples. The empirical optimal schedule required 12 minutes and yielded (0.42 ± 0.19) bubbles per sample, averaged over 12 samples. Finally, the theoretical optimal schedule required 11 minutes and yielded (0.00 ± 0.17) bubbles per sample, averaged over 9 samples.

Decompression of Humans A Priori

In this section we describe our initial attempts to generate decompression tables for humans based on the physical ideas and physiological parameters discussed above and in the Yount paper (20). We begin by assuming that human blood and tissue are similar to gelatin. We then modify and extend the development of the section on optimal decompression of gelation to accommodate such additional information as may be relevant and available at this time. We anticipate that other changes will be required as the data and our understanding improve, but we believe that it is important to determine as early in the program as possible whether this approach

is feasible. Specifically, we are looking for a pressure schedule $p_{amb}(t)$ that satisfies Eq. 1 at every point within the body

$$p_{amb}(t) \geq \tau(t) - 2\gamma/r_c^{max} = \tau(t) - p_{ss}^* \qquad (17)$$

In calculating p_{ss}^*, we have assumed that the body is homogeneous, and we have modified Eq. 6 in two ways:

1) We have changed the no-crushing bubble-formation threshold from 12 psi for gelatin to 5.9 psi for humans. This is based on a nitrogen partial pressure of 79% × 14.7 psia = 11.6 psia at sea level and on a range of 23,000 - 25,000 feet (5.9 to 5.5 psia) quoted as the minimum bends altitude by Gray (9) as a result of massive trials on USAAF cadets. That is, we have subtracted 5.7 psia from 11.6 psia to obtain 5.9 psi.

2) We have taken into account restoration of gas nuclei via tissue elasticity, an effect which is negligible for gelatin. This was accomplished by allowing the crushing term to decay exponentially with a time constant of 44 minutes, which is within the range of 30 - 70 minutes found by Aggazotti and Ligabue (1) for dog gastrocnemius muscle to regain its original volume after being crushed to 11 ATA.

Thus we have replaced Eq. 6 for gelatin by the new equation

$$p_{ss}^* = 5.9 \text{ psi} + 0.383\ (p_m - p_o)\ \exp(-t/44 \text{ min}) \text{ psi (humans)} \qquad (18)$$

where t is the elapsed time since the crushing or compression occurred.

The a priori calculation of the gas tension τ is straightforward in the simple case of a homogeneous slab of gelatin having uniform thickness and accurately known diffusion coefficient D. In humans, on the other hand, one is dealing with a complex capillary-tissue geometry and a diffusion coefficient that is poorly understood and may vary with location, tissue type, time, and so on (10). Such complexity in detail tends to obscure the basic simplicity of the underlying phenomena, and we have chosen to avoid these issues at this early stage by using an existing computer program, TAMU, developed at Texas A&M University, to calculate the maximum value of $\tau(t)$, given as input an assumed set of tissue time constants σ_i.

In the calculations reported here, we have used three separate sets of tissue time constants, all of which are at least partially empirical. The first set consists of the single time constant σ_1 = 75 minutes, which duplicates the single tissue type of the British decompression procedures described in Work in Compressed Air Special Regulations, 1958. The second set of time constants, σ_1 = 1 minute and σ_2 = 128 minutes, was derived by Dieter (7) in fitting the U.S. Navy Standard Air Decompression Tables of 1958 (17). Finally,

we have used a set of 12 tissue half times ($\tau_{1/2} = 0.693\ \sigma$) from the TEKTITE saturation dive (bottom time of 14 days, breathing mix of 92.8% N_2 and 5.2% O_2, and depth of 100 fsw). The actual half times are: 5, 10, 20, 40, 80, 120, 160, 240, 320, 480, 560, and 720 minutes.

In Table VB-1, a number of decompression schedules are compared for a depth of 200 fsw and a bottom time of 30 minutes. These are:

1) U.S. Navy (18),
2) TAMU
3) Buehlmann (5),
4) Hills (12),
5) Royal Navy (14),
6) Fife (8)
7) Yount-GB (based on Eqs. 17 and 18 and a single exponential time constant of 75 minutes from the British compressed air regulations),
8) Yount-USN (based on Eqs. 17 and 18 and exponential time constants of 1 minute and 128 minutes derived by Dieter from the U.S. Navy tables),
9) Yount-TEKTITE (based on Eqs. 17 and 18 and tissue half times of 5, 10, 20, 40, 80, 120, 160, 240, 320, 480, 560, and 720 minutes from the TEKTITE saturation dive).
10) No Crush-TEK. (which uses the TEKTITE tissue half times and assumes no crushing, i.e., Eq. 18 is replaced by $p_{ss}^* = 5.9$ psi).

For the routine dive shown in Table VB-1, the Yount-GB and Yount-USN schedules require no decompression stops and surface in 3.33 minutes. This is too fast. The Yount-TEKTITE procedure, on the other hand, is remarkably similar to that of Buehlmann in profile and to those of the U.S. Navy, TAMU, and Buehlmann in total ascent time. It is significant that a broad range of time constants is required, even by dives of short duration, if Eqs. 17 and 18 are to be retained. Finally, the No Crush-TEK. decompression is unreasonably long, implying that a broad range of time constants is not sufficient and that crushing should be taken into account.

In Table VB-2, various decompression schedules are compared with the original TEKTITE procedure. The other schedules were calculated for an air dive with nitrogen pressure equal to that of the TEKTITE schedule (air depth of 126 fsw). The TEKTITE procedure was actually used by humans without incident. Also shown are the predictions of the TAMU, Buehlmann, and Hills computer programs, as well as the Yount-GB, Yount-USN, and Yount-TEKTITE calculations just described. The Yount-GB and Yount-USN decompressions are again too fast. The Yount-TEKTITE profile is very similar to that of the original TEKTITE dive, although the total ascent time is about 20% longer. This decreases to about 14% when the use of

Table VB-1. Comparison of various decompression schedules for a 200-fsw depth and 30-min bottom time

Schedule	Time to First Stop	Ascent 180	160	140	130	120	110	100	90	80	70	60	50	40	30	20	10	Total Ascent Time
U.S. NAVY	2.67													2	9	22	37	73.33
TAMU	3.00												2	6	11	25	30	77.00
After Buehlmann	2.20								1	1	3	4	3	5	10	14	36	79.20
Hills	3.00									1	1	2	3	8	65	69	81	233.00
Royal Navy	2.20													5	5	140	2	157.00
Fife	2.40	2	2	2		2		3		4		5		8		10		38.00
Yount-GB	3.33																	3.33
Yount-USN	3.33																	3.33
Yount-TEKTITE	1.83								1	1	2	3	3	3	10	11	39	74.83
No Crush-TEK.	1.00			1	2	2	3	4	4	5	8	11	11	22	27	48	71	220.00

Table VB-2. Comparison of various decompression schedules for project TEKTITE 100-fsw saturation dive (14-day bottom time)

Depth, fsw	TEKTITE	Times, Breathing Air, Min					
		TAMU	After Buehlmann, 1973	Hills	Yount-GB	Yount-USN	Yount-TEKTITE
100-90	10	0.33			0.33	0.33	0.33
90	60						
85	90						
80	100	259.00		41	23.00	39.00	319.00
75	110						
70	120	331.00	209.00	45	26.00	44.00	350.00
65	360						
60	140	355.00	327.00	49	27.00	46.00	376.00
55	160						
50	160	383.00	296.00	55	30.00	50.00	408.00
45	10 Oxy 150 Air						
40	130	416.00	352.00	29	32.00	55.00	445.00
35	20 Oxy 150 Air			28			
30	360	454.00	479.00	29	35.00	61.00	490.00
25	30 Oxy 150 Air			32			
20	150	501.00	420.00	33	40.00	67.00	545.00
15	50 Oxy 120 Air			36			
10	160	559.00	721.00	39	44.00	76.00	614.00
5	60 Oxy 110 Air			42			
TOTAL	2960 (+170 Oxy) (3130)	3258.33	2804.00	492	257.33	438.33	3547.33

oxygen in the TEKTITE profile is taken into account by doubling the oxygen time.

If the time constants used in calculating decompression tables have any physiological significance, then presumably the full range is present whether the dive is of short of long duration. It follows that the same set of time constants should be used universally in any general computational routine. The fact that the Yount-TEKTITE prescription gives sensible predictions for both the short dive of Table VB-1 and the saturation dive of Table VB-2 suggests that this constraint is compatible with, and perhaps even required by, the other assumptions (Eqs. 17 and 18) of the *a priori* procedure.

While the *a priori* procedure apparently requires a broad range of time constants, these need not be associated with tissue half times *per se*. In particular, it may be possible to generate sensible tables by assuming a broad range of restoration time constants and, for example, a single tissue type. Justification for such an approach can be found in the relatively long persistence of the acclimatization observed among compressed air workers (19).

ACKNOWLEDGMENT

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C. ZERO-SUPERSATURATION APPROACH TO DECOMPRESSION OPTIMIZATION: B. A. HILLS

The fairly clear demarcation of the "bounce dive" curve leaves little doubt that it is worthwhile to use mathematics to try to formulate the optimal decompression of a diver following any exposure in excess of the conditions represented by this curve. This led to numerous calculation methods to which partial physiological interpretation has been added later. Although some true mathematical models have since emerged, the original Haldane (3) calculation method still underlies the formulation of most decompression schedules in common use today. There have been countless revisions of the number of tissues, their half times and their M values, but what of the method itself?

Haldane Calculation Method

In the Haldane calculation routine, it is assumed that the rate of elimination of inert gas from any tissue is proportional to the difference between the tension of that gas in the particular tissue (p_t, say) and its tension in blood (p, say). Thus the rate of loss of tissue gas content (C) can be expressed as

$$dC/dt = -k(p_t - p) \qquad (1)$$

where k is the proportionality constant.

This very popular assumption, which dates back to the original Haldane approach (3), is perfectly reasonable if the rate-controlling process is blood perfusion or diffusion across a single membrane (12).

However, what is much more serious in these conventional approaches is the further assumption that the total gas content of the tissue (c) is then related to the tissue tension (p_t) by Henry's law

$$C = Sp_t \qquad (2)$$

where S is the solubility. Substitution for C in Eq. 1, with subsequent integration, gives the simple exponential form for the change in p_t in which the half time of the hypothetical tissue is then given by 0.693 S/k

i.e., $$\Delta p_t = \Delta p_o[1 - \exp-(kt/S)] \qquad (3)$$

for a change (Δp) in blood tension at time t=o.

All designers of diving tables are particularly familiar with this very simple expression, yet its validity depends entirely upon Eq. 2, i.e., whether we can substitute the total gas content for the concentration in applying Henry's law. To be able to do so we must be sure that the whole gas content has remained in true physical solution.

COMPETING GAS PATHWAYS

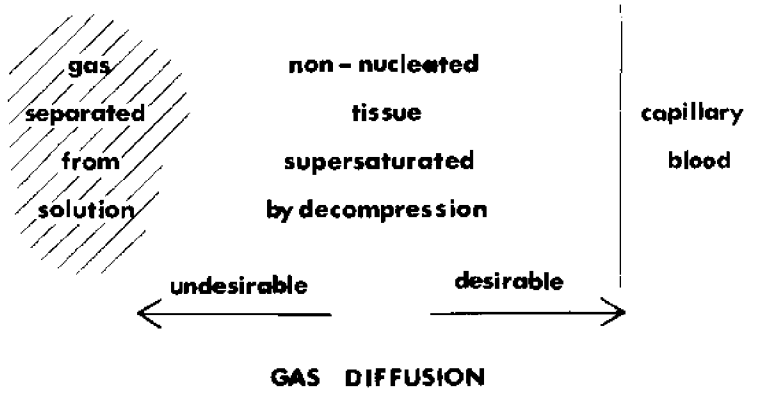

Fig. VC-1. Two basic pathways in which gas can diffuse if left in supersaturated solution by decompression.

NUCLEATION SPECTRUM

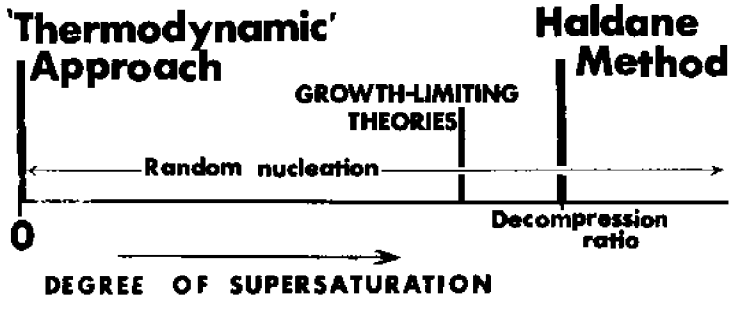

Fig. VC-2. Tissue micro-regions represented by a continuous spectrum of degrees of tolerance to true supersaturation; this illustrates how Haldane (3) and thermodynamic (6) approaches represent opposite ends: best and worst possible cases, respectively.

occurrence, basing decompression upon the few micro-regions, probably out of many million possibilities, where all gas in excess of saturation has been "dumped" into the gas phase. This is the "worst possible" since not only does it represent the maximum amount of gas which can separate from solution, but there is then the minimum driving force for its subsequent elimination via capillary blood.

If some of that gas is present as bubbles, or other forms of gas separated from solution, then not only is Eq. 2 invalid, but also the simple linearized expression for gas transfer (Eq. 3). In the past, the argument has been put forward that there are no bubbles present because the diver does not have bends, but this is just not true. There is now no doubt that "silent" bubbles can be present in the asymptomatic diver. The evidence ranges from direct observation by X-ray (5), Doppler detection (15), and conductivity measurements (10) to numerous indirect methods, all of which show that this also applies to the particular tissue type(s) responsible for decompression sickness (9).

This casts serious doubt upon the use of the exponential function to describe gas elimination. Hence the vast numbers of M values needed in some of the latest modifications (2) to the Haldane method are probably required simply to allow for the inadequacy of the exponential as the basic time function. It must be remembered that each constant is a degree of freedom in the format, so there comes a time when there is little point in calculating at all. With as many as 600 constants, the method becomes so empirical that one might just as well make a direct plot of depth against time.

Gas Separation From Solution

The major objection to the Haldane calculation method is the assumption that all gas has remained in true physical solution in tissue. After a typical decompression beyond the natural or inherent unsaturation of the tissue, we can expect regions of supersaturation (see Fig. VC-1). These can then "dump" their excess gas either into capillary blood or into the gaseous phase in regions where nucleation has occurred. The philosophy behind decompression is, therefore, one of changing environmental parameters in such a way as to emphasize one path at the expense of the other, i.e., to encourage gas in solution in tissue to diffuse towards capillary blood rather than towards gas nuclei.

This leads to an infinite number of possible models, depending upon the distribution of the bubbles or the nuclei as their sites of potential growth. The mathematical complexity of approaches based upon the growth of a single bubble (14) is quite horrendous and even this treatment ignores desaturation of tissue by gas in allowing for elimination to capillary blood.

However, the two extreme cases in the random nucleation spectrum which underlies cavitation permit some form of mathematical description (see Fig. VC-2). The Haldane approach (3) represents the best possible occurrence with no separation of gas from solution until the tissue exceeds the metastable limit to supersaturation described by the M value. On the other hand, the "thermodynamic" approach (6) represents the worst possible

The concept of dumping gas into asymptomatic bubbles was conceived (1) many years before the thermodynamic approach, but was abandoned for an apparent lack of driving force in eliminating gas separated from solution. But let us take a look at what driving force there might be, even after invoking the zero-supersaturation principle.

Zero-Supersaturation Approach

Since oxygen is metabolized, any substitution of oxygen for nitrogen at the alveolar level need not be reflected at the tissue level. Moreover CO_2 produced does not compensate, since it is much more soluble than O_2 and it is total gas tension, not concentration, which determines the imminence of cavitation. This tendency for tissue to remain unsaturated by gases with respect to ambient absolute pressure is enhanced by the shape of the oxyhemoglobin dissociation curve and has been predicted from measurements of total gas tensions in venous blood and the composition changes of subcutaneous gas pockets (4,16).

However, a more direct means of demonstrating an inherent unsaturation in tissue under steady-state conditions is provided by the use of a constant-volume cavity in the form of a noncollapsible plastic tube permeable to all gases and placed subcutaneously in animals (6) (Fig. VC-3). The various gases all equilibrate with the adjacent tissue, so that the absolute pressure within the capsule represents total tissue gas tension 12 hr after any change in ambient pressure or in composition of breathing mix.

It is, therefore, most significant to find that, opening these tubes after 24 hr, they have developed a partial vacuum relative to ambient pressure. This did not change, day after day, even for air breathing at normal atmospheric pressure. Moreover, this partial vacuum relative to ambient pressure increased with change of pressure or breathing mix roughly in accordance with the increase in inspired PO_2 (6).

If we now consider the subject in which this steady-state partial vacuum has been established, and remove the rigid support, the ambient pressure would now be directly applied to that gas. In fact, the absolute pressure of the contained gas would now be compressed by an amount equal to the partial vacuum, thus providing that amount of driving force for its elimination via capillary blood. Since pocket gas tensions of O_2, CO_2 and H_2O rapidly revert to venous values after any pressure change, this inherent unsaturation in tissue is largely imposed as a driving force for eliminating the inert gas (6) (Fig. VC-4).

