

HAWAU-W-88-003

CIRCULATING COPY
Sea Grant Depository

University of Hawaii Sea Grant
College Program

Project: Influence of Pulmonary Gas
Embolism on Cardiopulmonary Functions

Project No: R/HP-5

UNIHI-SEAGRANT- CR - 91 - 01

MAN IN THE SEA

Volume II

Y.C. Lin
K.K. Shida
Editors

A BEST PUBLICATION

**PUBLISHED BY
BEST PUBLISHING COMPANY
POST OFFICE BOX 1978
SAN PEDRO, CALIFORNIA 90732
ALL RIGHTS RESERVED.**

**NO PART OF THIS BOOK MAY BE REPRODUCED, STORED IN A
RETRIEVAL SYSTEM, OR TRANSMITTED IN ANY FORM OR BY ANY
MEANS, ELECTRONIC, MECHANICAL, PHOTOCOPYING, MICRO-
FILMING, RECORDING, OR OTHERWISE, WITHOUT WRITTEN
PERMISSION FROM THE PUBLISHER.**

**COPYRIGHT © 1990, BY BEST PUBLISHING COMPANY
PRINTED AND BOUND IN THE UNITED STATES OF AMERICA**

**INTERNATIONAL STANDARD BOOK NUMBER:
ISBN 0-941332-13-6**

**LIBRARY OF CONGRESS CATALOG CARD NUMBER:
89-81306**

Contents

| | |
|----------------------------------|-------|
| Contents of Volume I | xiii |
| List of Authors and Contributors | xv |
| Preface | xix |
| Acknowledgements | xxi |
| Introduction | xxiii |

PART A. SPORTS DIVING

1

Physiological Responses to Head-Out Water Immersion

John A. Krasney

| | |
|---|----|
| I. Introduction | 1 |
| II. Kidney and Endocrine Adjustments | 5 |
| A. Thermal Conditions | 5 |
| B. Hydration Conditions | 5 |
| C. Duration of Immersion | 7 |
| D. Mechanisms of Diuresis and Natriuresis | 7 |
| III. Hormonal Responses | 15 |
| A. Vasopressin | 15 |
| B. Plasma Renin Activity | 16 |
| C. Atrial Natriuretic Peptide | 17 |
| D. Circadian Dependency | 17 |
| IV. Fluid Shifts | 18 |
| V. Pulmonary Effects | 24 |
| VI. Regional Circulation | 25 |
| VII. Conclusions | 29 |
| Acknowledgements | 29 |
| References | 29 |

2 Physiological Limitations of Humans as Breath-Hold Divers

Y.C. Lin

| | |
|--|----|
| I. Introduction | 33 |
| II. Diving Time | 35 |
| A. Predicted Breath-Hold Time | 36 |
| B. Observed Breath-Hold Time | 36 |
| C. Breath-Hold Breaking Points | 38 |
| D. Breath-Hold Time of Professional Divers | 39 |
| III. Diving Depth | 40 |
| A. Predicted Diving Depth | 40 |
| B. Diving Depth of Professional Breath-Hold Divers | 41 |
| C. Breath-Hold Diving Depth Records | 41 |
| IV. Hypothermia | 42 |
| V. Decompression Sickness | 44 |
| VI. Drowning and Near Drowning | 45 |
| A. Shallow Water Blackout and Drowning | 45 |
| B. Near Drowning | 45 |
| VII. Cardiac Arrhythmia and Diving Bradycardia | 47 |
| A. Head-Out Immersion | 47 |
| B. Submersion | 48 |
| VIII. Summary | 50 |
| Acknowledgements | 52 |
| References | 52 |

3 Arterial Gas Tensions and Hemoglobin Concentrations of the Free Diving Antarctic Weddell Seal

Warren M. Zapol, Roger D. Hill, Jesper Qvist, Konrad Falke, Robert C. Schneider, Graham C. Liggins, and Peter W. Hochachka

| | |
|---------------------------------|----|
| I. Introduction | 57 |
| II. Techniques | 59 |
| A. Flush Solution | 60 |
| B. Blood Sampling System | 60 |
| C. Nitrogen Tension Measurement | 61 |
| D. Hemoglobin and Hematocrit | 61 |
| III. Results | 62 |
| A. Arterial Hemoglobin | 62 |
| B. Arterial Oxygen Tensions | 63 |
| C. Arterial Nitrogen Tensions | 64 |

| | |
|----------------------------|----|
| IV. Discussion | 65 |
| A. Nitrogen | 65 |
| B. Hemoglobin | 67 |
| Acknowledgements | 70 |
| References | 70 |

4 **Energetics of Breath-Hold Diving in Korean and Japanese Professional Divers**

Y. S. Park, K. Shiraki, and S.K. Hong

| | |
|--|----|
| I. Introduction | 73 |
| II. Swimming Suit Divers | 75 |
| A. Heat Exchange During Diving Work | 75 |
| B. Limited Diving Time | 76 |
| III. Wet-Suit Divers | 77 |
| A. Heat Exchange During Diving Work | 77 |
| B. Thermal Insulation During Diving Work | 82 |
| IV. Summary | 85 |
| Acknowledgements | 86 |
| References | 86 |

5 **Safety of Sports Diving: Comparison of Novice and Expert Divers**

Y. Mano, M. Shibayama, T. Mizuno and J. Ohkubo

| | |
|--|-----|
| I. Introduction | 87 |
| II. Fatal Diving Accidents | 88 |
| III. Non-Fatal Diving Accidents | 89 |
| IV. Inner Ear Barotrauma | 94 |
| V. Comparison of Diving Profiles Between Novice and Expert Divers | 95 |
| A. Characteristics of the Diving Recorder | 95 |
| B. Subjects | 95 |
| C. Procedures | 96 |
| D. Results | 96 |
| VI. Discussion | 100 |
| VII. Conclusion | 102 |
| References | 103 |

PART B. HYPERBARIC MEDICINE

6 Hyperbaric Medicine During Spaceflight

Joey B. Boyce and Jeffrey R. Davis

| | |
|--|-----|
| I. Introduction | 105 |
| II. Spaceflight Physiology | 107 |
| A. Space Motion Sickness | 107 |
| B. Body Fluid Shift | 107 |
| III. Medical Kit | 107 |
| IV. EVA System | 108 |
| V. Decompression Sickness | 108 |
| A. Relative Risks | 110 |
| B. Altitude DCS | 110 |
| C. Treatment | 112 |
| VI. Escape System | 112 |
| VII. Manned Maneuvering Unit | 112 |
| VIII. Space Station Freedom | 114 |
| A. Health Maintenance Facility | 114 |
| B. Hyperbaric Airlock | 115 |
| IX. The Future | 115 |

7 Hyperbaric Oxygen in a Modern Hospital Practice

*Mahito Kawashima, Hiroaki Tamura, Katsuhiko Takao
and Teruhiko Tsunosue*

| | |
|---|-----|
| I. Introduction | 117 |
| II. Method of HBO Administration | 118 |
| A. The Chamber | 118 |
| B. Patients | 119 |
| III. Results | 122 |
| A. General | 122 |
| B. Osteomyelitis | 123 |
| C. Chronic Obliterative Vascular Diseases | 126 |
| D. Necrotizing Fasciitis | 128 |
| IV. Discussion and Conclusion | 128 |
| References | 129 |