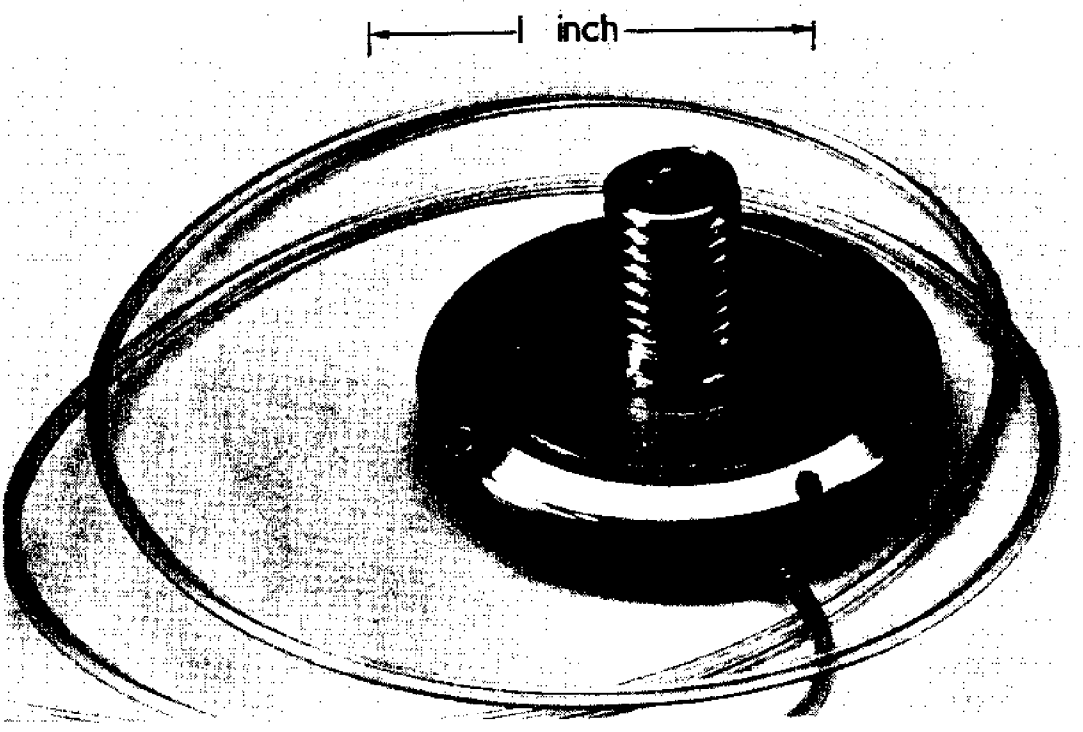

Fig. VC-3. Capsule which was deposited subcutaneously to demonstrate partial vacuum developed and maintained in living tissue under steady-state conditions (6).

Worst Possible Case

Having dispelled the old idea that there can be no driving force for eliminating gas separated from solution, it can now be described analytically (8) as

$$\Delta P_{N_2} = (1 - F_{IN_2})P + c \qquad (4)$$

where P is absolute ambient pressure, F_{IN_2} is the volume fraction of inert gas and c is a small constant relative to normal diving pressures.

This expression is particularly interesting since it predicts that there is a greater driving force for inert gas elimination at greater pressure. Moreover, it implies that a diver should spend more time at greater depth where he will have a greater driving force, i.e., ΔPN_2 is greater for higher values of P in Eq. 4.

This is the exact converse of conventional Haldanian reasoning whereby the U.S. Navy, in particular, give their divers the largest first "pull" towards the surface in the belief that they are then establishing the greatest elimination rate, i.e., reducing p in Eq. 1 to increase (dC/dt). This is fine, provided p_t is not reduced

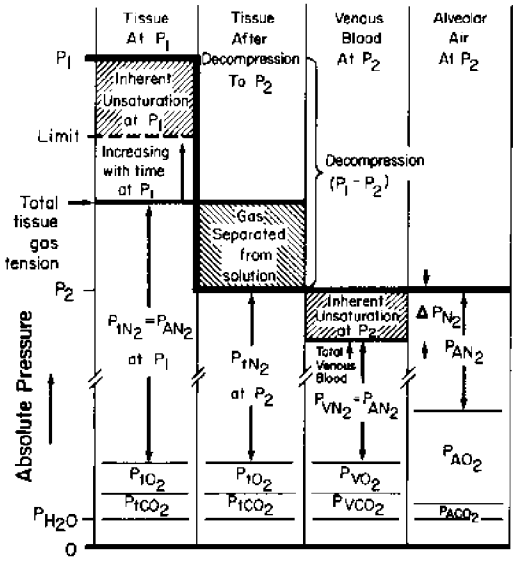

Fig. VC-4. Application of zero-supersaturation principle to a decompression from P_1 to P_2, showing how total tissue gas tension is cut off at P_2 and new inherent unsaturation in venous blood at P_2 becomes driving force for eliminating gas "dumped" from solution.

by cavitation, when a long first pull would be the worst thing they could do.

Vital Experiment

This difference in the effect of pressure on driving force led to a very simple experiment which can clearly differentiate between the two approaches.

If a diver completes a standard U.S.N. table up to the end of the advocated 20-ft stop, when he would be better off moving to the 10-ft stop according to the Haldanian reasoning on which that table was based, i.e., (-dC/dt) is increased in Eq. 1 by reducing p. On the other hand, if the zero-supersaturation principle holds, the diver should be able to complete his decompression in less time by remaining at 20 feet and then 'dropping out' directly to the surface. When tested at the Royal Naval Physiological Laboratory, goats could decompress in less time when surfacing direct from 20 feet than 10 feet, and in even less time when "dropping out" from 30 feet (7). This is compatible with Eq. 4 and the zero-supersaturation concept, but totally incompatible with Eq. 1 and, hence, with the Haldane concept of limited supersaturation.

This result has led to the conclusion (7) that standard U.S.N. schedules do not prevent cavitation, as the calculation method would imply, but could be regarded as treatment tables, controlling the volume of bubbles to below a pain-provoking threshold.

Practical Implications

It does not matter whether the process controlling the rate of uptake of inert gas by tissue is blood perfusion (Fig. VC-4) or diffusion (Fig. VC-5); the zero-supersaturation principle cuts off the total gas tension at the absolute pressure. This concept has provided a better prediction (6) of bends cases in practical trials of Haldane-type decompressions than the supersaturation concept on which they were designed.

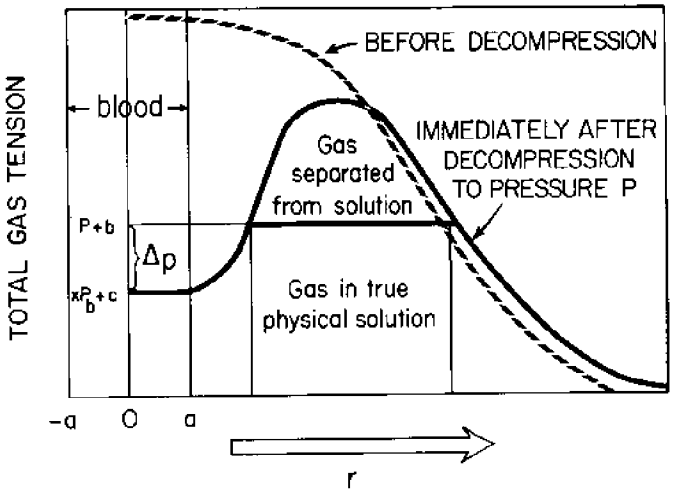

Fig. VC-5. Application of zero-supersaturation principle to diffusion of gas into tissue from a capillary. This shows how total gas tension is cut off at a pressure just in excess of ambient absolute pressure (P), b being a small correction for surface tension and tissue compliance.

Decompression Optimization

It is easy to criticize the Haldane calculation method, or even to show that the thermodynamic approach is more appropriate, but how may we invoke the zero-supersaturation principle describing the "worst possible" case to optimize decompression?

Taking the radial diffusion model as an example (Fig. VC-5), this leads to a format consisting of three basic steps:

1) Rapid decompression to the point of true saturation of tissue by gases, i.e., just taking up the unsaturation due to: (a) the inherent unsaturation and (b) any failure of inert gas to equilibrate between tissue and alveolar air.

2) Gradual decompression in which depth is adjusted such that the peak total gas tension just fails to exceed it (Fig. VC-6).

3) Direct surfacing from about 25 feet, precipitating gas, but to just below the pain-provoking dimensions.

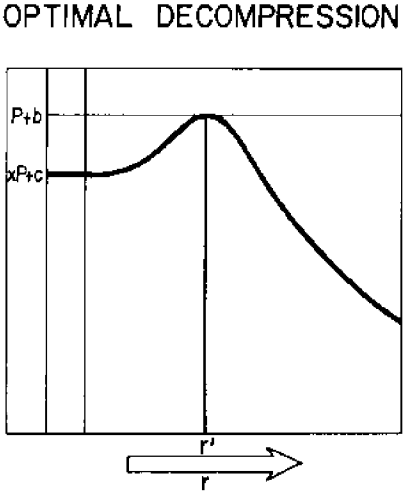

Fig. VC-6. Demonstrating computation principle underlying decompression optimization based upon zero-supersaturation concept.

This model results in the type of format shown in Fig. VC-7. The feature (6,7) is that decompression is more efficient by keeping the diver longer at the deeper stops and introducing further stops deeper than advocated by U.S.N. tables. This is exactly compatible with the experience of Okinawan pearl divers operating in Australian coastal waters (6). With no detectable medical or scientific input, these people have devised purely empirical diving schedules due to the economic incentive to minimize decompression time. Moreover they regularly take 30-50% less time to decompress than the U.S.N. tables would suggest.

Computation

The steps for optimizing according to the zero-supersaturation principle have been programmed for the computer, which can then provide a format for any combination of depth and exposure time, e.g., that shown in Fig. VC-7. It is felt that the merits of the approach are:

1) a fundamental physiological basis consistent with the laws of physics,
2) no need to invoke more than one tissue type,
3) only two constants are needed, and hence there are no more than two degrees of freedom.

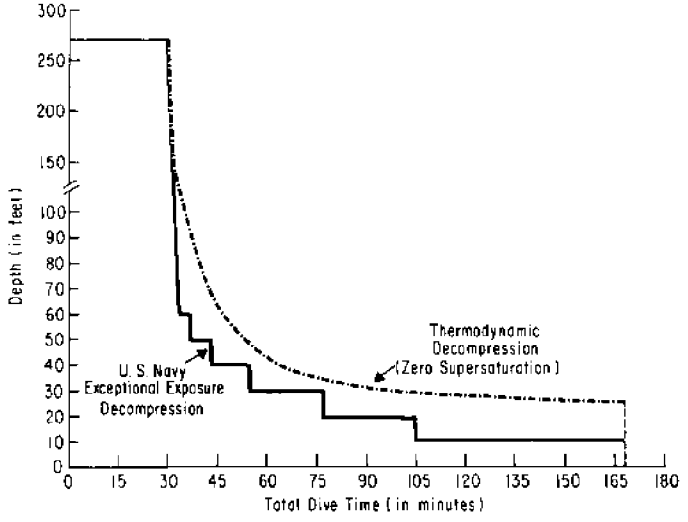

Fig. VC-7. Typical decompression profile derived by application of zero-supersaturation principle to radial diffusion model shown in Fig. VC-6.

The last point means that any data offers a much more exacting test of the model compared with current calculation methods which offer as many as 600 constants to fix, and hence require 600 degrees of freedom to make the Haldane model fit the data.

Oxygen Optimization

The ideal model predicts not only the optimal depth at any time during a decompression but also the optimal composition of the breathing mix at that point. A continuous change of breathing mix would not be practical, but a schedule of ideal times at which to switch between four gas mixes is certainly feasible. However, this then requires another constraint by which to dispense O_2 and hence an index based upon the imminence of oxygen toxicity.

One set of units for quantifying this further constraint is provided by summing the pulmonary oxygen toxicity units (POTU's) as defined by Lambertsen (13). However, these do not allow for regression of the toxic effects of O_2 upon switching to air breathing. Hence another index has been derived, the cumulative oxygen toxicity index (COTi) (11) which can regress upon switching to a less toxic breathing mix

$$COTi = \Sigma(\Delta PO_2/\Pi O_2)_n \qquad (5)$$

This sums the changes in PIO_2 as fractions of the inspired oxygen partial pressure (ΠO_2) needed to induce marginal toxicity over the same time span, decreases contributing negative terms. Thus the

subject is more likely to show toxic manifestations than the dose-time curve used as a reference if the COTi exceeds unity.

This index is simple to program on the computer and, moreover, it is relatively easy to integrate this program with the one for optimizing decompression. Thus there is mutual interaction between the programs with simultaneous optimization of both depth and inspired gas composition according to the two constraints, the imminence of oxygen toxicity and adherence to the principle of zero-supersaturation.

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D. PATHOPHYSIOLOGY OF DECOMPRESSION SICKNESS: KENT H. SMITH, PHYLLIS J. STEGALL, AND BRIAN G. D'AOUST

Dorland's Medical Dictionary (23rd ed.) equates caisson disease, compressed air illness and decompression sickness. It further defines decompression sickness as a "disorder characterized by joint pains, respiratory manifestations, skin lesions and neurologic signs occurring in aviators, divers, and compressed air workers following rapid reduction of air pressure." In light of present information, this definition appears to be limiting, and rather than introducing another category of "silent decompression sickness," I should like to generalize the definition as:

> Any group of signs and/or symptoms which result from a reduction in ambient pressure.

Many occupations require man to work under environmental pressures other than atmospheric, each change of pressure bringing problems of physiological and biochemical adjustment, some relatively inconsequential, some potentially serious. If the reduction of pressure is not carried out in a controlled manner, one or more of the wide range of symptoms known as decompression sickness may be produced. Although the fundamental physics and physiology of decompression have generally been understood, there still is insufficient knowledge and understanding to indicate the best practical approach to be used to formulate safe decompression procedures and uncertainty as to how best to effect treatment. Few decompression profiles now in use can be considered absolutely safe despite many years of attempts to solve the perplexing decompression problem. Until the exact cause and the pathophysiologic course of decompression sickness is well defined, the solution to the problem will continue to be as elusive as it has been for the past one hundred years. Recent evidence indicates that gas emboli are present in divers using "acceptable" decompression schedules and that such separated gas has a variety of hematological, vascular, and tissue effects, any of which may not be sufficient to produce symptoms.

Pain in one or more large joints has been the most common clinical sign of decompression sickness. Pol and Watelle noted in 1854 (37) that these limb pains were cured by returning the suffering person to the raised pressure at which he had been working. It was suggested then that the pain was due to bubbles formed, when the pressure was reduced, from the gases which had dissolved in the tissue fluids during exposure to raised pressure. To date, this hypothesis has not been invalidated.

The failure of currently accepted decompression schedules to prevent symptoms of decompression sickness has been accounted for by Kidd and Elliott (22) as a result of 1) an error in decompression theory, 2) an error in decompression practice, and/or 3) an anomalous response of the individual.

The magnitude of the exposure to pressure is not a reliable guide for assessing the severity of a case of decompression sickness (24), and cases may well arise from what may appear to be innocuous exposures well within accepted minimum decompression limits (1).

Whatever the cause of an inappropriate decompression, minute bubbles of inert gas are thought to form, coalesce and grow, exert pressure on nerve elements, interact with the hemostatic mechanism, damage vascular endothelia and/or occlude some part of the circulation. The presenting symptoms may be sudden and dramatic, involving vital centers, or delayed, localized, and merely uncomfortable. Secondary effects of tissue damage and edema may be superimposed on this picture.

Gas Uptake and Elimination

Since Haldane first proposed a systematic method for calculation of decompression tables, it has been assumed that the body saturated and desaturated symmetrically, that is, both processes could be described by the same mathematical function. This was challenged by Hempleman (15), on the basis of a number of repetitive dive experiments with goats, which indirectly suggested the presence of "silent bubbles," a factor long suspected to require consideration. Theoretical predictions indicated that non-symmetry in uptake and elimination would be expected if bubbles were present during a decompression, because gas equilibration depends on the presence of a gradient in pressure, which bubble formation would eliminate. Le Messurier and Hills (25) further stressed this idea and cited both physical experiments and the unusual diving practices of native divers in the Torres Straits as evidence. However, until 1967 no experiment sufficiently direct to be conclusive had been undertaken on surviving animals or men either to demonstrate that gases were eliminated differently during acceptable decompression, or that bubbles were present in tissues and/or blood. Work in this laboratory from 1967 to 1974 has provided significant new perspectives on both of these questions. Studies by Spencer and Campbell (44) demonstrated an effect of certain bronchodilatory drugs in lessening the incidence of decompression sickness in guinea pigs. Although the decompressions were extreme, they demonstrated the importance of physiological and pharmacological variables in the pathogenesis of decompression sickness.

Over the past three years our studies have provided estimates of gas uptake and elimination rates in awake dogs, by direct analysis of mixed venous blood inert gas content after compression and decompression. It has been found (Fig. VD-1) that a decompression from saturation at only 33 feet of seawater (fsw) to the surface, previously supposed to be without detectable effects, produces a decrease in the apparent rate at which nitrogen is cleared from the

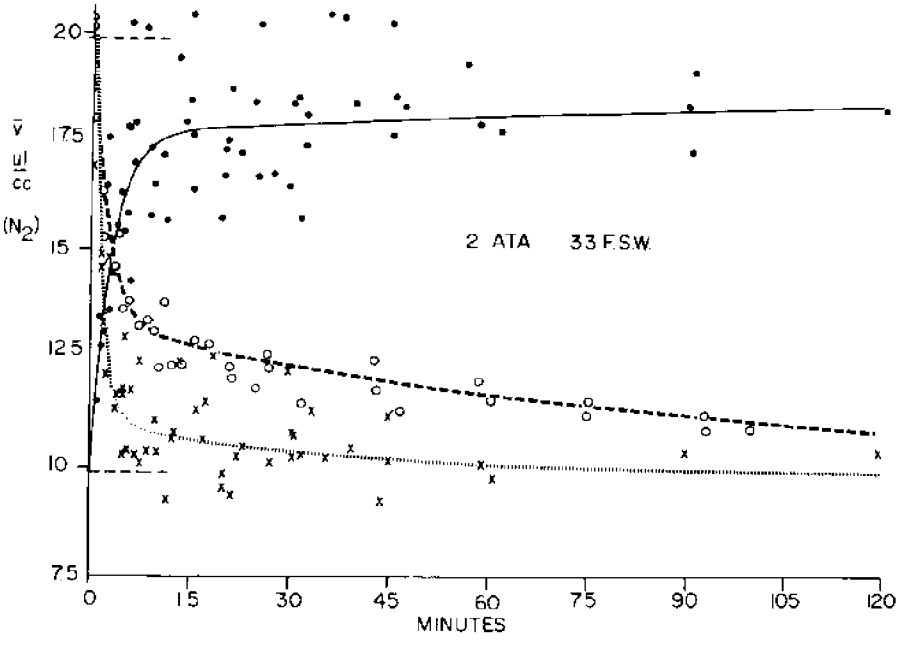


Fig. VD-1. Mixed venous blood N_2 content $\bar{V}N_2$ µl/ml in awake dogs plotted against time a: following pressurization to 33 fsw (•); b: following decompression to surface from saturation at 33 fsw (x); and c: following gas switching to 20% O_2-80% He in tracheostomized dog at atmospheric pressure (o). All lines are least squares fit of respective data to equation $Y = B_1e^{-k1t} + B_2e^{-k2t}$. Note functional asymmetry imposed on N_2 elimination by decompression. Same results obtain at 66 and 100 fsw. Both pressurization and decompression were rapid with respect to time scale (< 2 min).

tissues to the blood. This is illustrated in Fig. VD-2, where observed and expected rates are compared.

The result is of major significance to the formulation of decompression procedures, as it indicates that the decompression itself is decreasing the rate of gas elimination. While these studies were proceeding, other investigations attempting to actually demonstrate the presence of bubbles were underway in this laboratory.

Critical Conditions for Phase Separation

It is not surprising that fundamental studies on bubble formation have provided little guidance in the design of decompression procedures. Since Harvey's thorough studies (10), it has been widely accepted that fluids can withstand tremendous supersaturations without bubble formation, and yet in other cases some fluids

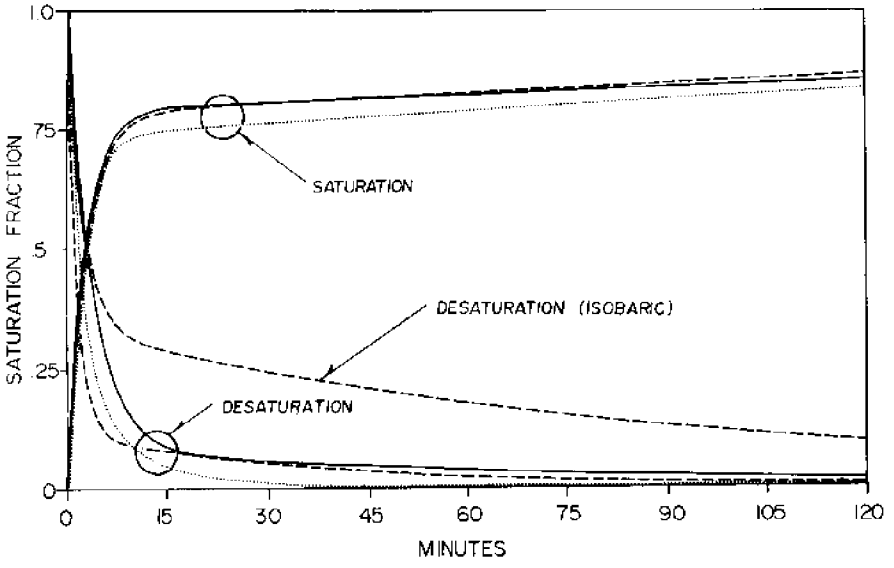


Fig. VD-2. Fractional state of N_2 saturation of mixed venous blood ($\bar{V}$) (sampled from pulmonary artery of awake dogs) vs. time following compression to and decompression from 33 (---), 66 (•••) and 99 (---) fsw. Curves are based on least squares fit of data to equation

$$Y\ (= N_2 \text{ content}) = B_1 e^{-k_1 t} + B_2 e^{-k_2 t}$$

Similarity of all uptake (increasing) curves and all desaturation (decreasing) curves despite different pressures lends support to the data. Dissimilarity of saturation and desaturation curves as a group suggests effects of decompression on desaturation. Mixed venous blood appears to desaturate much more rapidly than it saturates; however, calculation of quantity of N_2 which could be removed by venous blood in any given time indicates that much N_2 remains in the animal. Similarity of form of isobaric desaturation curve to saturation curves indicates that decompression per se induces alteration in elimination rate. In this case absolute pressure was 33 fsw (1 atmosphere) and desaturation was accomplished by suddenly switching breathing gas to 20% O_2/80% He in a tracheostomized awake dog. Only isobaric desaturation shows theoretically predicted symmetry with saturation.

can exhibit bubble formation with supersaturations of only a few percent. This suggests a difficulty in determining critical conditions for phase separation (3). Recent work by Hemmingsen (14) and D'Aoust and Smith (7) indicates the importance of solubility as well as supersaturation as a determinant of bubble formation.

Supersaturations which are one-tenth of those allowed in a 20-min half-time tissue in man have been shown to produce bubbles in vivo. Idicula (19) demonstrated bubbles in vivo in pigs produced by means of counter-diffusing an insoluble gas against a highly soluble gas with which the animal was saturated. These bubbles were present with supersaturations as low as 30% of one atmosphere. Ikels (20) observed similar evidence using several in vitro systems.

It has also been known that young salmon supersaturated with air by dams spilling excess water or rapid temperature increases in rivers are eventually killed by supersaturations as low as 20% (7). This effect takes several hours to become apparent in experimental situations--a time which is much greater than the saturation time of the fish. This suggests that not only the degree of supersaturation but also the time over which a tissue is exposed to the supersaturation is an important factor.

Detection of Separated Gas

Since decompression sickness had long been identified with the unphysiologic evolution of gas from body stores in the form of bubbles, techniques for the ultrasonic detection of gas bubbles in tissue and blood were attempted that would allow their acoustic visualization (Fig. VD-3). That bubbles were present in a diver was not suspected until he presented signs of decompression sickness prior to ultrasonic visualization of the bubbles. In the original Doppler studies, Spencer et al. (44) implanted Doppler flow transducers on the posterior vena cava and descending aorta of domestic sheep. They demonstrated that Doppler bubble signals in the posterior vena cava preceded signs of decompression sickness. Other studies using this technique supported the satisfactory use of the Doppler in the detection of bubbles (11,28). The studies in our laboratory (41) demonstrated that the longevity and sometimes the character of the bubble signal were not entirely consistent with the theory of bubbles in the vascular system. Assuming normal conditions of diffusion were present (17), one would not expect a bubble to remain in the circulatory system for the extended periods of time during which the characteristic sounds were heard. Thus, it was suspected that coagulation particles in the cardiovascular system were being detected by the Doppler system. We have subsequently demonstrated in this laboratory that bubbles were present in many successful decompressions, but that these bubbles had an effect on the hemostatic function and possibly on the integrity of the vasculature.

Effects of Inadequate Decompression

Inadequate decompression may be either symptomatic or asymptomatic, but even the latter can be associated with a severe pathogenesis resulting in acute or latent sequelae. These may range

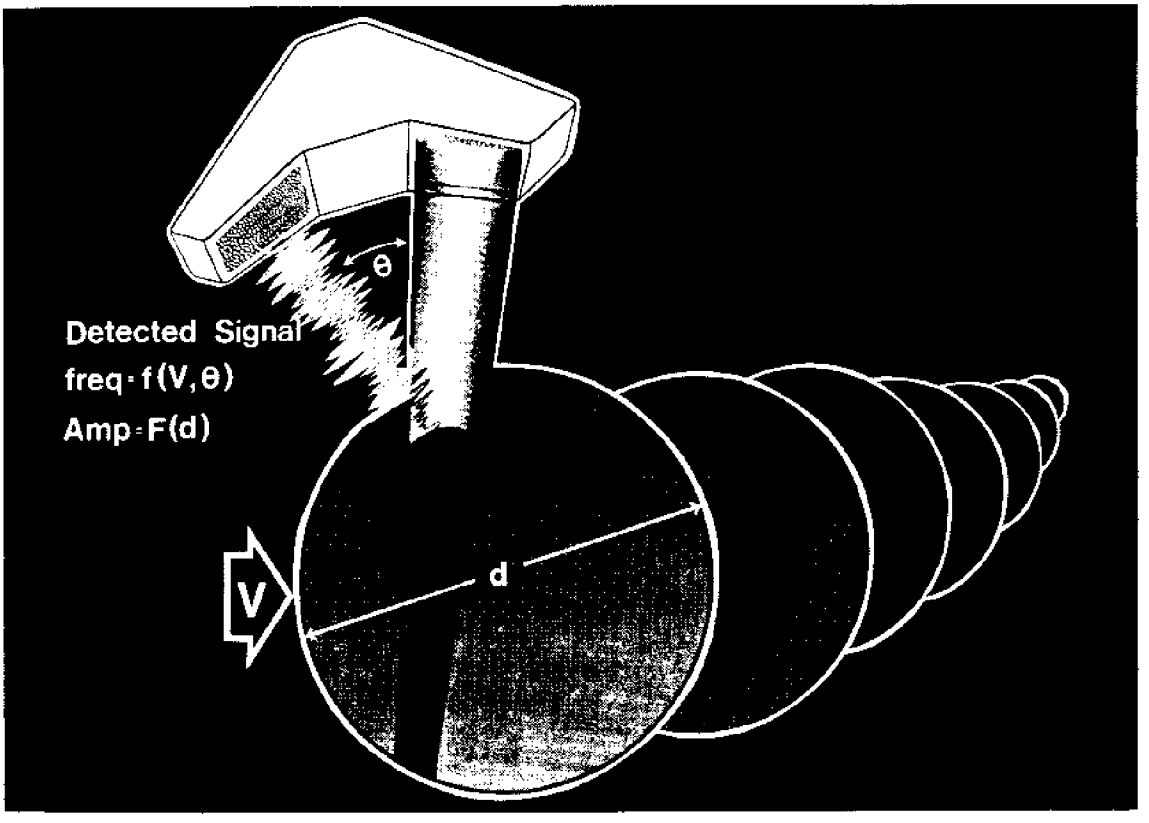


Fig. VD-3. Schematic illustration of ultrasonic Doppler bubble detection of gas emboli in pulmonary artery or other vessel. Coherent ultrasonic energy at 5 mH is shown beamed from 1 piezoelectric barium titanate crystal into vessel through body wall. Detection of reflected signal of 5 mH ± Doppler shift of 500 - 2000 Hz is accomplished by the other equivalent crystal mounted at an appropriate angle. Appropriate electronic circuits detect and display Doppler shift frequency, a function chiefly of incident angle and velocity of reflecting element; amplitude is a function chiefly of "d" and other geometric factors. When a bubble or particle of different acoustic impedence passes through insonified regions, a characteristic frequency/amplitude combination or audible "chirp" which depends on particle size, velocity and position in the sonic field appears in reflected signal. These events can be detected electronically and counted by imposing certain types of signal processing. Result is a valuable tool for evaluating safety of new and existing decompression procedures.

from a simple transient consumptive coagulopathy, which is not clinically manifested, to complete auditory loss, vestibular disorientation, paraplegia, or aseptic bone necrosis.

Blood-Bubble Interaction

Agglutination of red cells in decompression sickness was considered in 1937 by Swindle (47) and End (8). Other investigators have more recently demonstrated that in severe decompression sickness with vascular bubble formation, microthrombi involving platelet aggregates occurred in a manner similar to that seen in disseminated intravascular coagulation (36). Platelet and red cell aggregates were seen in the pulmonary vasculature of rats which had decompression sickness. Similar thrombi were seen in the pulmonary vessels of rabbits which had been infused intravenously with air. Rats with artificially elevated platelet counts were shown to have an increased incidence of bends, and serotonin injected intraperitoneally into rats before decompression led to a mortality of nearly 100%.

Interaction of platelets with surfaces was thought to be an important stimulus in the initiation of thrombosis and consequently was of considerable concern when intravascular bubbles were present (31,34). Contact of platelets to surfaces has been shown to cause release of their constituents which includes serotonin and nucleotides such as adenosine diphosphate (ADP) (18,34,43). Released ADP causes platelet aggregation and is thus important in the development of a platelet mass (2,18). By these mechanisms, the stimulus provided by contact with an appropriate surface is able to produce a platelet mass without the precipitation of blood coagulation (9). This platelet mass may serve as a focus for the initiation or acceleration of the coagulation mechanism by leading to the formation of thrombin (32). Intra-arterial air infusion (36) caused platelets to form soft friable masses of aggregates at the air-blood interfaces. These aggregates did not form compact masses but instead fragmented easily when they were washed away in a flowing blood stream.

In studies by Philp et al. (34), serotonin, adenosine, ADP, and air infusions all produced hypotension. In pulmonary arterial embolism, active vasoconstriction and mechanical blockages of the vasculature were said to cause rise of the pulmonary arterial pressure (6,32). The vasoconstrictive substance, serotonin, can be expected to play an important role in hemodynamics subsequent to pulmonary vascular aero- or thrombo-embolism (27). Circulating gas bubbles from decompression sickness are surely destined to end up in some filtering organ such as the lung or liver, and thus cause local and systemic hemodynamic effects.

Although nucleation and bubble formation can occur in the arterial blood, it has been hypothesized by Lever (25) and shown

by Spencer and Oyama (45) that gas bubbles pass through the pulmonary circulation to the arterial system. This can occur if the bubbles are small enough to pass through intrapulmonary arteriovenous shunts which have a larger diameter than normal pulmonary capillary vessels (33).

From hematologic studies done in this laboratory (42,40), it is apparent that hemostatic abnormalities do occur following asymptomatic hyperbaric exposure. These abnormalities have been monitored by following kinetic measurements of the survival of hemostatic factors. Concentrations or platelet quantitative counts reflect changes in storage pools and production rates as well, and are therefore not reliable indicators of hemostatic consumption. The finding of both increased platelets and fibrinogen utilization in the immediate post-dive period suggested that a condition of intravascular consumption was produced by diving (Table VD-1).

Table VD-1. Platelet and fibrinogen survival in swine during first post-dive week

	Observations	Platelet Survival, days	Fibrinogen Survival, days
Controls	10	5.02 ± 0.13	4.12 ± 0.09
Baselines	5	2.78 ± 0.41	3.21 ± 0.50
Anticoagulation	2	3.58 ± 0.02	2.64 ± 0.05
Platelet Inhibitors	4	4.80 ± 0.29	3.63 ± 0.31

Both platelet and fibrinogen survivals were significantly shortened following single asymptomatic exposure. Anticoagulants alone failed to prevent increased utilization of these factors; combined anticoagulant-platelet function inhibitors therapy did reverse consumption.

However, the fact that the consumption could not be prevented by anticoagulation alone, as has been shown to be appropriate therapy for other states of intravascular consumption, and that prevention required combined therapy with heparin and platelet function inhibitors, aspirin and persantin (50,51), indicates that more than a single mechanism was operating to produce the increased platelet and fibrinogen consumption. This was supported by the findings that fibrinogen survivals returned to normal by the end of the first compression/decompression exposure week (Fig. VD-4), and that the continued abnormality involved only platelet consumption (Fig. VD-5).

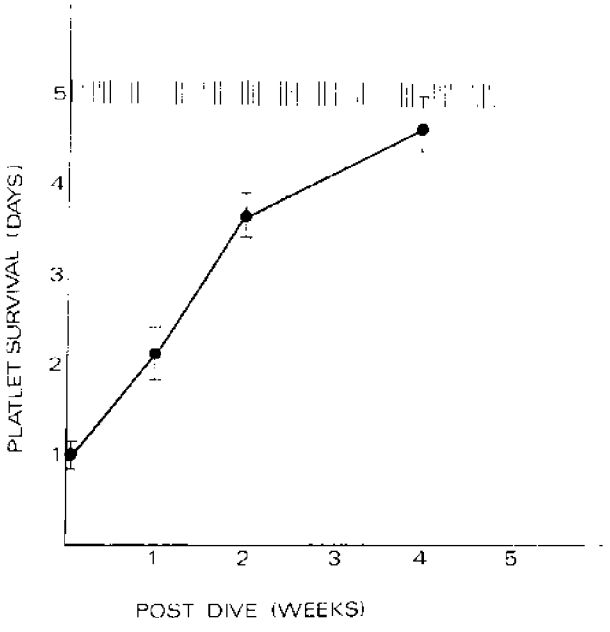
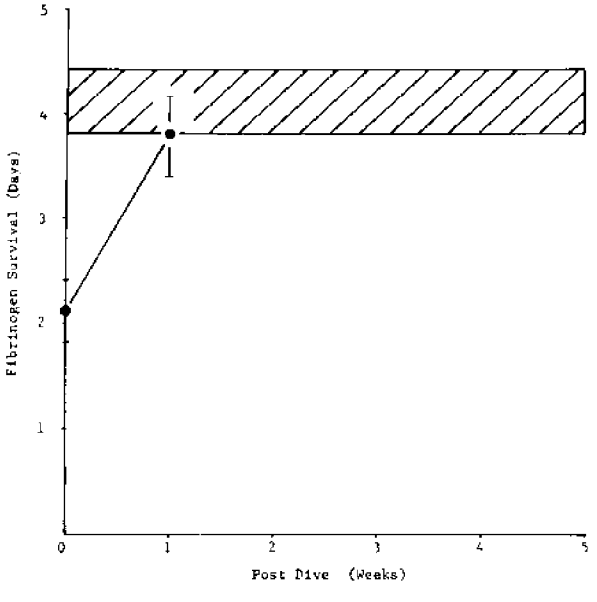


Fig. VD-4. Swine fibrinogen survival rates returned to normal at end of 1st compression/decompression wk.