8 **Medical Advances in Compressed Air Construction Work**
 Eric P. Kindwall

| | |
|--|-----|
| I. Introduction | 131 |
| II. Decompression Procedures | 132 |
| III. Comparison of Tables | 137 |
| IV. Clinical Evaluation | 144 |
| V. Summary | 147 |
| Acknowledgements | 147 |
| References | 147 |

9 **Diving Accident Management in Remote Areas**
 — Current Concepts and Controversies
 Carl Edmonds

| | |
|---|-----|
| I. Introduction | 149 |
| II. Arterial Gas Embolism (AGE) | 150 |
| III. Pulmonary Barotrauma — Other Than Air Embolism | 151 |
| IV. Decompression Sickness (DCS) | 152 |
| V. Drowning | 153 |
| VI. Hypothermia | 153 |
| VII. Inner Ear Barotrauma | 153 |
| VIII. Infections | 155 |
| IX. Marine Animal Injuries | 156 |
| A. Neuromuscular Venom | 156 |
| B. Fish Spine Injuries | 156 |
| C. Jellyfish | 156 |
| X. General | 157 |
| Appendix | 157 |
| A. Oxygen Administration Equipment | 157 |
| B. Underwater Oxygen Recompression | 158 |
| C. Positioning, Moving and Transport of the Patient | 159 |
| D. Aviation Medivac | 160 |

10 Changes Found in the Central Nervous System from Autopsies on Divers

Ian M. Calder

| | |
|--|-----|
| I. Introduction | 163 |
| II. Neuropathology of Decompression Sickness (DCS) . . . | 164 |
| III. Morphological Observations | 165 |
| A. Acute Changes | 167 |
| B. Sub-Acute Changes | 167 |
| C. Chronic Changes | 168 |
| IV. The Brain and DCS | 173 |
| V. DCS and the Inner Ear | 174 |
| VI. Discussion | 175 |
| VII. Conclusion | 177 |
| Acknowledgements | 178 |
| References | 178 |

PART C. MANNED SUBMERSIBLES

11 The Alvin Program

Barrie B. Walden

| | |
|--|-----|
| I. Introduction | 181 |
| II. History | 182 |
| A. The Early Years | 182 |
| B. Support Ship R/V Lulu | 182 |
| III. Accomplishments | 183 |
| A. Recovery of a Hydrogen Bomb | 183 |
| B. Research Applications | 183 |
| C. International Cooperation: The Mid-Atlantic Ridge . . | 184 |
| D. Increasing Capabilities | 185 |
| IV. Flagship R/V Atlantis II | 186 |
| V. The Future | 189 |

**12 Biological Explorations in the Mid-Ocean Realm:
Food Webs, Particle Flux and Technological
Advancements**

*Marsh J. Youngbluth, Thomas G. Bailey and
Charles A. Jacoby*

| | |
|--|-----|
| I. Introduction | 191 |
| II. Species Diversity Discovery | 193 |
| III. Vertical Distribution Patterns | 193 |
| IV. Particle Flux | 194 |
| A. Fecal Pellet Transport and Zooplankton Behavior . . | 195 |
| B. Marine Snow Aggregates and Gelatinous Plankton . | 196 |
| V. Metabolism of Pelagic Fauna | 198 |
| VI. Submersible as a Mobile Laboratory | 198 |
| VII. Some Recommendations | 201 |
| VIII. Facilities, Programs, and Projects | 202 |
| IX. Summary and Conclusions | 203 |
| Acknowledgements | 204 |
| References | 204 |

13 U.S. Navy Deep Submergence — 88

Michael R. Popovich

| | |
|-------------------------------------|-----|
| I. Introduction | 209 |
| II. History | 210 |
| III. Mission Profile | 211 |
| IV. Equipment | 211 |
| A. Electrical | 211 |
| B. Hydraulics | 212 |
| C. Sonars | 212 |
| D. Photographic Equipment | 212 |
| V. Interior | 213 |
| VI. Exterior | 213 |
| VII. Data Interfaces | 213 |
| VIII. Recovery | 214 |
| IX. Operations in 1988 | 214 |
| X. Summary and Conclusion | 217 |

EPILOGUE

| | | |
|-----------|-------------------------------------|------------|
| 14 | The Future of Man in the Sea | |
| | <i>John P. Craven</i> | 219 |
| | Author Index | 225 |
| | Subject Index | 227 |

Contents of Volume I

PART. A SATURATION DIVING — COMPRESSION

- 1 The Contribution of Animal Experimentation Toward the Development of Rational Compression Schedules for Very Deep Diving**
Ralph W. Brauer
- 2 The Compression Strategy in the Alverstoke Deep Dives Series**
Z. Török
- 3 Development and Evaluation of Compression Procedures for Deep Operational Diving**
Russell E. Peterson, Ragnar J. Vaernes and C.J. Lambertsen
- 4 The French Deep Diving Scientific Program on Oxygen-Helium, Trimix and Oxygen-Hydrogen Gas Mixtures**
B. Gardette, C. Lemaire, J.C. Rostain and X. Fructus
- 5 The Value of Trimix 5 to Control HPNS**
P.B. Bennett and H. Schafstall

PART B. SATURATION DIVING — PRESSURE ADAPTATION

- 6 Natriuresis and Hypoxic Signs at Pressure — Cellular Considerations**
James M. Goldinger, Michael E. Duffey, Daniel J. Wilkinson and Suk Ki Hong

- 7 **U.S.-Japan Cooperative Diving Research (1973-1985)**
S.K. Hong
- 8 **Thermal Regulation in Dry and Wet
Hyperbaric Environments**
Sueko Sagawa and Keizo Shiraki
- 9 **Saturation Diving in the National Oceanic and
Atmospheric Administration Office of
Undersea Research**
William S. Busch
- 10 **Nitrox and Heliox Saturation Diving in China**
Zhong Yuan Shi

PART C. SATURATION DIVING — DECOMPRESSION

- 11 **Confidence in Decompression Safety**
P.K. Weathersby
- 12 **The Physics of Bubble Formation: Implications
for Improvement of Decompression Tables**
David E. Yount
- 13 **The Role of Complement Activation in
Decompression Sickness**
*C.A. Ward, D. McCullough, D. Yee, D. Stanga
and W.D. Fraser*
- 14 **Animal Models in Decompression**
Edward H. Lanphier and Charles E. Lehner
- 15 **Doppler Evaluation of Decompression Tables**
R.Y. Nishi

EPILOGUE

- 16 **The Future of Manned Diving**
David H. Elliott

List of Authors and Contributors

- Thomas G. Bailey**, Department of Marine Sciences, Harbor Branch Oceanographic Institution, 5600 Old Dixie Highway, Fort Pierce, FL 34946
- Peter Bennett**, F.G. Hall Environmental Laboratory, Box 3823, Duke University Medical Center, Durham, NC 27710
- Joey B. Boyce**, Flight Medicine Clinic SD2, NASA — Johnson Space Center, Houston, TX 77058
- Ralph W. Brauer**, Program in Science and Culture, University of North Carolina at Wilmington, Wilmington, NC 28406
- William S. Busch**, NOAA Office of Undersea Research, 6010 Executive Blvd., Rockville, MD 20852
- Ian M. Calder**, Institute of Pathology, London Hospital Medical College, London, E1, 1BB, England, U.K.
- John P. Craven**, Law of the Sea Institute, University of Hawaii, Honolulu, HI 96822
- Jeffrey R. Davis**, Flight Medicine Clinic SD2, NASA — Johnson Space Center, Houston, TX 77058
- Michael E. Duffey**, Department of Physiology, State University of New York at Buffalo, School of Medicine and Dentistry, Buffalo, NY 14214
- Carl Edmonds**, 25 Battle Blvd., Seaforth, N.S.W. 2092, AUSTRALIA
- David H. Elliott**, Robens Institute of Health and Safety, 40 Petworth Road, Haslemere, Surrey GU27 2HX, England, U.K.
- Konrad J. Falke**, Freie University, Department of Anesthesiologie, Berlin D1000 Berlin 19, WEST GERMANY
- W.D. Fraser**, Defence and Civil Institute of Environmental Medicine, 1133 Sheppard Ave. W., P.O. Box 2000, Downsview, Ont. M3M 3B9 CANADA
- Xavier Fructus**, COMEX SA, 36 Bd. des Oceans, 13275 Marseille, Cedex 9, FRANCE
- Bernard Gardette**, COMEX SA, 36 Bd. des Oceans, 13275 Marseille, Cedex 9, FRANCE
- James M. Goldinger**, Department of Physiology, State University of New York at Buffalo, School of Medicine and Dentistry, Buffalo, NY 14214