Fig. VD-5. Post-dive platelet consumption persisting for 4 wk, thought to be result of platelet response to decompression-induced vessel injury.



Since earlier in the study there was some histologic evidence of endothelial damage, a condition which has been reported by others (48), the persisting shortened platelet survival was thought to be possibly the result of the platelet response to vessel damage, with increased adhesion and aggregation to the site of vascular injury. The prolonged time necessary for platelet survivals to return to normal appeared to reflect the time required for healing of the damaged vascular endothelium. The increased platelet utilization which occurred in the immediate post-dive period suggested that there might have been sufficient tissue injury to release tissue thromboplastin into the circulation so that a minor component of intravascular consumption was produced. Alternatively, changes in the microcirculation, perhaps due to blood-bubble-induced flow changes, might have been enough to produce stasis in fibrin formation in areas of low flow. Subsequent studies in our laboratory have demonstrated endothelial damage as the result of inadequate decompression.

Lung and Blood-Bubble Interactions

The presence or absence of bubbles is clearly an important factor which must be considered as potentially modulating, physically and physiologically, the rate of gas elimination both from the tissues (16) and the lungs (45). There is also the possibility that separated gas in tissues can produce local reactions, not only changing local perfusions but also mobilizing the systemic responses in the area. If this happened in the lung, changes in $\dot{V}A/Q$ distribution could be expected. It has been shown by Chryssanthou (4) that the lungs of animals with decompression sickness can be the source of a smooth muscle activating factor (SMAF) which can have deleterious effects when administered to other animals. In this connection it is important to note that the lung has been shown to have the ability residing at or near the endothelium of the pulmonary vasculature, both to convert angiotensin I to angiotensin II and to inactivate bradykinin. This suggests that the lung plays a role as a physiological systemic regulator (13,39). Thus it is not difficult to imagine that the concurrence of pulmonary gas embolism, either from decompression sickness or aero-embolism, could cause the lung to mediate cardiovascular and ventilatory changes quite independently of the control systems mediating its role as a gas exchange organ.

Bubbles, Blood, and Vestibular-Auditory Dysfunction

Diving-induced deafness and vestibular dysfunction have recently been recognized as important problems in individuals exposed to compressed gas atmospheres (21,12,29,38). The condition encompasses almost every phase of human diving, from breath-hold and shallow air SCUBA dives to deep saturation helium/oxygen dives. Hemostatic and thrombotic problems have been proposed as potential

etiologic factors which, according to McCormick, could largely be prevented by the prophylactic administration of heparin (30).

It is interesting with respect to the aseptic bone necrosis sequela described below that hemorrhage in the perilymph has been correlated with the incidence of a vestibular attack and subsequent cochlear-auditory dysfunction in dived animals (30). It was noted that animals subjected to the same helium-oxygen dive conditions, but not having attacks, did not show hemorrhage in the inner ear.

Aseptic Bone Necrosis

Aseptic bone necrosis, a latent sequela of inadequate decompression manifested by localized destruction of selected long bones in the body, has been studied extensively in our laboratory (40,42,46). Coagulation function studies, done on animals in which aseptic bone necrosis was produced by compression/decompression exposures, have demonstrated a significant reduction in platelet and fibrinogen survival times. Platelet function inhibitors and anticoagulants were used to prevent the hemostatic changes; however, those animals in which anticoagulants and platelet function inhibitors were used succumbed to the aseptic necrosis in a shorter period of time with fewer compression/decompression episodes. One hypothesis possibly explaining this event includes a hemorrhagic infarct at the site of the lesion as the reuslt of endothelial damage. Fig. VD-6 demonstrates hemorrhage present in the bone

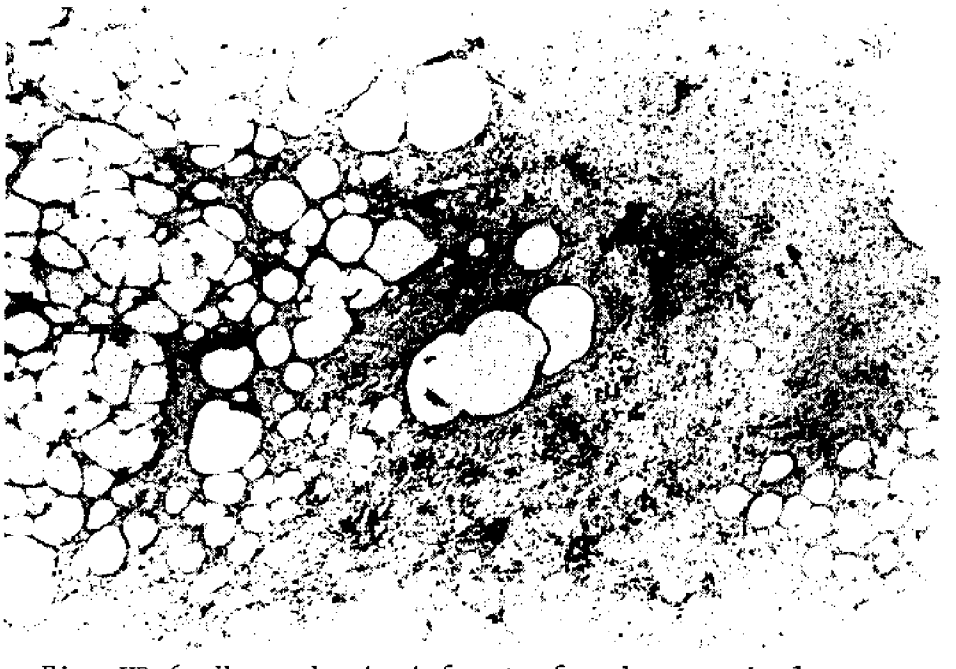


Fig. VD-6. Hemorrhagic infarcts found extensively in bones of swine exposed to inadequate decompression.

following inadequate decompression. Aseptic bone necrosis is also a sequela to certain bleeding disorders in human medicine unrelated to diving.

The most common pain associated with decompression sickness is deep bone pain, usually located at a juxta-articular position. Rheologic impairment has been demonstrated (8,49) and the pain associated with decompression sickness has been successfully treated by using the infusion of low molecular weight dextrans and heparin (5,23). These agents, by their platelet-function inhibiting action, anticoagulation function and lipemic clearing action, can establish a more normal rheology in the affected area and thereby reduce the pain. This is not dissimilar from the pain experienced by sickle cell crisis patients for whom similar therapeutic modalities are used.

Techniques for determining decompression procedures are producing a higher than satisfactory morbidity of decompression sickness and bubbles in otherwise asymptomatic dives. Since the formation of the bubble is clearly so instrumental in the etiology and pathogenesis of decompression sickness, it is essential that we confirm the blood-bubble-tissue interactions. The risks and hazards of diving will remain until such time as certain fundamental concepts of inert gas uptake and elimination, bubble formation, and the pathophysiological consequences of inadequate decompression are demonstrated, defined and understood.

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E. HUMAN ASEPTIC BONE NECROSIS: D. N. WALDER

Although bone necrosis was first described in compressed air workers in 1911 (1) and in divers in 1941 (4) it is only in recent years that much attention has been paid to this insidious danger of hyperbaric exposure.

As so many causes have been suggested for aseptic bone necrosis in man, there is a danger of assuming that it is a non-specific reaction of bone to some underlying common fault. To do so at present would be a mistake, because our knowledge of aseptic necrosis of human bones depends so heavily on radiological evidence, which can be misleading.

There can be no doubt that one form of aseptic bone necrosis may follow exposure to a hyperbaric environment (as in compressed air work and diving). Curiously, unlike decompression sickness, the condition does not seem to be associated with hypobaric exposure (as in high-altitude flight). The prevalence of bone necrosis in divers appears to be related to the care with which they decompress, since the prevalence in well-disciplined groups such as the British Navy is only a few per cent (5), though amongst Japanese diving fishermen, for instance, it can approach 60% (7). Our experience with 1,216 North Sea divers suggests a gross prevalence of about 2%, of which approximately one-half are potentially disabling juxta-articular lesions.

Although so far we know of no divers working in the North Sea who have developed symptoms from their lesions, it would be prudent not to be too optimistic. Current decompression procedures may be reasonably satisfactory in avoiding serious bone damage following "bounce" diving, but with the fundamental change which is occurring in the pattern of commercial diving in the North Sea and the increase in saturation diving it is impossible to forecast what the incidence in the future might be. Our experience with British compressed air workers shows that there is a marked difference between the prevalence of bone necrosis in those men whose exposures have been limited to less than four hours, as compared to those exposed for more than four hours. In other words, as the man approaches saturation the risk of bone damage becomes greater. It is also relevant to point out that in the U.S., where great efforts have been made in recent years to prevent the occurrence of bone necrosis in compressed air workers, not only were decompression times lengthened but exposure times have also been shortened. For instance, the Washington state tables limit the work period to four hours or less when the pressure exceeds 30 m equivalent water depth. Because the widespread use of saturation represents a comparatively recent change in the nature of diving, it is essential to continue to study long bone changes in divers at least over the next decade.

It has been suggested that bone necrosis is in some way linked to the presence of nitrogen in the breathing gas. If this were so, using helium and avoiding nitrogen should reduce the prevalence of bone necrosis, or even eliminate it. But, of course, to avoid nitrogen altogether would have a profound influence on some of the diving techniques currently being developed. However, the only objective evidence available so far does not support this idea. In a recent analysis of North Sea divers, a smaller proportion of those who had dived to less than 50 m (air diving) had bone necrosis compared to those who had dived to greater depths (helium-mixture diving) as shown in Table VE-1.

Table VE-1. Bone necrosis in commercial divers, depth and bone lesions (April 1975)

Depth, m	No Bone Lesion	Bone Lesion	No. of Cases
<50	361	1	362
50 or more	823	25	848
Total	1184	26	1210

χ^2 with Yates' correction = 7.39;
$P < 0.01$

A question which often arises is whether a diver who develops a bone lesion should be advised to stop commercial diving. In the British Navy (6) it is recommended that any diver who has "radiological aseptic bone necrosis A (meaning juxta-articular lesions) or B (meaning shaft lesions)", whether definite ('positive') or uncertain ('doubtful') should, as a prophylactic measure against further damage, be restricted to relatively safe diving. In practice, such a person is excluded from trial and experimental diving, and limited to dives using the diving tables of BR 2806 (the Diving Manual) above the limiting line, with a maximum depth certainly not greater than 75 m (250 ft). "Any person with an 'A' lesion should be limited to oxygen diving breathing apparatus Pattern 5562 until he can be assessed by SMO Underwater Medicine, who will arrange clinical referral where necessary." What is the position with commercial divers? Disregarding any legal considerations, the medical facts seem to be as follows.

Shaft lesions in themselves are not important, in so far as experience indicates that they never lead to pain or disability. It must not be forgotten however, that in a very few rare cases a

shaft lesion has been claimed to be the seat of neoplastic change (3, 8). There is also a greater chance of a man with a shaft lesion developing a juxta-articular lesion than a man without a shaft lesion. The data on which this opinion is based are admittedly thin, but they are the best we have. Of 38 compressed air workers with shaft lesions who continued their hyperbaric exposures, 8 (21%) had developed signs of juxta-articular lesions on subsequent radiographic examinations. At the moment the man should be told the facts but allowed to continue diving.

The presence of a juxta-articular lesion is important in itself, and the man should be advised to give up diving. Some of these lesions will undoubtedly progress and lead to collapse of the joint surface with pain, disability, and secondary arthritis. Unfortunately, it is not possible to predict from the radiographic appearances which lesions will progress and which will remain stable, so we are working on this aspect of bone necrosis at present. It seems possible, for instance, that those juxta-articular lesions which on radiological appearance are known as dense areas (A1) are less likely to break down than those which are described as linear opacities (A3). It is hoped that eventually, either through more detailed studies and more experience using radiographic techniques, such as tomography, or by the application of bone scanning using bone-seeking radioisotopes, it may be possible to determine when a lesion can be said to have undergone complete repair and is therefore of no further potential danger.

To avoid further misunderstandings it should be mentioned that "bone islands" are not the result of diving and certainly do not give rise to symptoms even if they occasionally change size. In a survey of Registry material carried out by Griffiths (personal communication) the prevalence of bone islands in two groups of men was compared. One group consisted of 100 commercial divers (ages 23-40) with at least 4 years of experience who had dived to depths greater than 50 m; the other group consisted of 100 men within the same age range who were declared medically fit for compressed air, but had never before been exposed to pressure. No statistically significant difference was found between the numbers of bone islands, compared with the control group.

Any man who has been stopped from further diving will naturally want to know whether that will prevent this juxta-articular bone lesion from getting worse. The answer can only be, "No." Perhaps a prolonged period of no load-bearing by the affected joint might be beneficial, though in view of the long duration necessary for this treatment and the uncertainty of its success, it does not seem reasonable to suggest such a line of management. What then is the point of ceasing to dive? The reason is simple. To have one major joint disabled is quite bad enough; if another joint were to be affected, the man would be faced with the probability of two major joints in trouble and that is a severe disability. As has

already been shown, men with shaft lesions have a high risk of getting further lesions. When the figures for compressed air workers with juxta-articular lesions are considered, those who continue to undergo hyperbaric exposures stand an even higher chance (39%) of more lesions developing, while stopping this type of work, as might be expected, practically stops the occurrence of more lesions.

The fact that so many compressed air workers who have a bone lesion and continue to expose themselves to pressure get another lesion suggests that perhaps some men are more susceptible to bone necrosis than others. This suggestion is also supported by the fact that several of the compressed air workers with bone lesions are known to have developed them after only one or two exposures to modest pressures. In general terms the risk of developing a bone lesion increases the more times men expose themselves to pressure (Table VE-2).

Table VE-2. Bone necrosis in compressed air workers (Clyde Tunnel)

Exposures	<600	>600	
Men with bone lesions	22	41	63
Men without bone lesions	90	70	160
	112	111	223

Exposures were at 18-34 psig;
$\chi^2 = 8.22$; $\underline{P} < 0.005$

Much of the epidemiological data about aseptic necrosis of bone due to hyperbaric exposure comes from our experience with compressed air workers because the numbers, both in terms of men and number of exposures, is much greater than with divers. Experience so far suggests that the nature of the bone lesion is the same in the two groups but that there is at least one interesting difference, namely the distribution of the lesions. Juxta-articular lesions of the femoral head are rare in divers, but in compressed air workers they are about as common as lesions of the humeral head (Table VE-3).

One of the research difficulties has been trying to reproduce this condition in animals by exposing them to decompression. On the assumption that the condition in man is due to gas emboli, the

Table VE-3. Distribution of bone lesions in compressed air workers and commercial divers

Site	% Total Lesions	
	Comp. Air Workers (629 lesions in 281 men)	Divers (47 lesions in 25 men)
Humeral Head	29	42
Femoral Head	16	2
Lower End Femur	40	44
Upper End Tibia	15	12

explanation for failure in animals may be related to the discrepancies in circulation times and tissue clearance half times between the animals and man, which would lead to short bubble life. We have carried out some experiments in rabbits in which an intra-arterial injection of glass microspheres of 60-μ diameter have been used to simulate bubbles (2). In the animals it has been possible to produce bone necrosis in the femoral head which appears to be similar to that seen in man, so that this technique offers one line along which it may be possible to proceed in the study of this condition.

The more information which is obtained about human aseptic bone necrosis, the more likely it is that a proper understanding of this condition will emerge. Hopefully, it will eventually be possible either to prevent this condition from occurring, or to make sure when it arises that complete healing occurs.

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SESSION VI: CLINICAL STUDIES ON COMMERCIAL AND SPORTS DIVERS

SESSION VI: CLINICAL STUDIES ON COMMERCIAL AND SPORTS DIVERS

A. INTRODUCTION: E. BECKMAN

The following group of four papers represents the results of studies carried out by the Marine Medicine Division of the University of Texas Medical Branch, Galveston, over the past year on a group of 16 commercial and two sports divers.

The two sports divers (C.M. and H.L.) were each seen during the acute phase of their illness, i.e., within less than 24 hours after the accident. The others were first seen in the chronic phase of their injury. The post-injury interval varied from a minimum of 10 days (K.C.) to over 2 years, so that the degree of recovery varied from the initial phase to being complete.

These papers represent the related observations made by the consultants from various medical disciplines who collaborated in these studies. In addition to the findings that are documented, the patients were also referred to consultants in other specialties as indicated. For example, four of the patients had complaints which required referral for urogenital consultation. Thirteen divers were referred to the radiologist for consultation for the purpose of being evaluated for divers' osteonecrosis. Two of these divers had suspected lesions, which were confirmed. In addition, two other divers were found to have unsuspected juxta-articular lesions, and two others were found to have suspicious lesions which require follow-up study.