1

Physiological Responses to Head-Out Water Immersion

John A. Krasney

| | |
|---|----|
| I. Introduction | 1 |
| II. Kidney and Endocrine Adjustments | 5 |
| A. Thermal Conditions | 5 |
| B. Hydration Conditions | 5 |
| C. Duration of Immersion | 7 |
| D. Mechanisms of Diuresis and Natriuresis | 7 |
| III. Hormonal Responses | 15 |
| A. Vasopressin | 15 |
| B. Plasma Renin Activity | 16 |
| C. Atrial Natriuretic Peptide | 17 |
| D. Circadian Dependency | 17 |
| IV. Fluid Shifts | 18 |
| V. Pulmonary Effects | 24 |
| VI. Regional Circulation | 25 |
| VII. Conclusions | 29 |
| Acknowledgements | 29 |
| References | 29 |

I. INTRODUCTION

In recent years, there has been a renewed interest in the physiological aspects of head-out water immersion (WI). WI represents a simple, non-invasive maneuver which is a very useful tool for investigating mechanisms that are involved in the regulation of blood volume. WI has been used as a therapeutic modality for centuries and it is probably correct to say that we are only just beginning to understand the nature of the profound physiological alterations which can be elicited by simply sitting in

a warm bath. WI is clinically important as a major potential therapeutic maneuver for clinical fluid retention disorders such as cirrhosis, nephrosis, and possibly even congestive heart failure (Epstein, 1978; Gauer and Henry, 1976; Gilmore, 1983; Lin, 1984; Norsk and Epstein, 1988).

In addition, in view of the fact that a significant portion of the population now engages in WI through the use of hot tubs and Jacuzzi baths for recreational purposes, it is important to understand the general health implications of long-term immersions in warm water.

As a consequence of the buoyancy effect imparted by water, WI has been considered to be a useful model for understanding the effects of hypogravity or the weightless state. Thus, WI has also become an important tool for the study of space physiology (Blomqvist and Stone, 1983; Gauer and Henry, 1976; Hargens, 1987). Furthermore, an analysis of the physiology of WI is of crucial importance in order to obtain a complete understanding of the physiology of diving and man in the sea.

Although earlier investigators had suggested that WI should lead to a displacement of blood away from the dependent regions of the body into the thorax, Gauer and Henry (1976) were the first to provide convincing evidence that cardiac volume increases during this maneuver. In fact, Gauer and Henry considered WI under thermoneutral conditions to be the investigative tool of choice for studying the effects of plasma volume expansion (Gauer and Henry, 1976). This view is based on the simple, non-invasive nature of WI and the highly reproducible nature of the response.

The "Gauer-Henry Hypothesis" states that WI leads to a central translocation of blood which expands intrathoracic structures. The distention of these intrathoracic structures in turn leads to activation of mechanoreceptors which bring about reflex hormonal and neural adjustments which cause an increase in urine flow and an increase of renal sodium excretion. Gauer and Henry were the first to provide evidence that the diuretic response of the kidney to central volume expansion could be abolished by cutting the vagus nerves in the neck in anesthetized dogs (Gauer and Henry, 1976; Gilmore, 1983).

The original view advocated by Gauer and Henry (1976), as well as other investigators, is that the increase in salt and fluid output by the kidneys ultimately leads to a true correction of the total plasma volume with a decline of the intrathoracic volume back to the pre-immersion levels. Recent evidence indicates that the latter view is to some extent correct, however it must be modified to take into account the fact that a major fluid shift occurs during WI which acts to elevate the plasma volume (Fig. 1). Hence, the primary role of the kidney in the water immersion response is to minimize the increase in plasma volume which

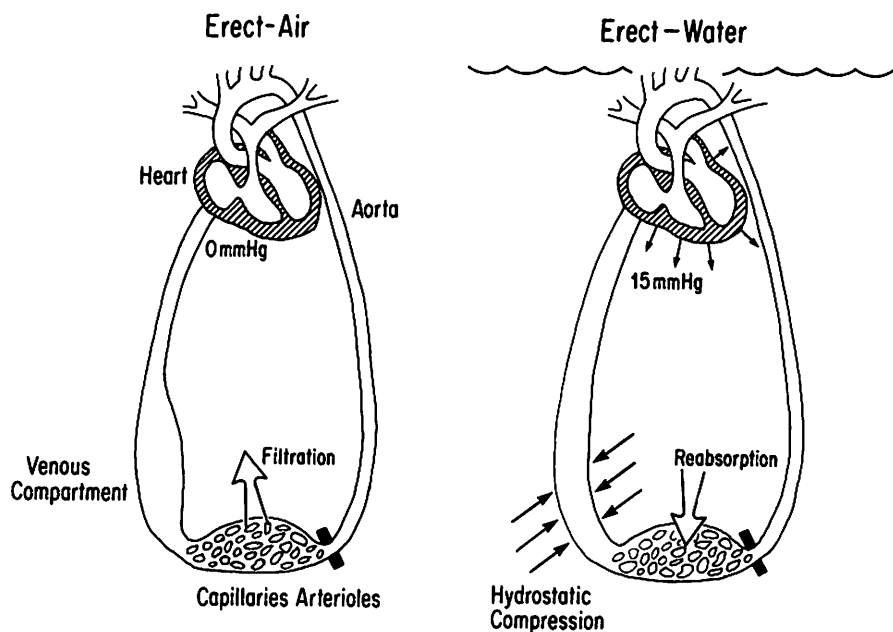


FIG. 1. In the human standing in air, there is engorgement of the dependent veins along with a relative filtration of fluid out of the dependent capillaries. In addition, the cardiac volume is small (left panel). During thermoneutral water immersion in the erect position to the level of the neck, there is hydrostatic compression of the venous compartment which diminishes venous capacitance and shifts blood into the chest. The latter effect raises central venous pressure and increases cardiac volume. In addition, fluid is reabsorbed from the tissues into the capillaries to elevate the blood volume.

would be otherwise quite massive in the absence of a diuresis and natriuresis (Miki *et al.*, 1986a; Miki *et al.*, 1987a).

The current concept is that central volume expansion occurs in WI both because of a primary increase of the venous return owing to extravascular compression of veins by the hydrostatic pressure and because of a fluid shift which increases the plasma volume (Fig. 1). The ensuing increases of atrial and ventricular end-diastolic pressures lead to an increase of the cardiac stroke volume and the cardiac output by engaging the Frank-Starling length-tension mechanism. The elevation of the cardiac output can be sustained for some time (Arborelius *et al.*, 1972; Gauer and Henry, 1976; Lin, 1984). If the WI is carried out under thermoneutral conditions, then by definition, the systemic O_2 consumption does not change. Therefore, an unusual situation quickly develops during WI whereby the systemic blood flow and O_2 delivery exceed the apparent O_2

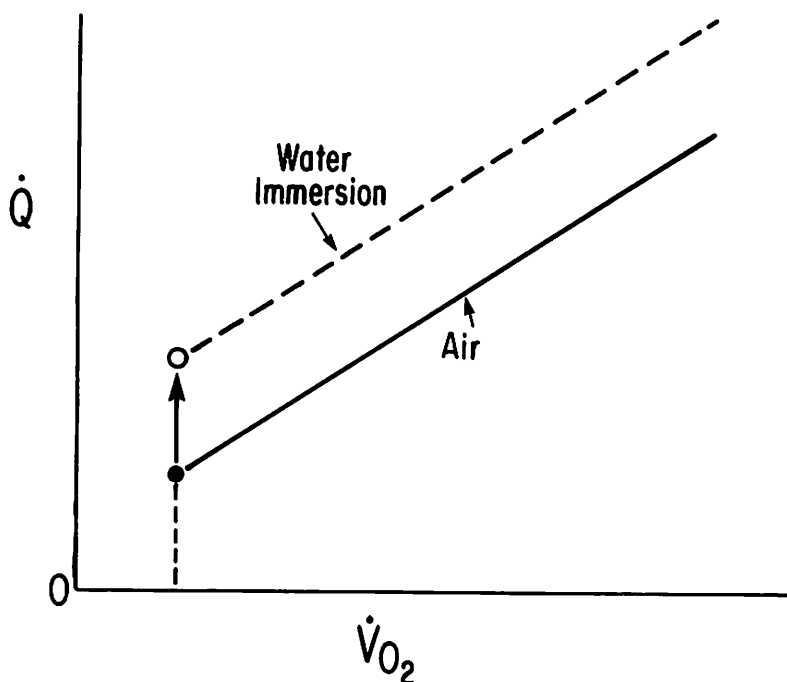


FIG. 2. During WI under resting conditions, (arrow) systemic oxygen consumption (\dot{V}_{O_2}) remains constant, while cardiac output (\dot{Q}) is elevated. Therefore, the \dot{Q}/\dot{V}_{O_2} relation is elevated at rest. Presumably, this relation would remain elevated (dashed line) relative to the air condition (solid line) during exercise as \dot{V}_{O_2} increases. Thus, a systemic "luxury perfusion" occurs in WI.

requirements of the tissues (Fig. 2). A basic principle of blood flow regulation is that flow is rather precisely regulated in accord with the tissue metabolic demands. This is generally referred to as autoregulation of blood flow (Hajduczuk *et al.*, 1987b). Thus in thermoneutral WI, the autoregulation relationship appears to be modified substantially. The reasons for the persistence of this "luxury perfusion" are not entirely clear at the present time but these issues are important relative to the application of WI for therapeutic purposes (Hajduczuk *et al.*, 1987b). Studies of the distribution of the elevated cardiac output during WI have proved to be quite interesting as will be developed in a subsequent section.

In addition to human studies, investigations of the physiological responses to head-out WI have been carried out in a variety of other species.

These include the monkey, rat, sheep, pig, and the dog (Peterson *et al.*, 1980; Peterson *et al.*, 1987). Systematic investigations have been confined to the dog and the monkey (Krasney *et al.*, 1984; Peterson *et al.*, 1987). Over the past several years, our laboratory has focussed specifically on the canine model of WI as well as on human studies and this presentation will emphasize the results obtained from studies of these two species. Animal models of WI permit more extensive studies than can be carried out in human subjects.

II. KIDNEY AND ENDOCRINE ADJUSTMENTS

A. Thermal Conditions

In conducting studies of WI, it is important to define the temperature of the water bath because of the thermoneutrality issue. The thermoneutral temperature for the animal occurs where neither core temperature nor oxygen consumption changes. In the human, this temperature is generally reported to be about 34 or 35°C (Arborelius *et al.*, 1972; Epstein, 1978; Gauer and Henry, 1976). However, this has not proven to be the thermoneutral temperature for other mammalian species. For example, the thermoneutral temperature for the conscious trained dog is about 37°C. If awake dogs undergo WI at 35°C, they will generally begin shivering within about 20 minutes. Experimental conditions can influence the thermoneutral temperature also. For example, the thermoneutral temperature of the anesthetized dog is about one degree warmer, e.g. 38°C (Krasney *et al.*, 1982). Deviations from thermoneutrality on either the warm side or cold side can lead to profound alterations of the physiological response patterns. The studies described herein will generally refer to thermoneutral WI unless indicated otherwise.

B. Hydration Conditions

Hydration conditions can also have important influences upon the physiologic response pattern to WI, both in quantitative as well as qualitative terms. WI experiments can be performed where the fluid losses in urine and from blood sampling are replaced during the study. These are referred to as volume repletion experiments. If fluid losses are not replaced, then the study is referred to as a volume depletion experiment. In addition, there may be hydration protocols carried out prior to the experiment. For example, subjects may be asked to ingest a bolus of water prior to WI with no additional fluid given during the study (Claybaugh *et al.*,

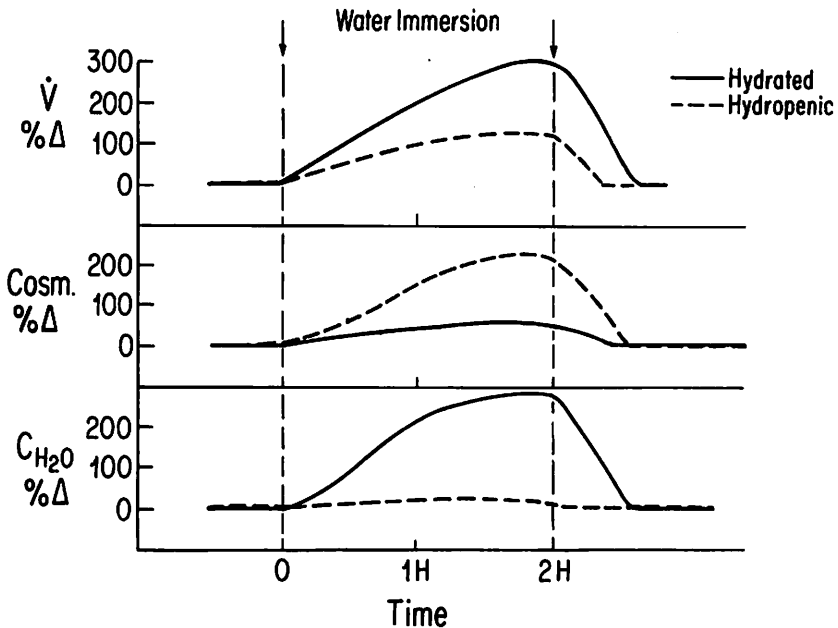


FIG. 3. Renal response of hydrated vs hydropenic humans to WI. Data represent percent changes from pre-immersion levels (%Δ). \dot{V} , urine flow; C_{osc} , osmolar clearance; C_{H_2O} , free water clearance. Data are redrawn from Behn *et al.* (1969).

1986). In our dogs, we generally prehydrate the animal with 2% of the body weight (about 200 ml) of 0.45% NaCl solution given intravenously over a 20 min period before WI. This is done to increase the basal urine flow in order to have enough urine volume to perform renal clearance measurements (Hajduczkow *et al.*, 1987a).