The following chart (Table VIA-1) demonstrates the extent of the multi-disciplinary evaluation carried out and also shows the multiple lesions which were revealed in many divers by the use of the multi-disciplinary examination approach.

Table VIA-1. Results of multi-disciplinary examination of injured divers, carried out at UTMB

Subject	Age, yr	Dive Experience, yr	Oto-neurological	Neuro-logical	Neuro.-Psychological	Ophthalm-ology	X-ray
J.A.	33	11	+	+	+	+	(-)
G.L.	32	2	+	+	(?)	+	(-)
L.A.	32	9	+	+	+	+	(+)
D.M.	30	12	+	+	+	+	(-)
J.B.	27	3	+	+	+	+	(-)
C.J.	34	14	+	+	+	+	0
T.B.	33	4	(-)	+	(?)	+	(-)
R.D.	38	15	+	-	(?)	+	(+)
W.D.	47	22	+	+	-	+	(+)
P.T.	28	6	+	(-)	-	+	(?)
J.M.	36	3 Mo	(+)	+	(?)	+	0
K.C.	32	10	+	+	+	+	0
D.M.	28	10	0	(-)	-	(-)	(?)
P.G.	28	4	0	0	-	-	(+)
R.T.	26	3	0	0	-	(-)	(+)
H.L.*	25		+	0	0	0	0
C.M.*	21	1 Mo	+	+	0	0	0
R.L.	23	2	0	0	-	0	0

* = sport diver; + = examined, positive, reported; - = examined, negative, reported; (+) = examined, positive, not reported; (-) = examined, negative, not reported; (?) = examined, results equivocal, not reported; 0 = not examined

B. OTONEUROLOGIC FINDINGS: P. E. WINKELMANN, V. G. CARUSO, M. J. CORREIA, J. T. LOVE, AND G. E. MILTENBERGER

Prior to 1960, many references to labyrinthine disorders appeared in the diving literature. Unfortunately, few cases were well documented. Vestibular and auditory symptoms were vague and were only mentioned in passing. Since then, however, several articles have appeared in which otoneurologic problems have been clearly defined. These articles emphasize the importance of otoneurologic diagnosis in divers and suggest both specific criteria for diagnosing vestibular and auditory involvement, and reasonable hypotheses for the mechanisms of injury (3,9-12,18-23,27-29,31). Edmonds (8) recently presented a detailed classification of the etiology of vertigo related to diving and described the characteristic features of each class. Based on cases reported in this article, we would like to expand this classification and modify it slightly, as shown in Fig. VIB-1. We believe that our ability to localize otoneurologic pathology results from the extensive and broadbased test batteries we use to identify peripheral and central injuries to the auditory and vestibular systems. We feel that these tests or similar tests which we shall describe should be part of every otoneurologic workup of injured divers.

Methods

Between September 1974 and May 1975, we examined 14 out of 18 commercial and sport divers who presented with auditory and vestibular symptoms attributed to incidents which occurred while diving. Eleven of these showed positive findings and will be reported on in this paper. The time between the incident and our examination varied from 18 hours to 24 months. A complete history, physical examination, routine audiometry, a central auditory test battery, and a vestibular test battery were performed. Using this evaluation, we found that we could classify our injured divers according to mechanism and site of injury, as shown in Fig. VIB-1.

Where practical, all patients were asked to fill out a Decompression Sickness and Air Embolism Questionnaire. The questionnaire specifically requests a detailed diving history, a description of the incident, details of the dive, any treatment, and response to treatment. Each patient was interviewed and a history of his present illness and past medical problems was taken. In those cases where patients were seen several months following the incident, detailed medical reports were requested from the physicians who examined the patient near the time of his diving incident.

A thorough otoneurological examination was performed, as outlined by Busis (4). This consisted of a general head and neck examination, tuning fork tests, tests of temporal orientation and memory for person, place, and time, general motor and sensory

VERTIGO IN DIVING - ETIOLOGICAL CLASSIFICATION
(Modified After Edmond's)

Due to Unequal Vestibular Stimulation

1. Caloric
   a. Unilateral external auditory canal obstruction
      (1) Cerumen
      (2) Otitis externa
      (3) Miscellaneous
   b. Tympanic membrane perforation
      (1) Shock wave
      (2) Middle ear barotrauma of descent
      (3) Forceful autoinflation
2. Barotrauma
   a. External ear barotrauma of descent
   b. Middle ear barotrauma of descent
   c. Middle ear barotrauma of ascent
   d. Forceful autoinflation
3. Inner Ear Barotrauma
   a. Fistula of the round window
   b. Fistula at the oval window
4. Decompression Sickness
   a. Peripheral decompression sickness
   b. Central decompression sickness
   c. Peripheral and central decompression sickness
5. Air Embolism
6. Miscellaneous

Due to Unequal Vestibular Responses

1. Caloric
2. Barotrauma
3. Abnormal gas pressures
4. Sensory deprivation

Fig. VIB-1. Classification of etiology of otoneurologic injury to divers (modified after Edmonds (9)). Modifications include addition of fistula of oval window under inner ear barotrauma, subclassification of decompression sickness into 3 categories, and addition of air embolism.

function, cranial nerve function, cerebellar function, and deep tendon and pathological reflexes.

Pure tone air- and bone conduction, speech, and discrimination tests were performed. When a sensorineural loss was found, Bekesy Audiometry, Short Increment Sensitivity Index, Carhart Tone Decay, Alternative Binaural Loudness Balance, Articulation Gain Function, and Acoustic Reflex Decay tests (1) were done to distinguish cochlear from retrocochlear lesions. Routine pure tone and speech audiometry is often normal in patients with localized or diffuse central nervous system disease. Therefore, a central auditory test battery as developed by Lynn, et al. (24-26), and modified by Willeford (personal communication), was used in testing these divers. This battery consists of four tests: the Binaural Separation and Filtered Speech tests which have been correlated with temporal and

parietal lobe pathology (25), and the Binaural Fusion and Alternating Speech Perception tests which have been correlated with brainstem pathology (24).

A vestibular test battery (VTB) which utilizes electro-oculography to measure horizontal and vertical eye movement was administered to each diver. Each diver also completed a vertigo questionnaire. The VTB includes oculomotor tests and vestibulo-ocular tests. The oculomotor tests are pendular eye tracking (7), optokinetic eye tracking (5), alternate gaze tracking (14), and static gaze (17). The vestibular end organs and vestibular nerve are tested by the vestibulo-ocular tests in which the labyrinths are stimulated and various parameters of the resulting electronystagmograms (ENG) are analyzed. The vestibular apparatus are stimulated by bithermal caloric, rotatory (torsion swing test) (13), and positional stimuli (with the patient in different positions relative to gravity) (2). In addition to static positional tests, we use the Hallpike maneuver to differentiate between central and peripheral pathology. We interpret a classic Hallpike response (15) as a sign of peripheral pathology. In all, we use the caloric test, the torsion swing test, spontaneous eye movements, and the Hallpike maneuver as tests of peripheral pathology. The torsion swing test, the oculomotor tests, and failure to suppress nystagmus with vision (16) serve as our differential tests for central vestibular system pathology.

Report of Cases

Case 1: This 30-year-old experienced commercial diver made repetitive dives to 168 fsw on July 10, 1974. His residual nitrogen time from his previous dive was 6 min, and his total bottom time on this dive was 15 min. He decompressed on a 170/20 (depth/bottom time) table (30) and approximately 5 min after reaching the surface noted the onset of blurred vision, dizziness, and difficulty expressing himself. He requested recompression and was treated on Table 6 (U.S. Navy Diving Manual) (30). He stated that after treatment he felt slightly improved, but he continued to feel quite unsteady while walking and was still having a great deal of difficulty with verbal expression. He was hospitalized for observation and further evaluation and discharged several days later with a diagnosis of decompression sickness affecting the left vestibular labyrinth. After returning home he became aware of persistent bilateral tinnitus which was previously unnoticed, marked unsteadiness in the dark, intolerance to loud noises, expressive dysphasia, and clumsiness when trying to work with tools. He also reported that he frequently bumped into walls and doors. His wife noted that the left side of his face did not move symmetrically and that he had become quite forgetful and short of temper. Physical examination on September 26, 1974 revealed an apparently healthy adult Caucasian male. The general ENT examination was normal. Mental testing revealed impairment of recent memory. There was decreased sensation

over the left side of the face and a mild-to-moderate left facial weakness, with widenings of the palpebral fissure and nasolabial fold. Motor strength was normal except for a mild drift of the left upper extremity. Sensory examination was normal except for impaired stereognosis and graphesthesia on the left. He walked with a broad-based gait, was unable to walk tandem, had a positive Romberg test, and demonstrated gross dysmetria and clumsiness, with rapid alternating movements bilaterally. Separate neurological psychometric evaluation revealed an organic brain syndrome. Routine audiograms showed a mild high-frequency bilateral sensorineural hearing loss, unchanged from a pre-injury study of October 8, 1973. Performance on all four central auditory tests was poor. The VTB showed possible ocular overshoots (14), asymmetrical pendular eye tracking, and a 25% optokinetic asymmetry suggesting borderline central vestibular system pathology. Repeated testing since that time has shown no change in the above findings, and he is classified as a case of central decompression sickness.

Case 2: This 32-year-old commercial diver with 10 years of experience noted the onset of whirling vertigo at his 50-ft water-stop while decompressing from a 60-min dive to 240 ft on a gas mix of 14% oxygen, 86% helium on August 23, 1972. He vomited and became confused. He was then brought to the surface in a basket and was recompressed and treated on Table 6A (30). After treatment he was still confused and continued to complain of whirling vertigo. The following day he was recompressed to 165 ft and decompressed at a rate of 4 ft/min. Following this treatment, he stated that he felt much better and was much more alert, but was still experiencing intermittent episodes of vertigo. Since that time he has noted balance and coordination problems, has had difficulty remembering things, concentrating, and expressing himself, and has suffered from increased irritability and sleep disturbance. An otoneurological examination, performed on August 31, 1972, showed anisocoria and retinal hemorrhages, as well as a broad-based gait, a positive Romberg, and a past-pointing on the right. ENG evaluation revealed a right-beating nystagmus, a positive-gaze nystagmus, an asymmetrical torsion swing test, and a reduced response on the right to caloric stimulation. Physical examination on September 26, 1974 revealed impairment of memory, weakness of the right upper extremity, decreased sensation over the right side of the face, a decreased corneal reflex on the right, and a spontaneous right-beating horizontal nystagmus. In addition, he was noted to walk with a broad-based gait and was unable to walk tandem without difficulty. He past-pointed on the right and had poor fine motor coordination. The hearing evaluation was normal. The VTB findings included a pathological right-beating spontaneous nystagmus (7.0°/s) and a pathological right-beating positional nystagmus in whole body and head turn right and left lateral positions. The VTB was readministered to this patient on February 8, 1975. At that time of testing, signs of central pathology within the

vestibulo-ocular system were noted. Specifically, an optokinetic asymmetry of 27% (right beating greater than left beating) and an elevated threshold (bilaterally) on the torsion swing test were noted. No difference in caloric response was noted at this testing and we suspect that central compensation of the labyrinthine peripheral insult had occurred. On the basis of the labyrinthine peripheral findings (August 31, 1972) and the central findings (February, 1975), we classify this patient as a case of mixed central and peripheral vestibular decompression sickness.

Case 3: On November 8, 1973, this 46-year-old veteran commercial diver descended to 246 fsw on air for 12 min. Decompression proceeded uneventfully until he reached his 10-ft stop, according to his pneumofathometer, and found himself on the surface. He then descended 10 ft on a weighted line and completed his last stop. Approximately one hour after leaving the water, he was sitting in the mess hall when he noted the sudden onset of decreased hearing, high-pitched tinnitus in the right ear, and whirling vertigo which rendered him unable to walk unassisted. He was then decompressed and treated on Table 5 (30). After 15 min of recompression on 100% oxygen, the vertigo subsided and his hearing improved. Following treatment he noted some decrease in hearing on the right, but complained mainly of tinnitus. He continued to dive until August 30, 1974, when he sought medical attention for persistent tinnitus, hearing loss, and episodic vertigo. Otoneurologic examination at that time was normal. Audiometrics revealed a right sensorineural hearing loss of cochlear origin, and initial ENG showed a right-beating spontaneous nystagmus with eyes closed, a direction-changing positional nystagmus, an asymmetrical torsion swing test, and a decreased response in the right ear to caloric stimulation. Otoneurologic examination on April 10, 1975 was again normal. Audiometric tests were unchanged (Fig. VIB-2). The central auditory tests and vestibular test battery were normal. This is classified as a case of peripheral vestibular decompression sickness in which central compensation has occurred.

Case 4: On May 26, 1975, this 22-year-old nurse was sport diving in the Gulf of Mexico at a depth of 30 fsw. She had been swimming into a strong current for about five minutes when she accidently dislodged her mask. She remembers trying to clear her mask and aspirating seawater, but she was unaware that she was ascending until she reached the surface. She believes that she held her breath during this ascent. On deck she complained of weakness, imbalance, and tightness in her chest. She was able to get to her cabin without assistance. Forty-five minutes later she was found to be confused and semiconscious. When aroused, she said that she could not see and could not move her left arm. She deteriorated rapidly and was comatose when placed in a chamber on deck for recompression. Treatment on Table 6A (30) was begun and she regained consciousness at about 100 ft. After 25 min at 165 ft,

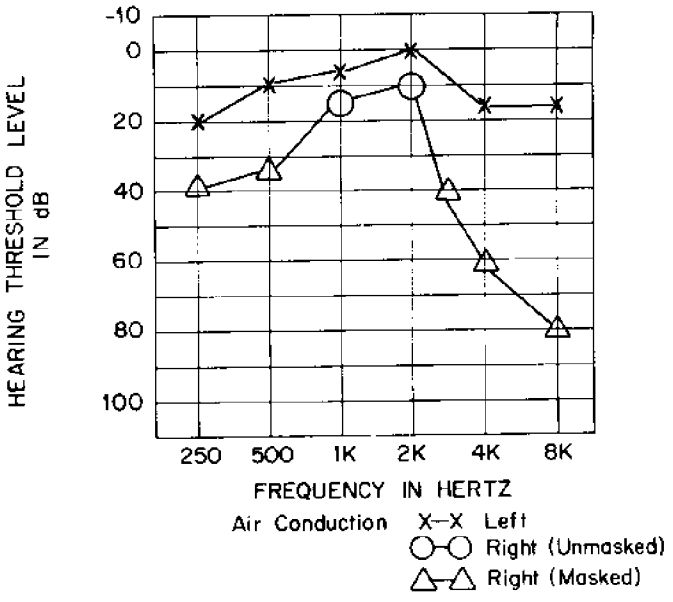

Fig. VIB-2. Case 3. Audiogram: this demonstrates mild low-frequency and severe high-frequency hearing loss in right ear.

she seemed completely normal. She presented the next morning complaining of imbalance, weakness and difficulty concentrating. Examination revealed weakness of the left upper extremity, anesthesia, and weakness of the left side of the face, and a positive Romberg with eyes closed. Air fluid levels were present in the middle ears bilaterally on otoscopic examination. The lungs were clear to ausculation and percussion, and there was no evidence of pneumothorax on radiographic examination. She was again treated on Table 6A (30). Re-examination the following morning revealed return of pinprick sensation in the left face and left arm although thermal differentiation was abnormal and mild facial weakness persisted. She no longer complained of imbalance and the Romberg test was no longer positive. There was also mild residual weakness of the left upper and lower extremities. After her final decompression, pure tone audiometry showed borderline normal scores and she was unable to complete the central test battery because of nausea. The VTB showed a 22% directional preponderance, but failed to provide sound evidence of a central defect. The torsion swing test showed depressed responses, slope and threshold in both runs (Fig. VIB-3). This patient's etiology is classified on the basis of the history and physical examination as a case of air embolism causing vestibular symptoms.