Our data indicate that, over a 100 min period of WI, conscious dogs show a diuresis under both replete and deplete conditions. In both situations, the diuresis consists of an increase of sodium excretion and an increase of free water clearance. There are, however, significant differences, in that both the free water and sodium excretion responses, as well as the net diuresis response of the dog are much less when volume depletion is allowed to occur (Sondeen *et al.*, 1988).

The response of hydrated humans differs significantly from that of dehydrated or hydropenic subjects. In hydrated humans the diuresis consists of a prominent free water component with a smaller increase of

sodium excretion and osmolal clearance. By contrast, in hydropenic humans, the diuresis is smaller in magnitude and consists mainly of an increase of sodium excretion and osmolal clearance, with no increment of free water clearance (Fig. 3).

C. Duration of Immersion

The duration of the WI period is also of importance because it takes time for the renal responses to develop. However, one of the most impressive observations is that the diuresis and natriuresis are usually sustained for several hours if WI periods of 3-6 hours are used (Claybaugh *et al.*, 1986). Thus, the magnitude of the total excreted urine volume can be observed to be many times greater than that which could simply be derived from the fluid volume usually contained in the plasma compartment alone. Since hematocrit measurements indicate that plasma volume may be normal or elevated after several hours of WI, despite the loss of a large volume of fluid, the source of the excreted fluid must be from outside the plasma compartment (Miki *et al.*, 1987a).

D. Mechanisms of Diuresis and Natriuresis

The mechanisms which initiate and sustain the diuresis and natriuresis are at least partly related to the central volume expansion. There are many mechanoreceptors located within various parts of the heart and the great vessels which supply the nervous system with information as to the dimensions of cardiac and vascular structures by responding to stretch.

1. Cardiopulmonary Receptors

The *Mechanoreceptors* located within the heart have their afferents located in the vagus and sympathetic nerves and are referred to as cardiac receptors, or cardiopulmonary receptors (Bishop *et al.*, 1983; Gilmore, 1983). The most well-studied of these receptor types are located in the atria and their afferents travel in myelinated fibers in the vagi (Fig. 4). These receptors have their peak discharge during the cardiac cycle during the "v" wave of the atrial pressure pulse and are termed the "Type B" receptors. These receptors have been considered to be the classical "volume" receptors although there are many additional receptor types which could play a role in volume regulation. Stimulation of type B afferents by inflating a balloon in the left atrium typically leads to a reflex depression of vasopressin and renin secretion, along with a depression of

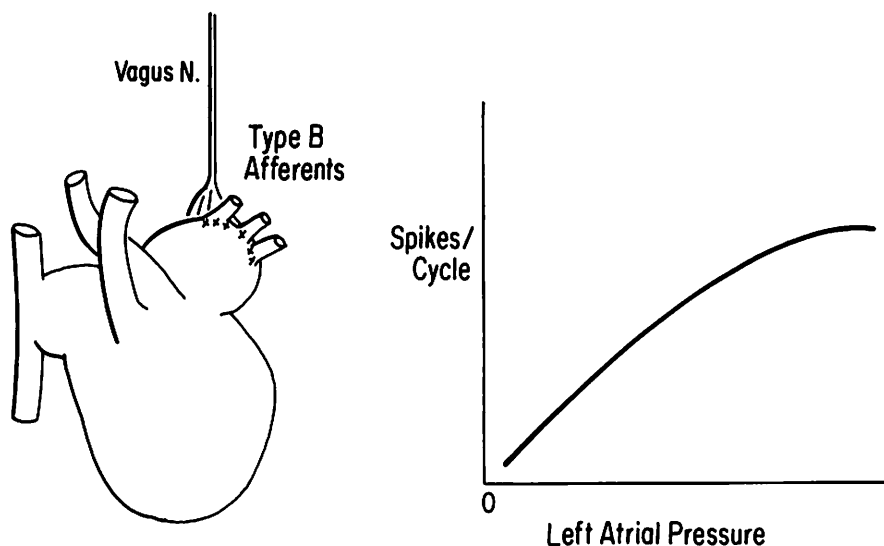


FIG. 4. Type B mechanoreceptor myelinated afferents have their receptors located mainly near the pulmonary vein-left atrial junction. The afferent fibers travel to the medulla (nucleus tractus solitarius) via the vagus nerves. The activity of these afferents increases as atrial pressure is elevated progressively.

renal sympathetic nerve activity. Since the latter responses have been reported to occur during volume expansion and WI, the type B receptors have been considered to play a pivotal role in the response to central distention.

However, receptors having non-myelinated or C fiber afferents, which carry cardiac mechanoreceptor information, comprise the largest cardiac afferent input (Bishop *et al.*, 1983). The role of these receptors remains uncertain because, although stimulation of vagal C fiber afferents can inhibit vasopressin and renin secretion as well as renal sympathetic activity, this input also elicits profound bradycardia, hypotension, and apnea. The latter responses are certainly not characteristic of the WI response (Bishop *et al.*, 1983; Epstein, 1978).

2. Arterial Baroreceptors

Other Receptors which are concerned with the regulation of arterial blood pressure are located in the carotid sinus and aortic arch and are

termed the high-pressure arterial baroreceptors. Mechanical stretch of these regions not only lowers arterial pressure and heart rate, but also depresses vasopressin and renin secretion, as well as renal sympathetic activity (Gauer and Henry, 1976; Gilmore, 1983; Thames, 1978; Thames *et al.*, 1982).

During WI, there is a rise of cardiac filling pressures. The cardiac output is increased as well as arterial pressure. Apparently, there is a shift of blood from the venous compartment into the arterial compartment because the rise in arterial pressure is entirely related to the increase of cardiac output. If anything, total peripheral resistance tends to decline (Arborelius, 1972; Lin, 1984). The heart rate response varies among species. In the human, the heart rate tends to slow or remain the same (Lin, 1984). Whereas, in the dog, the heart rate tends to increase or remain the same (Hadjuczok *et al.*, 1987a; Yoshino *et al.*, 1988). While cardiac stroke volume tends to increase, the magnitude of the increase depends upon whether the heart rate changes. In any case, the magnitude of the rise in cardiac output, whether achieved via an increase of stroke volume, or heart rate, is similar in humans and dogs, about 25-35%. Moreover, there is a tendency for arterial pulse pressure to rise and this is a function of how much the stroke volume increases (Hajduczuk *et al.*, 1987a).

3. Cardiac Nerves and Cardiac Denervation

These stereotyped cardiovascular responses probably engage cardiac receptors as well as arterial baroreceptors. However, the relative importance of cardiac receptors as opposed to arterial baroreceptors in eliciting the reflex adjustments remains to be determined. One view is that the arterial baroreceptors have become more important in volume regulation in upright species such as monkeys and humans, while the cardiac receptor input is more important in the more horizontal or quadruped species, such as the dog. This issue has not been resolved. Unfortunately, specific denervation studies cannot be performed in humans and experiments of this type are limited to subhuman species. In addition, the response of the conscious monkey to volume expansion and WI differs dramatically from the response of the anesthetized monkey to these experimental maneuvers and the conclusions about species differences may have been based on misleading information obtained from anesthetized monkey studies (Peterson *et al.*, 1987).

Studies of conscious dogs in our laboratory subjected to chronic extrinsic denervation of the heart have revealed that cardiac nerves make an important contribution to the renal functional response pattern during WI (Hadjuczok *et al.*, 1987a). Cardiac denervation was carried out

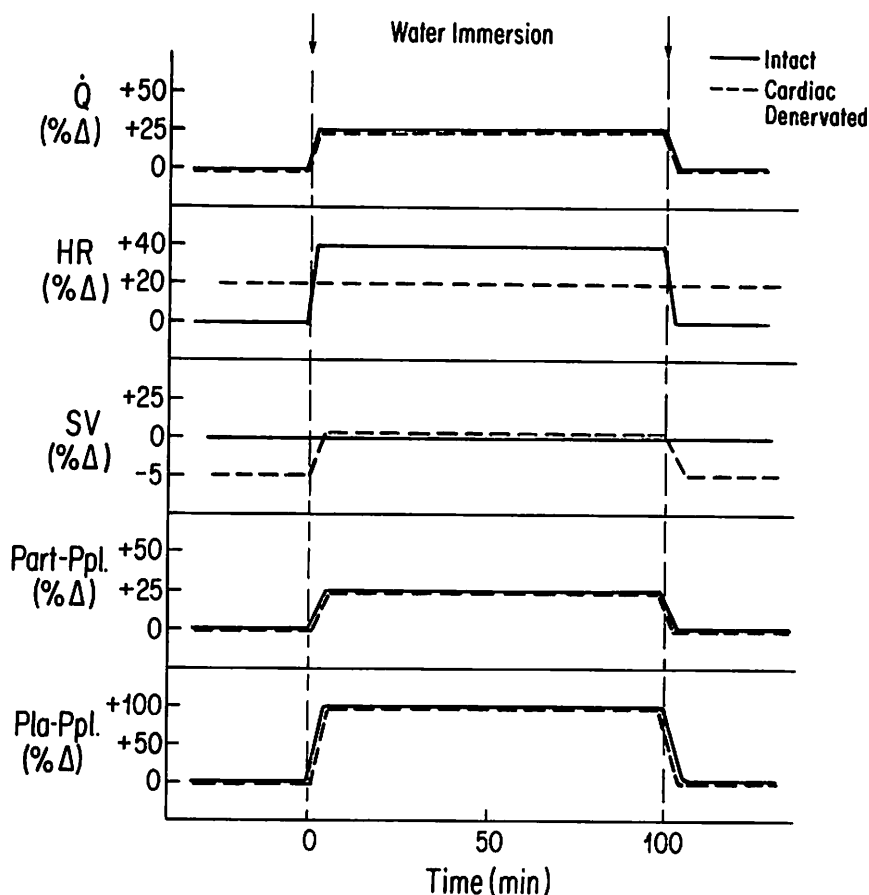


FIG. 5. Cardiovascular responses of intact vs cardiac-denervated trained conscious dogs to WI. Data represent percent change from pre-WI levels. \dot{Q} , cardiac output; HR, heart rate; SV, stroke volume; Part-Ppl, arterial pressure minus pleural pressure; Pla-Ppl, left atrial pressure minus pleural pressure. Note that the rise in cardiac output is similar in both intact and cardiac-denervated dogs; the elevation is achieved by different mechanisms. Total peripheral resistance is unchanged in either situation. Data are redrawn from Hajduczek *et al.* (1987a).

using the one-stage intrapericardial approach of Randall *et al.* (1980). Denervation was verified by both the abolition of cardiac responses to vagal and sympathetic nerve stimulation and by absence of cardiac responses to left atrial injections of tyramine in the chronic state. In addition, interruption of certain cardiac afferents was verified by inflating a balloon which had been implanted chronically in the left atrium. In the

sham-operated dogs, this procedure led to an acceleration of the heart rate and a diuresis and natriuresis. These responses, which have been attributed to activation of atrial type B afferents, were abolished in the cardiac-denervated dogs (CD).

The sham (S) and CD dogs showed increases of atrial, left ventricular, and aortic transmural distending pressures immediately upon WI at 37°C (Fig. 5). In both groups of dogs, the cardiac output increased about 25% immediately upon WI and the response was sustained for the entire 100 min WI period. However, the mechanism whereby the cardiac output increased differed between the two groups of dogs. In the sham dogs, the heart rate increased and stroke volume was constant. In the CD dogs, the heart rate was constant whereas the stroke volume increased (Hadjuczok *et al.*, 1987a). The rise in heart rate in the sham dogs represents a manifestation of the Bainbridge response (Yoshino *et al.*, 1988) which is abolished by CD. Apparently, the curtailment of cardiac filling time in the intact dog is enough to offset the tendency for stroke volume to increase. On the other hand, the primary tendency for stroke volume to increase consequent to the increase of transmural filling pressures is observed following CD. The arterial pressure increases to a similar extent whether or not the heart is denervated. However, the arterial pulse pressure was observed to increase only after CD.

It is interesting that the heart rate does not increase in the human during WI, in fact the heart rate tends to slow (Rowell, 1986). Therefore, the mechanism whereby cardiac output is elevated in WI is via an increase in stroke volume. In the dog, since the heart rate is elevated at the same time the arterial pressure is elevated, the arterial baroreceptors are reset (Yoshino *et al.*, 1988). The reasons for the absence of the Bainbridge phenomenon during WI in the human are unclear. In the monkey, arterial pressure increases during WI while heart rate is unchanged. Therefore, some resetting of the arterial baroreceptors occurs in the sub-human primate as well (Peterson *et al.*, 1987; Yoshino *et al.*, 1988). Whether this change in baroreceptor responsiveness represents a continuum of adaptations occurring from the quadruped to the biped is unclear at present. However, another quadruped, the pig, shows no cardiac acceleration when being immersed (personal observations).

In the sham operated dogs, studied during conditions of volume repletion, WI elicits a diuresis and an increase of osmolal clearance (C_{osm}) which is detectable 20 minutes after entry into the water (Fig. 6). The increase of C_{osm} is due mainly to an increase of sodium excretion ($U_{Na}V$) as potassium excretion usually doesn't change very much. The increases of urine flow and $U_{Na}V$ are on the order of several hundred percent and they may persist for several hours. However, the sham dogs usually show