Case 5: This 25-year-old policeman was sport diving on March 20, 1975. He made repeated descents to a depth of about 25 fsw and noted no difficulty clearing his ears, though he customarily employed the Valsalva maneuver to clear. Almost immediately following his final ascent he noted tinnitus and the sensation of fullness in his left ear. On the way home he began experiencing

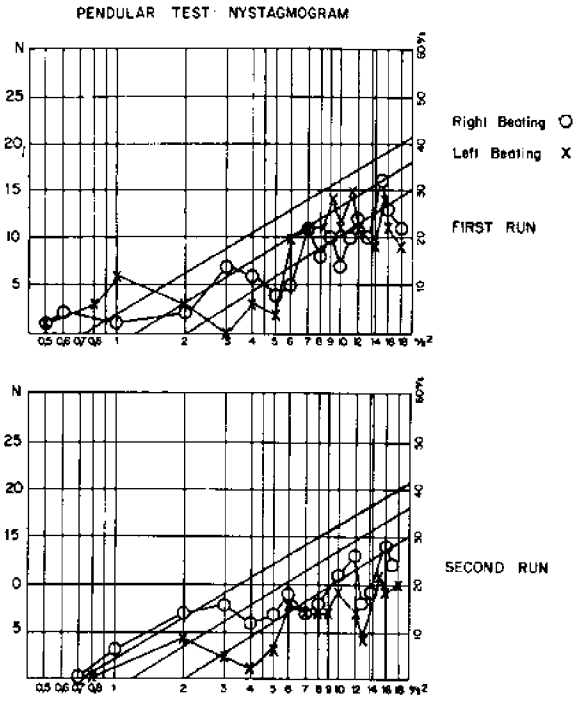

Fig. VIB-3. Case 4. Torsion swing test: results presented in this figure indicate bilateral hypoactivity for both right- and left-beating nystagmus with decreased slope and threshold.

whirling vertigo which was associated with nausea and vomiting and was precipitated by any head movement. He presented 24 hr later with persistent tinnitus and vertigo and states that he could stop the vertigo only by lying down with his head slightly elevated and turned to the right. Examination revealed a pale, dehydrated, Caucasian male in his mid twenties. Although otoscopic examination showed an air fluid level on the left, the Weber was midline, and Rinne was positive at 256 Hz and 512 Hz. The neurological examination was normal except for cerebellar tests which could not be done because the patient experienced severe vertigo when he closed his eyes. The audiogram revealed a bilateral high-frequency loss which was worse on the left. The VTB showed an 18.8°/s right-beating spontaneous nystagmus (Fig. VIB-4). In addition he had a 20% left unilateral weakness and 28% right-beating directional preponderance on caloric stimulation (Fig. VIB-4). A labyrinthine fistula was suspected and an exploratory left tympanotomy was performed the night of his admission. The stapes footplate was found to be fractured and subluxed into the vestibule with complete disruption of the annular filament as depicted in Fig. VIB-5. The stapes footplate was returned to its normal position and the fistula was closed with a fat graft taken from the lobule. The patient felt almost completely better immediately after the operation, and all vertigo and imbalance were gone by the third post-operative day. He returned to light-duty work two weeks later. Two months later the VTB remained abnormal with persistence of an 11°/s

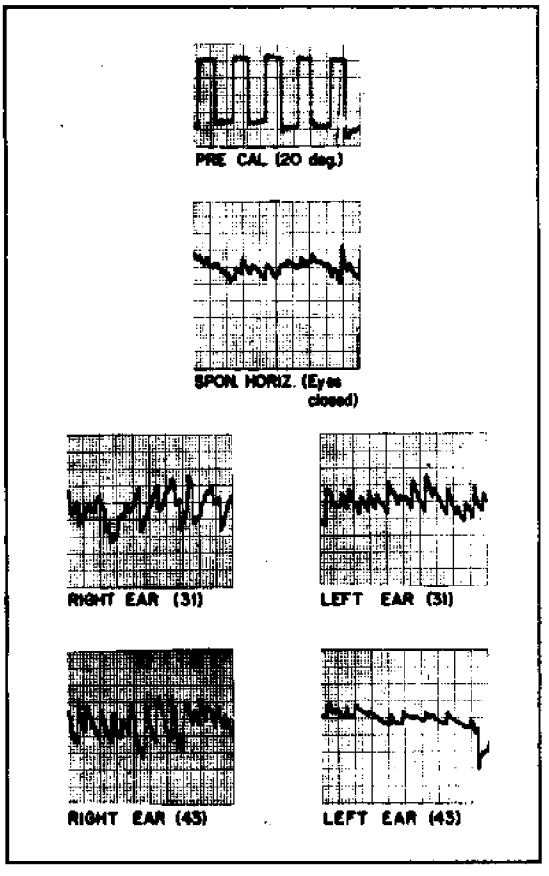

Fig. VIB-4. Case 5. Preoperative ENG tracings: this figure illustrates a right-beating (18.8°/s) spontaneous nystagmus and a right-beating directional preponderance (28% difference) caused by inability of left warm caloric to reverse right-beating spontaneous nystagmus.

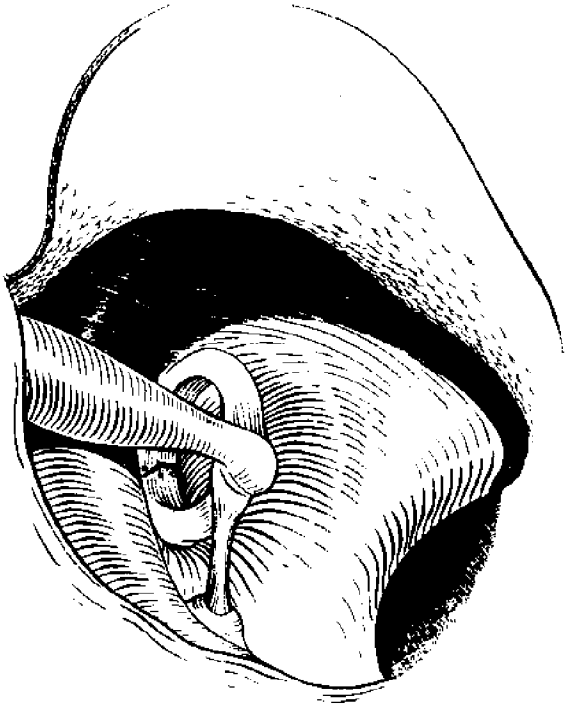

Fig. VIB-5. Case 5. Operative findings: stapes footplate fractured across middle and subluxed into vestibule; a perilymph fistula was noted.

spontaneous right-beating nystagmus. However, the patient has remained asymptomatic and there has been a 20-dB improvement in the pure tone threshold at 600 Hz and 8000 Hz. This case illustrates inner ear barotrauma, probably from auto-inflation.

Case 6: This 34-year-old man had been diving professionally for 14 years when he was injured on September 30, 1973. When injured, he was diving on air at 169 fsw. His compressor broke during the dive, sending oil into his air hose. He had dyspnea and ascended rapidly, developing back pain, nausea and vomiting, and losing consciousness. He was decompressed on Table 5A (30), but his nausea and vomiting persisted, associated with whirling vertigo. His vertigo persisted and became episodic, occuring one to two times per week. When first examined on December 5, 1974, his otoneurologic examination was normal. He had a mild high-frequency bilateral sensorineural hearing loss, and his central auditory tests were inconclusive. However, his vestibular test battery revealed marked bilateral vestibular weakness on caloric testing. This was reproduced on two occasions with no response to ice water calorics on one occasion. Additionally, the torsion swing test results showed marked bilateral vestibular weakness. We classify this as a case of bilateral peripheral vestibular decompression sickness.

Case 7: This 26-year-old professional diver had been diving for two years when injured on October 27, 1974. Decompressing from 140 fsw during a water stop at 30 ft on air, he noted nausea and pain and pressure across his face. He was recompressed for 10 min at 40 ft on oxygen and felt somewhat better. However, while driving home, he experienced whirling vertigo and a left facial weakness. This resolved over the next three weeks. On May 20, 1975, when first examined, his otoneurological examination was normal; his routine audiometry was normal; and his central auditory tests were normal, except for some possible asymmetry on his fusion tests. His VTB showed a pathologic left-beating spontaneous nystagmus which persisted in all head positions. His torsion swing test revealed a Phase II (13) compensation for a right peripheral vestibular lesion. Since his equivocal central auditory test scores on fusion tests were not substantiated by other brainstem signs or tests, we discount this finding and classify this as a case of peripheral vestibular decompression sickness.

Case 8: This 27-year-old man had been a professional diver for three years when he was injured on June 28, 1974. He had been diving to 560 fsw on a helium-oxygen mixture. At a 165-ft decompression stop, he developed whirling vertigo, nausea and vomiting, and an associated erythematous skin rash and right knee pain. (He developed right knee pain on decompression 1 1/2 years before, but it disappeared on recompression.) He was recompressed to 6 ATA for 8 hr. These symptoms resolved, but he remained unsteady on his feet and frequently bumped into things. He also noticed trouble

concentrating and expressing himself and trouble with recent memory. He tired easily and had altered sleep patterns. Otoneurologic examination on October 21, 1974, was normal. His audiogram was normal. However, his central auditory test battery was markedly abnormal for competing messages and filtered speech tests. These results were substantiated by neurological psychometric tests which revealed an organic brain syndrome with diffuse cerebral involvement. The vestibular test battery showed both central and peripheral findings. Central findings included asymmetrical pendular eye tracking (7,6) as well as poorly formed horizontal optokinetic nystagmus. No spontaneous nystagmus could be noted during the VTB. However, the patient had no response to bithermal calorics on initial visit with bilaterally depressed responses six months later. He is classified as a case of peripheral vestibular and central decompression sickness.

Case 9: This 32-year-old man had been a professional diver for ten years when he was injured. He had suffered multiple episodes of joint and cutaneous "bends" previously. After 38 min at 196 ft on air, he suffered abdominal pains and right leg numbness on his way up to the first decompression stop. He was sent to 200 ft for 120 min and then decompressed to 40 ft for 130 min on oxygen. Afterwards, his symptoms persisted and, in addition, he became unstable on his feet. His otoneurologic examination revealed diplopia on upward and downward gaze and he could not converge. He had a broad-based gait. Audiogram and central auditory test battery were not performed. His VTB revealed ocular dysmetria and pathological square wave eye movements (18). In addition to his organic brain syndrome diagnosed on separate psychometric evaluation and ophthalmologic injuries diagnosed on separate ophthalmologic examination, we interpret the above VTB findings as evidence of a central vestibular pathway injury, possibly involving the cerebellum, and we have classified his case accordingly as central decompression sickness.

Case 10: A 38-year-old professional diver was injured in August 1974. He missed several decompression stops while ascending from 250 fsw on helium and oxygen. His total bottom time was 60 min. He experienced vertigo and tinnitus without hearing loss. After recompression, he still had imbalance. When first seen in April 1975, he still had constant imbalance while walking and steady, high-pitched tinnitus in both ears. Occasionally, when bending over, he perceived the room to spin violently. His Romberg tests were markedly abnormal. His audiogram was normal except for a bilateral sensorineural high-frequency sloping loss. His central auditory test battery was normal except for low pass filtered speech which may be abnormal as a result of the high-frequency sloping loss. His VTB revealed a 26% right unilateral weakness, and the torsion swing tests suggested a centrally compensating peripheral lesion (13). This case is classified as a peripheral vestibular decompression sickness.

Case 11: A 31-year-old male had been a professional diver for nine years when he was injured in January 1974. While being decompressed from 200 fsw in a staged decompression chamber, he noted numbness in his left leg and face. He was decompressed back to 200 fsw and had a total of three separate chamber decompressions without relief of symptoms. When first seen in November 1974, he had the same symptoms plus memory problems, trouble concentrating, sleep difficulties, tinnitus, and imbalance. Examination revealed a wide-based gait and abnormal cerebellar signs, including finger-to-nose and Romberg tests. Routine audiogram was normal except for bilateral sloping high-frequency sensorineural hearing losses. His VTB was normal. However, his central auditory test battery revealed abnormal Binaural Separation tests of 60% bilaterally and abnormal low pass filtered tests of 50% bilaterally. His Alternating Speech Perception test was 50%. The psychometric testing revealed evidence of an organic brain syndrome, with marked memory impairment and a left-sided proprioceptive defect. This is supported by the results of the central auditory test battery. On the basis of the signs and symptoms, central auditory test battery and psychometrics, we classify this case as central nervous decompression sickness causing otoneurologic symptoms.

These cases illustrate the variety of injuries which may cause otoneurologic symptoms in divers. Of the cases presented, four were classified as having peripheral decompression sickness, three as central decompression sickness, and two as having both central and peripheral decompression sickness. One diver had inner ear barotrauma from an oval window rupture. One patient had otoneurologic findings from an air embolism.

ACKNOWLEDGMENT

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C. THE NEUROLOGICAL MANIFESTATIONS OF DECOMPRESSION ACCIDENTS: PATRICK J. KELLY AND BRUCE H. PETERS

The study of decompression sickness (Caisson's disease) in tunnel workers has suggested a wide variety of neurologic lesions (5, 6). Yet in decompression illness in commercial and military divers, the general belief that primary spinal cord involvement is its neurologic sequel has persisted into recent literature (3,7). In view of the fact that the ultimate pathophysiologic mechanism in each is vascular compromise with edema, hemorrhage, or infarction, and that there is probably little difference between decompression sickness in divers and in caisson workers, the disparity in the neurologic picture seen in each group is illogical (4).

Methods

Thirteen patients, evaluated for other system manifestations following decompression accidents, had complaints suggestive of CNS disorder and were referred for neurologic examination. The subjects were commercial divers who suffered decompression accidents from six months to two years prior to evaluation at our facility. The symptomatology and findings on routine neurological examinations performed on these patients is reported below.

Results

Table VIC-1 is a compilation of the manifest symptoms. The most common symptoms were headaches, personality changes, and deterioration

Table VIC-1. Symptoms of divers post-decompression illness

Headache	8
Personality change	9
Recent memory	8
Dyscoordination Gait/Extremity	6
Expressive dysphasia	3
Extremity paresthesia	5
Extremity weakness	4
Hearing loss	4
Urinary	3
Vertigo	3

$n = 13$

of recent memory. Six of the 13 patients complained of symptoms suggesting cerebellar midline and/or hemispheric involvement. Five subjects complained of extremity paresthesiae and four complained

of extremity weakness. Four patients complained of unilateral hearing loss. Vertigo and urinary symptoms were each found in three patients.

The findings on neurologic examination are given in Table VIC-2. Recent memory deficit, as evidenced by digit span testing, was seen

Table VIC-2. Neurological findings in patients following decompression illness

Recent memory impairment		7
Nystagmus		4
Cerebellar sign		7
Midline	2	
Hemispheric	2	
Both	3	
Brainstem		7
Hearing loss		3
Sensory loss		3
Pyramidal weakness		4
Negative exam		2

n = 13

in seven patients. Brainstem findings, such as facial weakness and/or hypesthesia or gaze dysconjugacy, were found in seven subjects. Cerebellar findings, such as midline (truncal) ataxia, hemispheric (limb) dystaxia, or a combination of both, were found in seven patients. Pyramidal weakness with hyperreflexia, clonus and the Babinski sign were found in four patients. Two patients had a negative examination.

Characteristically, the patients did not exhibit findings localized to one structural CNS locus, but had scattered findings attributable to supratentorial, brainstem, cerebellar, or spinal cord lesions, as demonstrated in Table VIC-3.

This can be illustrated by the following two cases.

Case 1. D.M., a 30-year-old male diver with six years' experience, was examined after he had had a decompression accident. Prior to his accident, he had been making repetitive air dives to 175 fsw. Upon arrival on the diving deck of the ship, he noted visual disturbances, some difficulty in expressing himself, and unsteadiness of his gait. He was placed in the decompression chamber and given treatment on USN Treatment Table 6, and his symptoms cleared slightly. Since that time, he has noted unsteadiness of

Table VIC-3. Lesion sites in commercial divers following decompression accidents as determined by neurological examination

Neocortical	7
Cerebellar	7
Brainstem	7
Spinal cord	3
Negative exam	2

his gait, occipital headaches, and difficulty in expressing himself. He also complains of poor recent memory and being unable to recall the simplest bits of information, such as street addresses, phone numbers, and so forth. His interaction with his surroundings has been slower, he feels, and he noted difficulty in processing his thoughts, word finding, and arithmetic problems. Paresthesiae and weakness of his left upper and lower extremity with the upper being weaker than the lower has been present since his accident.

On examination, the patient was an apparently healthy male who walked with a broad-based gait. He was unable to walk tandem, and the Romberg test was positive. Tests of cerebellar hemispheric function were performed poorly with gross dystaxia of upper and lower extremities bilaterally. Mental status examination revealed that the patient was oriented with fair judgment and fair grasp of general information. His recent memory was impaired so that he was unable to recall a series of more than four to five numbers in digit span. Cranial nerve examination demonstrated bilateral nystagmus on lateral gaze and facial hypalgesia on the left side with a decreased corneal response. A left peripheral facial weakness was also found. Weber testing lateralized to the right, with air conduction being greater than bone conduction bilaterally. Sensory examination revealed a decrease in graphesthesia and stereognosis on the left side of the body with mild construction dyspraxia. There was a mild left hemiparesis with a drift to the left upper extremity and distraction, mild hyperreflexia and Babinski on the left side.

Comment. This patient's findings suggest diffuse neurologic lesions in the cerebrum, namely the temporal, right frontal and parietal lobes, the archi- and neocerebellum or those pathways, and an intrinsic left lateral medullary plate brainstem lesion.

Case 2. L.A. is a 31-year-old male who has been a commercial diver for eight years. He had a decompression episode seven months prior to his evaluation here following a 230-ft saturation dive. While taking a shower after what was thought to be normal decompression,

he noted numbness of his left hand and decreased sensation of the left side of his face. On the next day, he noted clumsiness in his left leg. Since that time, he has complained of poor control of his anal sphincter, urinary frequency, nocturia, stress incontinence, and a decrease in his urinary stream. He has also been impotent. He also complains of bilateral tinnitus and reduced auditory acuity in his left ear.

He has noted a recent memory impairment, and complains of difficulty in expressing himself, being unable to find the correct words to do so. He has noted a personality change: he is irritable and short-tempered, when he had previously been easygoing.

Examination was grossly normal. Sensory examination revealed hypalgesia to pin in the left leg and lower thorax to the T9 level, with a mild degree of sacral sparing, though there was decreased proprioception and vibration bilaterally in the lower extremities, worse on the right than the left. Light touch was intact bilaterally.

There was no gross motor weakness, but the deep tendon reflexes were brisker on the right lower extremity than on the left, and there was a Babinski on the left. Gait was broad-based, and the patient was markedly ataxic with tandem gait. Romberg was positive. No cerebellar hemispheric signs could be elicited, however. Rectal examination revealed decreased tone with no voluntary contraction.

Comment. This case illustrates the type of problem commonly associated with diving decompression illness, namely a spinal cord lesion with a mild Brown-Sequard syndrome at the mid-thoracic level. There are also temporal lobe limbic symptoms and findings, and the suggestion of midline cerebellar involvement as well.