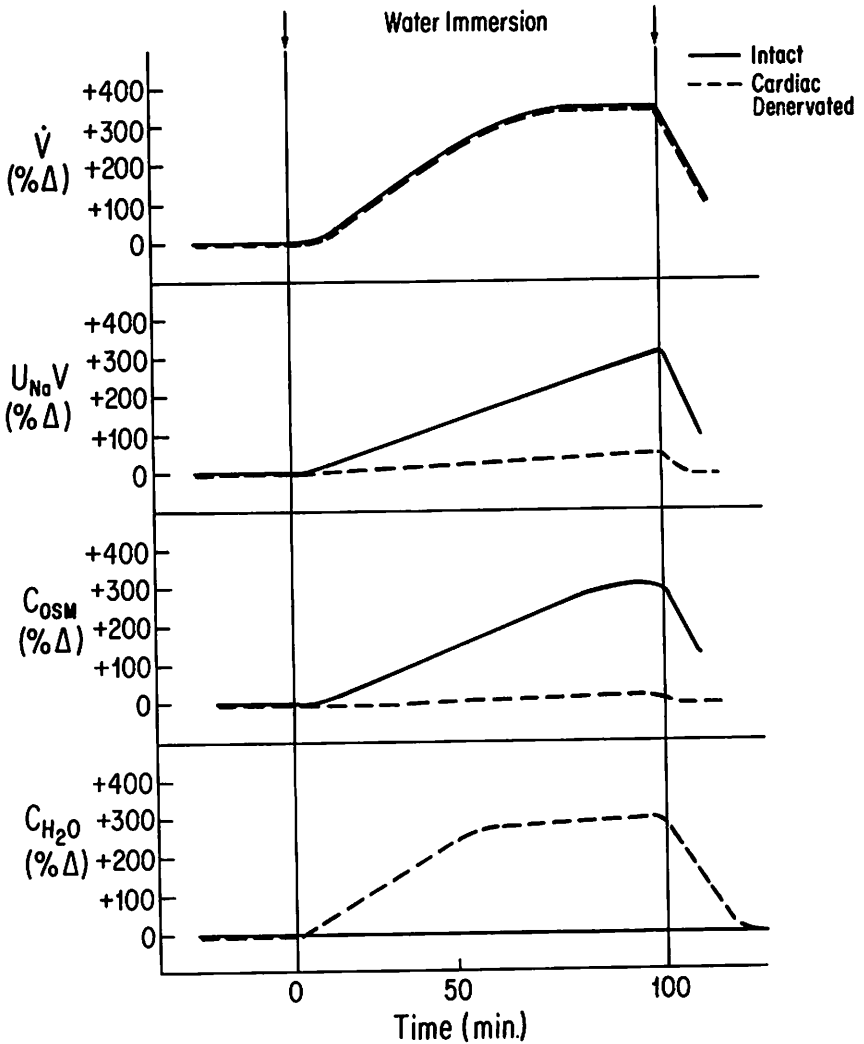


FIG. 6. Renal responses of intact and cardiac-denervated trained conscious dogs to WI. Data represent percent changes from pre-WI levels. Abbreviations similar to Fig. 3. $U_{Na}V$, sodium excretion. Note that the urine flow responses are identical in intact vs denervated dogs, and that the mechanisms for the increased urine flow differ in the two situations. Data are redrawn from Hadjuczok *et al.* (1987a).

small increments of free water clearance (C_{H_2O}). By contrast, the CD dogs show no change of sodium excretion, while the diuresis remains similar in both time course and magnitude. Therefore, the diuresis is achieved principally by a major increase of C_{H_2O} which is observed to occur only following chronic CD. Thus, the cardiac nerves make a primary contribution to the natriuresis observed in WI. After CD, other redundant mechanisms are unmasked which lead to a similar increase of urine flow.

The fact that it is possible to superimpose the diuretic responses of the kidney following WI both before and after CD is remarkable since the diureses are mediated by different mechanisms. Yet the same net fluid volume is lost.

Since there are only quantitative and not qualitative differences in the urine flow and sodium excretion responses in volume repleted dogs compared to volume depleted dogs, it is expected that similar response patterns should occur if these CD experiments are carried out during volume depletion.

4. Renal Sympathetic Nerves

In consideration of other mechanisms, it might be thought that since the arterial pressure tends to rise in WI, the renal functional response might simply be a consequence of a pressure diuresis or natriuresis (Hadjuczok *et al.*, 1987a). However, the CD experiments clearly indicate that there may be drastic alterations of the character of the diuresis after elimination of the cardiac nerves, yet the elevations of arterial pressure were of similar magnitude in both the sham and the CD dogs (Hadjuczok *et al.*, 1987a). Moreover, the glomerular filtration rate is unchanged in WI indicating that the renal response is independent of hemodynamic adjustments.

By exclusion then, there is one major mechanism which could contribute potentially in a major way to the natriuretic response to WI. Atrial distention has been demonstrated to elicit striking reflex adjustments of renal sympathetic nerve activity (RSNA) (Dibona, 1982). In addition, the studies of Dibona and others (Dibona, 1982; Dibona, 1985; Prosnitz and Dibona, 1978) clearly indicate that altered RSNA can have a major influence on renal sodium excretion in the absence of significant renal hemodynamic responses. Thus, it is quite tenable that a reduction of RSNA could lead to a natriuresis during WI in the absence of a change of GFR. In this regard, there is no change in renal blood flow during WI, despite the elevation of arterial pressure, suggesting that the kidney autoregulates very well during WI.

Miki *et al.* (1988b) have shown that WI of conscious dogs leads to a depression of RSNA on the order of about 50% below the pre-immersion

levels. In addition, denervation of the kidneys in these dogs largely abolishes both the diuresis and the natriuresis. Therefore, it may be concluded that the renal sympathetic nerves play a primary role in the initiation and the maintenance of the natriuresis and the diuresis during WI. An extension of the conclusion would be that while there are hormonal adjustments occurring in WI, these hormonal adjustments play only a secondary or modulating role in the responses which are mediated by reflex adjustments of RSNA. Indeed, the renal functional response is not dependent upon the extra-renal hormonal adjustments. In the renal-denervated dog, one would expect that hormonal changes similar to those which occur in the intact dog are taking place, yet there is no diuresis or natriuresis.

Therefore, the present day view of the cardiac, endocrine, and renal adjustments in WI would consider that the cardiac, or more specifically, atrial distention occurring during WI leads to a reflex suppression of RSNA via activation of atrial mechanoreceptors. This response is accompanied by hormonal adjustments which modulate the primary renal response depending upon the hydration state, the level of volume replacement, and circadian influences (Behn, 1969; Claybough *et al.*, 1987; Miki *et al.*, 1988a).

Additional support for the idea that hormonal changes serve to modulate the primary renal response is derived from studies of the effects of physical training on the renal response to WI. Claybaugh *et al.* (1987) have shown that, by comparison to sedentary or untrained subjects, trained runners and swimmers have attenuated diuretic and natriuretic responses to WI. These attenuated responses occur despite more sustained increments in stroke volume and cardiac output. At least part of the depression of the renal response could be attributed to altered hormonal responses.

5. Prostaglandins

In addition to the hormonal and neural systems involved in this response, Epstein has provided data which clearly indicates that renal prostaglandin excretion increases in a major way during WI. Prostaglandins have been shown to inhibit the action of vasopressin in stimulating cyclic adenosine monophosphate formation which in turn alters the permeability of the collecting duct (Miki *et al.*, 1987a). Indomethacin has been shown to attenuate the natriuresis of WI in man (Gilmore, 1983). Thus autoids such as prostaglandins may have a significant role to play in the intrarenal modulation of the natriuretic and free water responses of the kidney during WI.

6. Summary

The important implications of these data for humans in the sea are that, during head-out WI in the absence of significant thermal stress, there are powerful neural stimuli which provide the basis for increased elimination of sodium and water by the kidney. The magnitude and character of the renal response may be modified substantially by hydration conditions, the level of physical training, and circadian factors. The modifying influences may be mediated partly by hormonal influences, and partly by alterations of the RSNA mechanism.

It is important to keep these potential modifying influences in mind because factors such as the level of fitness or circadian alterations could have a marked bearing on the nature of the renal functional response occurring in trained divers upon immersion, or in trained astronauts in the weightless state.

III. HORMONAL RESPONSES

A. Vasopressin

The classical experiments of Gauer and Henry carried out in the anesthetized dog indicated that an increase of left atrial pressure leads to a diuresis which consists largely of a free water component (Gauer and Henry, 1976). Subsequent studies showed that this water diuresis was associated with a decline of plasma vasopressin levels and that the diuresis was inhibited by bilateral cervical vagotomy (Gauer and Henry, 1976; Gilmore, 1983). These early experiments thus led to the view that the atrial distention evoked by volume expansion elicited a reflex reduction of vasopressin secretion mediated via the vagus nerves. Thus a mechanoreceptor suppression of vasopressin was thought for many years to be a primary mechanism in the diuresis of WI.