Discussion

In our experience, pure spinal cord involvement alone has been rare in diving decompression illness. It appears that our patients would fall into the categories delineated by Rozsahegyi in his study of patients with Caisson's disease, in which he was able to classify patients into the following groups: 1) the syndrome of multiple focal injuries to the whole CNS; 2) multiple lesions of the CNS and upper brainstem; 3) injuries to the pons, medulla, and cerebellum; and 4) the syndrome exhibiting spinal lesions (5,6).

There is pathophysiologic controversy as to whether the neurologic lesions are produced by arterial embolization of bubbles or by epidural obstruction (1,2,3) and over the possibility that both mechanisms might be involved in decompression ilness (4). Spinal cord pathology does occur following decompression illness, and

statistically the spinal cord is a low probability target for arterial emboli, whereas the distribution of the cerebral, cerebellar, and brainstem findings involve lesions which could be explained on the basis of embolic interruption of the arterial blood supply.

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D. NEUROPSYCHOLOGICAL SEQUELAE OF DIVING ACCIDENTS: HARVEY S. LEVIN

Sequelae of neurological forms of decompression sickness have been amply documented in divers and caisson workers (7,8). The disorders described by Rozsahegyi (7) include 1) multiple focal injuries of the whole central nervous system (CNS); 2) multiple lesions in cerebrum and upper brainstem; 3) injury to the medulla oblongata, pons, and cerebellum; and 4) spinal lesions. The original report (7) and a later paper (8) both emphasized the frequent finding of neurotic and psychosomatic symptoms associated with CNS decompression illness.

Rozsahegyi reported that his patients frequently manifested irritability, anorexia, disturbance of sleep, alcohol intolerance, sexual dysfunction, and forgetfulness. He also noted a psychosomatic syndrome characterized by "pseudo-neurasthenia, hysteria, and pathological change of personality." Neurologic complaints included headache, dizziness, weakness, buzzing in the ear, and disturbance of hearing. The observed association between neurologic deficit and EEG abnormality with psychiatric disturbance led Rozsahegyi to postulate that the latter was a consequence of an organic lesion rather than of psychogenic origin. Psychiatric symptoms persisting for several years following the onset of decompression illness were restricted to cases in which clinical findings implicated the upper segments of the CNS. The author stressed the importance of careful clinical examination in view of the possibility of widely dispersed lesions of small size.

Although recent reviews of decompression sickness studies (4,7) have indicated that CNS lesions in man are often apparently confined to the spinal cord, it is plausible to speculate that subtle behavioral effects are usually overlooked. Consistent with this possibility, Biersner and Ryman (1) found that the psychiatric incidence rate among U.S. Navy divers was over twice that of a non-diving control group. In comparison with other Navy psychiatric patients, a disproportionate number of divers were diagnosed as having situational maladjustment. The present study was undertaken to assess neuropsychologic deficits and psychiatric disturbance in divers by utilizing quantitative behavioral measures that are known to detect psychologic manifestations of CNS injury.

Methods

Subjects. Fifteen commercial divers were given a comprehensive neuropsychological evaluation as part of an interdisciplinary clinical investigation undertaken at the University of Texas Medical Branch, Galveston. All of the divers studied were males complaining of symptoms following an accident; litigation was pending in most cases. Of the total group, the clinical history was

consistent with decompression illness in nine divers and hypoxic injury in a single case. Five divers who sustained injuries which did not cause signs or symptoms of CNS involvement were designated as a control group for comparison with a group of 6 divers whose complaints of CNS symptoms were substantiated by the test battery (impaired group). An additional group of 4 divers with equivocal neuropsychological findings were excluded from the data analysis for specific tests because their results raised the question of contamination by a deviant response strategy.

Biographic data for the control group and impaired divers are given in Table VID-1. Preliminary statistical comparison with the Mann-Whitney U Test did not disclose evidence of significant group differences with respect to age, years of education, or diving experience. Within the control group, divers were tested after an average time period of 15.7 months (SD = 10.2) following their diving accident. The recovery interval for impaired divers ($\bar{X}$ = 11.3 months, SD = 8.7) did not significantly differ from that of the control group, U = 12. In view of these findings, the control and impaired groups were compared on cognitive and personality

Table VID-1. Comparison of control and impaired divers on biographic and performance measures.

Dependent Variable	Control, n=5 $\bar{X}$	SD	Impaired, n=6* $\bar{X}$	SD	Mann-Whitney U
Age	29.4	9.3	31.0	2.4	7.0
Education	12.3	1.1	12.4	2.0	14.0
Diving experience - yrs	7.8	9.0	8.5	5.7	13.0
Full Scale IQ	107.0	10.7	85.3	10.9	1.5**
Verbal Scale IQ	106.0	11.8	86.7	13.1	4.0**
Performance Scale IQ	107.4	9.2	82.8	11.1	0.0††
Wechsler MQ	105.5	11.6	79.0	16.0	4.0**
Forward digit span	7.4	1.3	4.6	0.6	0.0††
Supraspan total	34.2	6.4	20.9	3.5	1.0†
Supraspan slope	1.2	0.3	0.9	0.4	6.0
Supraspan T_5	9.5	1.1	5.8	0.9	0.0††
Trail A, s	31.6	5.9	59.5	27.2	6.5
Trail B, s	68.2	30.1	126.0	24.9	1.0†
Tapping-dominant	49.8	4.3	30.1	14.8	5.0
Tapping-nondominant	46.3	2.9	31.8	9.8	2.0††

*only 5 impaired divers took the Supraspan test; **$P < 0.05$ (2-tailed); †$P < 0.01$ (2-tailed); ††$P < 0.005$ (2-tailed)

measures comprising the neuropsychological test battery. Equivocal patients were included only in the analysis of presenting complaints.

Procedures. Each diver was tested individually in a laboratory under controlled conditions. The portion of the complete test battery included in this report includes the Wechsler Adult Intelligence Scale (WAIS), Wechsler Memory Scale (11), Reitan Trail-Making (2), Reitan Finger Tapping (6), Supraspan Digit Storage test by Drachman and Leavitt (3), and the Minnesota Multiphasic Personality Inventory (MMPI). The Trail-Making test consists of printed circles randomly distributed on a sheet of paper. Trail A is formed by the subject drawing lines to connect the circles in numerical order. Line segments in Trail B must alternate between numbers and letters enclosed in each circle, i.e., 1-A-2-B, etc. Time required to complete the trail is the performance measure. The Reitan Finger Tapping involved 5 trials of 10 seconds during which the subject pressed a telegraph key with his index finger. Mean number of taps for the dominant and nondominant hands was the dependent measure. With the exception of the Supraspan, these instruments are standardized tests (10) routinely employed in clinical psychodiagnostic assessment.

The Supraspan task was tape recorded and presented through loud speakers situated in front of the subject. Three random sequences of 12 single-digit numbers were presented at a rate of 1 digit per second for 5 trials each. The subject was asked to repeat orally the sequence of numbers in the original order immediately following each presentation. Correct repetition of the initial and final numbers in each series was credited as were correct items adjacent to correct first and last items in a series. All groups of three or more items whose order corresponded to a similar sequence in any location in the digit series were scored as correct. Mean items correct for each of five trials were calculated for the three sequences of digits. Performance measures employed for the Supraspan test were: 1) mean total number of items correct across all five trials for the three sequences; 2) the slope of the learning curve relating number of correct items to trials; and 3) number of correct items on the fifth trial (T_5). A tape-recorded test of immediate memory which measured forward digit span was given prior to the Supraspan task. Beginning with a sequence of two digits, progressively longer random series were presented for two trials each. The longest sequence perfectly recalled at least once was considered the subject's digit span. A single impaired diver was referred for evaluation prior to the availability of the Supraspan tape.

Results

Inspection of Table VID-2 indicates a wide range of symptomatic complaints of both a neurologic and psychiatric nature. Irritability

Table VID-2. Frequency of neuropsychiatric complaints by divers

	Frequency	Percentage
Anxiety	1	15
Reduced Concentration	2	13
Depression	2	13
Fatigue	3	20
Irritability	10	66
Decreased Libido	2	13
Sexual Dysfunction	4	27
Sleep Disturbance	6	40
Decreased Hearing	2	13
Numbness	5	33
Visual Distortion	5	33
Buzzing in Ear	7	47
Forgetfulness	6	40
Dizziness	8	53
Clumsiness	5	33
Headaches	4	27
Disturbed Word Finding	7	47
Muscle Twitching	1	7
Pain	3	20

n = 15

was the most frequently mentioned behavioral disturbance; information obtained from the wives of the divers confirmed an intensification of marital discord. Psychosomatic and depressive aspects of the clinical picture also included sleep disturbance, sexual dysfunction, fatigue, and depression. Table VID-2 shows that dizziness, buzzing in the ear, word finding difficulty, and forgetfulness were the most frequent neurological complaints. Memory disturbance was often described in terms of misplacing household objects or forgetting plans from one day to the next. Divers with normal neuropsychological test findings tended towards fewer symptomatic complaints ($\bar{X}$ = 4.0) than divers manifesting behavioral deficits ($\bar{X}$ = 6.0) though the difference was not statistically significant (U = 8.5).

General decline in cognitive functioning of the impaired divers is suggested by their WAIS scores as compared to control divers (Table VID-1). The difference is particularly impressive on Performance Scale IQ, reflecting scores on timed tests sensitive to motor speed, coordination, and appreciation of visuospatial relations. Consistent with the possibility of reduced motor speed, finger tapping using the nondominant hand was markedly slowed in the impaired group whereas this effect was not obtained for the dominant hand.

Figure VID-1 shows the learning curves for acquisition of digits on the Supraspan storage task. The impression of superior Supraspan storage and forward digit span in the control group was substantiated by statistical analysis (Table VID-1). The Wechsler Memory Quotient (MQ), a composite measure of several short-term memory subtests, was also lower in the impaired group. Although control divers tended to complete both Trails in less time than the impaired group, the difference was significant only for the more complex Trail (B).

Standard scores on the MMPI were averaged within each group of divers and are shown in Fig. VID-2. Elevations in standard scores to 70 are two standard deviations above the standardization population (standard score conversion yields a mean of 50 and standard deviation of 10) in the pathological direction. Of the 10 clinical scales, 5 were elevated to at least 70 in the composite profile for the impaired divers, whereas the group profile of control divers did not include a single elevation of this magnitude. This pattern of results suggests severe emotional disturbance in the impaired divers. Mean number of scale scores above 70 was 1.8 in the control group (SD = 1.3) as compared to 4.0 (SD = 0.9) in the impaired group; these means were significantly different, (U = 3, $P < 0.03$). Acute distress, depression, and anxiety in the impaired group are apparent in the profiles given in Fig. VID-2. These divers also showed significantly greater somatic concern and disruption of cognitive efficiency. Elevations on scales 7 (Psychasthenia)

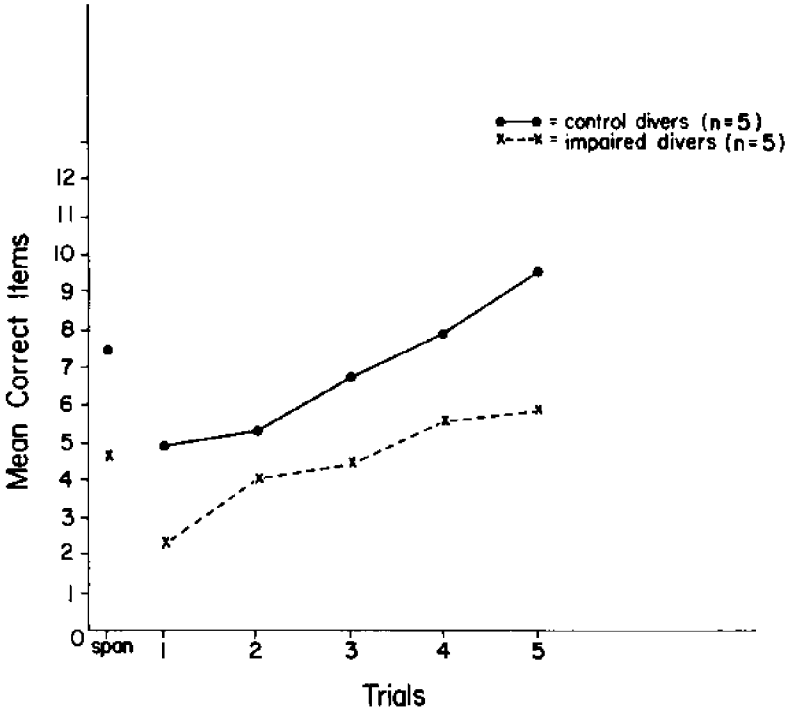

Fig. VID-1. Mean forward-digit span and Supraspan score across trails in control and impaired divers.

and 8 (Schizophrenia) are compatible with ruminative, if not obsessive, thoughts and excessive worry in the impaired divers. Pairwise comparison utilizing the Mann-Whitney U test confirmed significantly higher scores on the Hypochondriasis ($U = 3$, $P < 0.03$) and Psychasthenia ($U = 2.5$, $P < 0.02$) scales whereas the analysis of Depression scores approached significance ($U = 5$, $P < 0.08$). The impression gleaned from Fig. VID-2 of comparable scores by control and impaired diving groups on validity scales (e.g., ?, L,F,K) was supported by the results of analysis which did not approach significant levels.

Discussion

Utilization of standardized psychometric instruments and experimental memory tasks demonstrated the presence of residual neuropsychologic deficit in divers whose pattern of findings indicated CNS involvement. Persistent cognitive impairment and psychiatric disturbance were detected in several divers who were examined more than a year following their diving accident and decompression sickness.

Consistent with the description by Rozsahegyi (7) of the clinical picture associated with decompression illness, impaired divers in

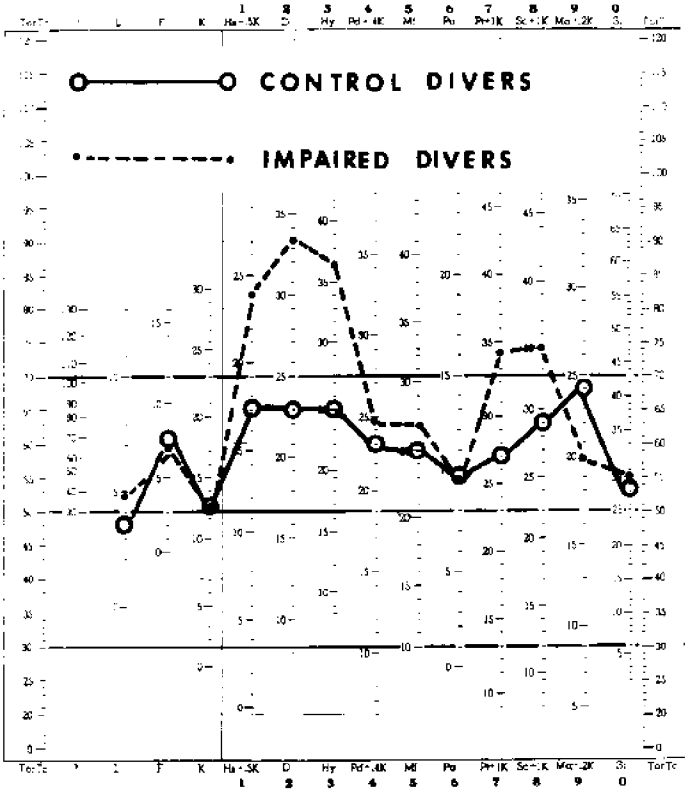

Fig. VID-2. Mean standard scores on validity and clinical scales of MMPI in control and impaired divers (Hs=Hysteria, D= Depression, Hy=Hypochondriasis Pd=Psychopathy, Mf=Sexual inversion, Pa=Paranoia, Pt=Psychasthenia, Sc= Schizophrenia, Ma=Mania, Si=Social Introversion,

the present study frequently complained of forgetfulness and demonstrated a memory deficit on psychological tests. Assessment also disclosed retardation of motor speed and decreased efficiency of information processing and storage. Previous clinical observations of psychosomatic and neurotic symptoms (7,8) were confirmed by results on a standardized personality inventory and by interview material. Psychologic malaise, depression, anxiety, somatic concern and ruminative thinking were pervasive among divers who manifested behavioral signs of CNS involvement. These characteristics were absent or greatly attenuated in divers whose findings and clinical history did not suggest CNS involvement.

Although the present findings accord with those reported by Rozsahegyi (7,8), they deviate from the prevailing view that CNS involvement in decompression sickness is essentially confined to the spinal cord (4,9). It must be recognized that the divers included in this investigation were not a representative sample of patients recovering from decompression sickness or diving accidents in general. However, the results provide persuasive evidence for the inclusion of neuropsychological assessment in future studies concerning the sequelae of decompression illness. Quantitative

behavioral measurement performed under controlled conditions may uncover deficits caused by diffuse lesions which are not of sufficient size to consistently produce abnormalities on routine neurologic examination.

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E. VISUAL DISTURBANCES FOLLOWING DECOMPRESSION SICKNESS: MICHAEL E. LIEPPMAN

Twelve professional divers with visual complaints following decompression sickness were evaluated. Severe accommodative and convergence insufficiency heretofore unrecognized was detected and studied. Accommodation refers to the adjustment of the eye for various distances by alteration of the convexity of the lens. The triad of accommodation, convergence, and pupillary constriction is called "the near reflex" (5).

Blurry vision is a common complaint in bent divers (2). Air embolism has caused known cases of central retinal artery occlusion with blindness (3). In cases this severe, the divers usually died from other CNS bubbles causing vascular occlusion or hemorrhage. Behnke showed that 100% O_2 at three ATA for three hours caused a transient constriction of the visual field to ten degrees (6). Visual cell death, retinal detachments, and cytoid body formation have been reported in animals subjected to 24 hours or more of O_2 at high pressures (6). The blurry vision in divers was felt to be due to vasomotor instability with decreased caliber of the veins (15%) and arteries (10%) on exposure to 100% O_2 (more under pressure) (6). Vitreous bubbles, tear bubbles, and nystagmus are also causes of blurry vision. These three are all reported to clear within one month of the episode. Accommodative insufficiency is another cause of blurry vision which has never before been described in divers. Bietti described temporary accommodation deficiency in high altitude pilots (1). The divers seem to have a more permanent disability.

Materials and Methods.

The study group consisted of 12 divers, who were examined from 2 wk to 36 mo after their bending accidents. They were between the ages of 26 and 46, and all but two had experienced symptoms between 5 and 7 atmospheres, while breathing compressed air.

Ocular Symptomatology (Table VIE-1). Eleven of the 12 divers complained of blurry near-vision and tired eyes after 5 min of reading. This was not relieved by their present optical prescription. Half of the study group complained of episodes of non-formed flashes of light and micropsia. Three of the divers had double-vision at near, and the other two complained that their night vision had suddenly become poor after the accident. These complaints were all studied subjectively as well as objectively.

Ophthalmic Examination. Microscopic exam of the anterior segments, indirect fundoscopy, tangent screen fields, optokinetics, color vision testing, refraction, and accommodation and convergence measurements were performed on each participant in the study. The

Table VIE-1. Ocular symptomatology; series of twelve case studies.

Symptom	Per cent	Importance
Blurry near vision	92%	Accommodative insufficiency
Asthenopsia-"eyes tire after 5 min reading"	92%	1. Accommodative insufficiency 2. Convergence insufficiency 3. Premature presbyopia
"White flashes of light", episodic non-formed visual hallucinations	50%	Occipital or parieto-temporal irritation
Micropsia-"Things up close become suddenly smaller, as if far away"	41%	Accommodative insufficiency Temporal lobe irritation
Double vision, Diplopia at near	25%	Convergence insufficiency
Nyctalopia (night blindness)	16%	Possible sensory retina damage

two divers complaining of night-blindness received dark-adaptation studies and electroretinography. Near points of accommodation and convergence were measured objectively and subjectively by the push-up method on the phoropter. Accommodative amplitudes were measured at 33 cm. Exophoria was measured by prism-cross-cover. Fusional convergence reserve was measured by double rotary prisms at 40 cm to break and to blur.

Findings.

All the divers corrected to 20/20 in both eyes. Four divers with myopia had worn spectacles since childhood. The near points of accommodation and convergence and their amplitudes were grossly below the lower limits of normal in 11 of the 12 cases. A control study of 8 divers who had never had decompression sickness showed normal values of accommodative and convergence amplitude. Tables VIE-2 and 3 are arranged in order of increasing time since the accident. Table VIE-2 shows the dramatic abnormalities of convergence and accommodation, while Table VIE-3 analyzes accommodative convergence-accommodation ratios and exophoria. Sixty

Table VIE-2. Optometric analysis

Case No.	Time of Accident	Age	Near Point of Accommodation, cm	Near Point of Convergence, cm	Amplitude of Accom. at 13" (Diopters) O.D.	O.S.
1.	2 wk	32	95	100	1.00	1.25
	2 mo		95	95		
	3 mo		85	87	1.25	1.50
	4 mo		60	65	2.00	2.00
2.		32	50	25	1.50	1.50
3.	3 mo	30	40	30	2.00	2.00
	7 mo		45	40	2.50	2.50
4.	4 mo	36	50	20	1.25	1.75
5.	6 mo	27	25	15	2.00	3.00
6.	7 mo	26	33	20	3.00	3.00
7.	10 mo	31	30	20	2.00	2.00
	13 mo		30	20	2.25	2.25
8.	15 mo	34	25	25	2.75	2.25
9.	18 mo	46	40	50	0.75	2.00
10.	25 mo	32	25	40	2.50	2.50
11.	30 mo	32	24	40	2.75	3.25
12.	36 mo	28	10	10	5.00	5.00

Table VIE-3. Optometric analysis

Case No.	Age	AC A	Phorias by Prism Cover Test (Prism Diopters) Near	Distance
1.	32	3.2	14 XT 14 XT 10 XT	7 XT 4 XT 4 XT
2.	32	3.2	7 XT	Ortho.
3.	30	4.0	5 X	Ortho.
4.	36	2.0	10 X	2 X
5.	27	4.8	3 X	Ortho.
6.	26	4.2	5 XT	2 XT
7.	31	4.0	5 X	1 X
8.	34	1.2	12 X	Ortho.
9.	46	3.6	6 X	Ortho.
10.	32	3.6	6 X	Ortho.
11.	32	5.2	2 X	Ortho.
12.	28	5.2	2 X	Ortho.

per cent show abnormal exophoria or tropia at near. Twenty-five per cent show abnormally low AC/A ratios.

Table VIE-4 compares the abnormally low accommodative amplitudes to the computed mean norm per age. Figure VIE-1 illustrates the low accommodative amplitudes related to normal per age as described by Duane's Curve. The near reaction was abnormal except for pupillomotor function which showed the normal pupillary constriction (near synkesis) upon stimulation of accommodation with the near card at 33 cm.

One diver (Fig. VIE-2), followed at regular intervals beginning two weeks after recompression therapeutics showed a slight improvement in accommodative amplitude followed by a plateau at the 2-diopter level. A pharmacological experiment utilizing Carbachol

Table VIE-4. Accommodative amplitude of diver compared to norm and low norm per age (Hofstetter, 1968)

Case No.	Age	Diver's Amplitude	Computed Low Normal	Computed Mean Normal
1.	32	1.00	7.00	8.3
2.	32	1.50	7.00	8.3
3.	30	2.00	7.50	9.5
4.	36	1.50	6.00	7.7
5.	27	3.00	8.25	10.4
6.	26	3.00	8.80	11.0
7.	31	2.00	7.25	9.2
8.	34	2.50	6.50	8.3
9.	46	1.37	2.50	4.7
10.	32	2.50	7.00	8.9
11.	32	3.00	7.00	8.9
12.	28	5.00	8.00	9.9

and Physostigmine instilled in the eyes of 6 of the participants showed that the accommodation lesion is not in the ciliary body, ciliary ganglion, or lens, as it is in premature presbyopia. That is, the lesion is not intraocular. This method was described by Tornqvist in 1971 (4).

One of the two divers with complaints of night-blindness had a depressed (abnormal) dark-adaptation study. However, the electro-retinogram was normal. Seven of the divers had some form of nystagmus (pathologic). Three of the divers had lattice degeneration of the retina, but this was probably present before the accident.

Therapeutics. The symptoms of blurry near vision and asthenopsia could be alleviated in all of the divers with reading glasses with increased plus power and base-in prism. The two divers with diplopia were also cured of that symptom by this therapy. Several different medical regimens failed to alleviate the micropsia and non-formed hallucinations.

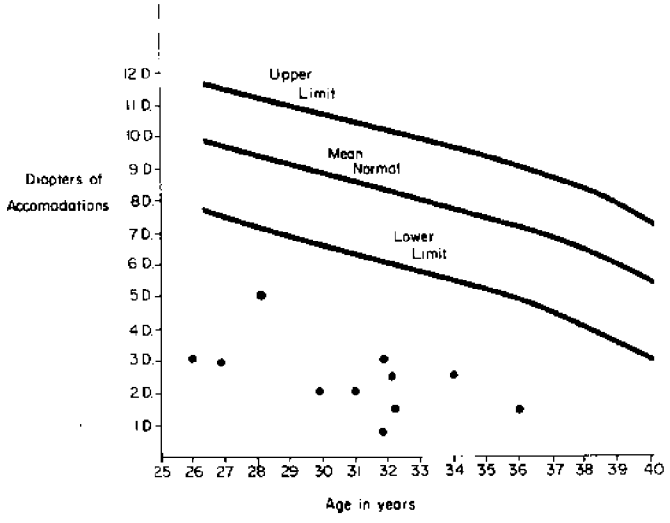

Fig. VIE-1. Accommodative amplitude related to normals per age (Duane, 1925).

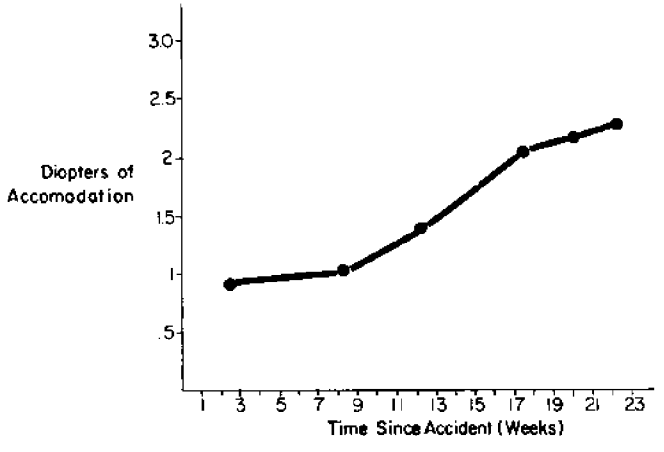

Fig. VIE-2. Accommodative amplitude related to elapsed time since accident (Case No. 1).

Primate Experiment. A Rhesus monkey was bent on a bounce profile dive in a pressure chamber on three separate occasions. Each time the animal was taken to 200 feet for less than 45 minutes. Retinoscopy after the first two dives revealed a decreased hyperopia. The animal died on the third dive, and the eyes were removed and studied pathologically to look for signs of ciliary body changes that would explain the accommodative insufficiency. None were found. The following abnormalities were noted: retinal hemorrhages, air bubbles in the anterior chamber, vitreous, cytoid bodies (cotton-wool spots), and focal retinal detachment.

Summary

A series of twelve divers with visual complaints following the bends were evaluated. All had accommodative and convergence insufficiency that did not clear with time. This is the first report to describe this longstanding visual disability in divers. Animal studies are now underway to localize these effects.

Address all questions to: Michael E. Lieppman, Department of Ophthalmology, University of Texas Medical Branch, Galveston, Texas 77550.

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CHAIRMAN'S SUMMARY: E. L. BECKMAN

The papers which were presented in this morning's sessions have clarified the problems which have faced investigators in diving research for many years. For the first time it would seem that these investigators have, in the vernacular, "brought it together", so that pressure physiology is becoming a single discipline and is no longer divided into the problems of the caisson worker, diver, and aviator.

I left the U.S. Navy research program in diving decompression approximately 10 years ago. At that time the U.S. Navy taught that dysbaric osteonecrosis was a disease affecting only caisson and tunnel workers. U.S. Navy divers, and those who followed U.S. Navy air tables, were believed to be immune to this disease.

The Navy also believed that the U.S. Navy air diving tables were sacrosanct, and that the use of the U.S. Navy diving tables did not produce decompression sickness with any significant frequency. More interesting was the belief which existed and has persisted in the Navy (and which has even been quoted in this symposium) that the CNS-type of decompression sickness which affects divers involves only the spinal cord. We were taught that in some miraculous way the tissues of the brain were not damaged. This belief persisted despite the excellent review by Rozsahegyi (2), which enumerates the various lesions in the telencephalic, diencephalic, mesencephalic, metencephalic, and myelencephalic structures of the brain, which he observed both clinically and at autopsy on caisson workers who had suffered from central nervous system decompression sickness.

In the development of diving decompression tables, the "main line" held that there were no such thing as silent bubbles or bubble micronuclei despite the excellent work by E. Newton Harvey (1) in 1948-50. The end point used in the development of decompression tables was that of joint pain. This was rationalized to be a safe end point because pain was believed to be due to only a single bubble which lodged at some focus and produced painful symptomology. Although these concepts did not hold together from a scientific point of view, they were nonetheless an accepted doctrine.

When I left Navy diving research, I came to Texas and met some of my Navy diver friends who had also left the Navy and had become commercial divers in the "oil patch". It was then that I learned that commercial divers, such as my friends who were former U.S. Navy divers, had problems with decompression sickness when they used the U.S. Navy air tables below 150 fsw for working dives. I learned also that they regularly had trouble with decompression sickness when they made repeated dives (i.e., one dive every 12 to 14 hours over a several day period), although still following

"the book". I learned too that the U.S. Navy helium diving tables produced decompression sickness so frequently that the commercial diving companies had developed their own helium diving tables. I obtained bone radiographs of a few of the commercial divers working in the Gulf who had been trained in the Navy and used the U.S. Navy air tables and was surprised to find that some of them suffered from severe dysbaric osteonecrosis. It began to appear that the U.S. Navy air diving tables were indeed not sacrosanct, and that their use might in fact produce dysbaric osteonecrosis. I had an opportunity to examine some commercial divers who had suffered from Type II, central nervous sytem decompression sickness. They appeared to have lesions affecting diverse parts of both brain and spinal cord.

The papers presented in these sessions mark a distinct advance in the concepts of the physics and pathophysiology of decompression sickness. Dr. Kent Smith's paper on the pathophysiology of decompression sickness was an excellent summary of the present concepts of this disease process as they are understood today. Indeed it is not a disease caused by a single bubble, but one resulting from large numbers of small bubbles which can be detected and grossly quantified by the use of the Doppler ultrasonic flowmeter. The number of bubbles, and by inference, the volume of air dispersed within the body has been related to the probability of the occurrence of decompression sickness and the degree of severity which can be expected. It is now known that bubbles develop during decompression from a "normal" U.S. Navy air dive. These bubbles may produce pathophysiology directly by obstruction of blood flow and indirectly by damage to blood vessel walls, leading to changes in hemocoagulation and ultimately to the production of platelet and red cell thrombi.

The significance of these findings was further amplified by Dr. Winkleman in his paper, "Otoneurologic Findings." The group of physicians which Dr. Winkleman represented studied injured divers post-accident in accordance with the disciplines of their respective specialities, i.e., otolaryngology, ophthalmology, neurology, neuropsychology, radiology, nuclear medicine and internal medicine. These clinicians have demonstrated that the lesions which occur in divers who have developed severe decompression sickness may affect many parts of the body simultaneously, i.e., lesions may occur in several parts of the brain and spinal cord, as reported in the neurological findings. Otolaryngologic studies revealed that the so-called "vestibular hit" may result from a lesion in the peripheral end organ, in the synaptic pathways of the metencephalic nuclei or in the cerebral radiations of those nuclei, either singularly or in mixed combinations.

In addition, the ophthalmologist, Dr. Mike Lieppman, revealed that not only do visual disturbances result from decompression

sickness per se, but he also suggested that the accommodative dysfunction found in 12 of 14 divers was indeed a central lesion and was probably associated with oxygen toxicity rather than decompression sickness. In addition, the neuropsychological studies revealed a degree of cerebral sickness. In addition, the neuropsychological studies revealed a degree of cerebral dysfunction in many divers commonly called "organic brain syndrome" which was undoubtedly related to multiple diffuse lesions of decompression sickness, perhaps similar to the lesions of the punch-drunk boxer, rather than to a single lesion.

Professor Walder reviewed the problem of dysbaric osteonecrosis and related it to inadequate decompression. Not only did he demonstrate lesions which occur in caisson workers, but also showed similar lesions which occurred in divers. It is indeed apparent that the human skeletal system does not differentiate between inadequate decompression used by caisson workers and that used by divers.

The paper presented by Dr. Pegg on decompression sickness during the Aegir dive in Hawaii served to emphasize the limits of our knowledge on decompression sickness prevention at the present time. In these relatively long-term saturation dives, to 500 feet, four of five subjects suffered decompression sickness affecting their joints during the decompression. The only thing that was unique to this dive was the length of time (3 weeks) that subjects remained under pressure. This period was considerably longer than that which was used by the U.S. Navy to develop the saturation diving decompression tables which were utilized by the Aegir medical officers in decompressing their subjects to the surface.

The papers presented by Professor D. Yount, "The Physics of Bubble Formation", and by Professor B. A. Hills, "Zero-Supersaturation Approach to Decompression", demonstrated the progress in the understanding of bubble formation in decompression sickness which has been made possible over the past 10 years as a result of the input of physical scientists and physical chemists. Indeed, the studies of the laws of physics and chemistry which govern the growth of bubbles are those belonging more to the domain of the physical scientists and chemists than to the physician. The concepts developed by Professor Yount and tested by the use of the gelatin model for the first time begin to bring together the ideas related to bubble nuclei and bubble growth as developed by Harvey (1) 25 years ago. These ideas have now been incorporated into a concept for the prevention of bubble growth during decompression.

The use of these concepts in the development of decompression tables was then described by Dr. R. H. Strauss in his paper, "The Physics of Bubble Formation: Implication for Improvement of

Decompression Methods." He demonstrated how the physical principles of bubble formation could be incorporated into a computer model for the calculation of decompression tables. The relevance of these tables was demonstrated by the use of the gelatin model, as well as on a series of dives with human subjects. Similarly, Dr. B. A. Hills' presentation, "Zero-Supersaturation Approach to Decompression," pointed out the difference between the gelatin model and human tissue. Since the living organism utilizes oxygen, there is an inherent unsaturation of gas tension within the living body. Professor Hills has utilized this unsaturation in developing decompression tables for decompressing divers by controlled rate of decompression so that no gas supersaturation can occur. On a theoretical basis this procedure would result in a completely safe decompression table so long as the model for the rate of gas uptake and elimination in the body accurately models the values which exist in the physical systems of the body. These concepts, as usual, all need further validation and human testing, and all clearly merit further evaluation and research; progress along these lines should result in the development of a more accurate physical and physiological model for the prevention of decompression sickness during diving, working in caissons, and flying.

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