Recently, however, it has been determined that the diuresis response to WI, atrial balloon inflation, or isoosmotic volume expansion in conscious dogs consists mainly of a natriuretic component with a smaller component contributed by an increase in free water clearance. It is clear that vasopressin can be reduced in WI and, if it is reduced, the mechanism probably involves mechanoreceptors since the plasma osmolality is generally unchanged in WI (Epstein, 1987). Unfortunately, as Epstein has pointed out (Epstein *et al.*, 1981; Norsk and Epstein, 1988), it is difficult to obtain a strong correlation of the renal functional response to WI with plasma vasopressin levels. For example, in well-hydrated individuals, vasopressin levels are low and they do not change

very much during WI. Yet there is a larger free water contribution to the diuresis. On the other hand, in dehydrated subjects, plasma vasopressin levels can be quite high, and plasma vasopressin levels decline in a major way in WI. However, the free water component is minimal in the diuresis occurring in WI in dehydrated subjects. Therefore, the precise role of vasopressin in WI has been difficult to establish. There is no doubt that vasopressin tends to decline in WI under certain circumstances (Norsk and Epstein, 1988). Moreover, Bie *et al.* (1984) have pointed out that the kidney is exquisitely sensitive to even small changes in plasma vasopressin which are difficult to detect. In addition, it may be that there are alterations of intrarenal modulators of the hydrosmotic effects of vasopressin during WI, such as prostaglandins, which in turn control the influence of vasopressin on free water excretion.

Recent experiments in our laboratory using the conscious dog model indicate that plasma vasopressin does not change significantly during WI whether the experiments are done under conditions of volume depletion or repletion. Therefore, the renal functional response to vasopressin in the intact dog appears to be independent of vasopressin. By contrast, there is a highly significant reduction of plasma vasopressin if WI is carried out in the cardiac denervated dog. The diuresis in the CD dogs consists entirely of a free water component which is well correlated with the reduction of plasma vasopressin (Hadjuczok *et al.*, 1987a).

The latter experiments suggest either that the cardiac receptors inhibit the release of vasopressin during WI, or that the altered hemodynamic response pattern occurring in WI after CD reduces vasopressin secretion. For example, the increase of aortic pulse pressure occurring when mean arterial pressure rises during WI in the CD dog may load the arterial baroreceptors to the extent that vasopressin is inhibited in that situation. In any case, the potential exists for vasopressin to be reduced in WI as a redundant mechanism for fluid elimination. Measurement of regional cerebral blood flow during WI using radiolabeled microspheres indicate that blood flow to the neurohypophysis is unchanged during WI in sham dogs. By contrast, there is a highly significant decline of blood flow to the neurohypophysis during WI in the CD dogs which correlates well with the decline of vasopressin observed in this situation. Thus, there is probably a coupling of blood flow to vasopressin secretion occurring in this circumstance (Hadjuczok *et al.*, 1987b).

B. Plasma Renin Activity

Plasma renin activity (PRA) has generally been reported to decline in WI in human subjects. In one study, PRA was not reported to change in

WI conscious dogs, however, if experiments are carried out in well-trained dogs, a decrease of PRA can be detected during both volume repletion and depletion (Sondeen *et al.*, 1988). In general, the reduction of PRA can be shown to be correlated with a decline of plasma aldosterone secretion (P_{ALDO}). However, the natriuresis of WI cannot be attributed to a decrease of P_{ALDO} directly because the onset of the natriuresis is too fast to be mediated by an aldosterone effect (Epstein, 1978; Epstein *et al.*, 1973). In humans, the diuresis of WI is not suppressed by pretreatment of the subjects with the mineralocorticoid, DOCA, although there is some suppression of the natriuresis. Hence, the renin-aldosterone system may partly contribute to the natriuresis of WI, but it has been demonstrated that a major WI natriuresis can occur in the absence of changes in renin and aldosterone (Hadjuczok *et al.*, 1987a).

C. Atrial Natriuretic Peptide

Stretch of the atria has been shown to increase the circulating levels of a peptide which has natriuretic properties. This peptide has been termed atrial natriuretic peptide or atripeptin (AP). AP levels are increased during volume expansion, atrial balloon inflation, and during WI (Goetz, 1988). However, the circulating levels of AP which are achieved during WI are such that they are not high enough to elicit a natriuresis (Goetz, 1988). On the other hand, it may be that the natriuretic response to AP has a temporal dependency as low doses of AP infused for longer periods of time have been shown to elicit a natriuresis (Goetz *et al.*, 1986; Goetz, 1988). Nevertheless, at this point, it is difficult to establish whether AP is natriuretic at physiological concentrations.

D. Circadian Dependency

The renal response to WI has a circadian dependence. WI of subjects during the daytime leads to a diuresis and a natriuresis. If the same subjects are immersed at night it is found that the diuresis and natriuresis are depressed significantly. The interesting feature of this response is that plasma levels of renin, aldosterone and vasopressin are suppressed and plasma AP levels are elevated in similar fashion during the daytime and during the nighttime. Thus, despite similar hormonal patterns, there are marked differences in the renal functional responses at night. These experiments are of critical importance because they indicate that the renal response is not directly correlated with the hormonal adjustments and that some other mechanism must be the primary cause of the renal responses (Miki *et al.*, 1988a; Krishna and Danovitch, 1983). It is also of

interest that the cardiac distention responses and therefore the central hemodynamic adjustments were similar during the day and night as reflected by impedance cardiography measurements (Shiraki *et al.*, 1986).

IV. FLUID SHIFTS

As mentioned earlier, it is difficult to find evidence which supports the view that there is a net reduction of the plasma volume as a consequence of WI. In fact, the usual report is that plasma volume either increases or remains the same during WI. In the late 1940's von Diringshofen (1948) postulated that a net fluid shift occurs from the interstitial compartment into the plasma compartment during WI. This shift was predicted based on a theoretical consideration of arterial, capillary, and venous pressure changes during WI.

The first experimental confirmation of the von Diringshofen hypothesis was provided by Davis and Dubois (1977). They studied anesthetized, splenectomized, and nephrectomized dogs during WI and found that plasma volume increased during WI based on measurements of hematocrit. Their results indicated that the plasma became hypotonic as a result of the fluid shift and they postulated that this factor led to the reduction of vasopressin at the onset of a water diuresis. They suggested that fluid actually shifted out of the cellular compartment into the plasma during WI.

Subsequently, Miki *et al.* (1986a) in our laboratory developed a method for the continuous measurement of blood volume in the conscious dog during WI. The dogs were chronically splenectomized and catheters were implanted to allow the establishment of an extracorporeal circuit which allowed for the continuous sampling of blood. Hematocrit was also measured continuously using a conductivity cell. Erythrocytes were labelled with ^{51}Cr and the blood was sampled using a gamma counting tube. In addition, the dogs had been implanted with porous tissue capsules in the upper forelimb and lower hindlimb to allow for the estimation of interstitial fluid pressure.

WI led to an immediate increase of plasma volume which increased progressively to reach a peak value after 35 min in the water. Thereafter, the plasma volume leveled off as the diuresis began at this point in time. The peak plasma volume increment amounted to 7% above the pre-immersion level. Upon removal of the dog from the water after 100 min, the plasma volume declined very rapidly to return to control levels (Fig. 7). In addition, plasma osmolality did not change, suggesting an isotonic fluid shift.

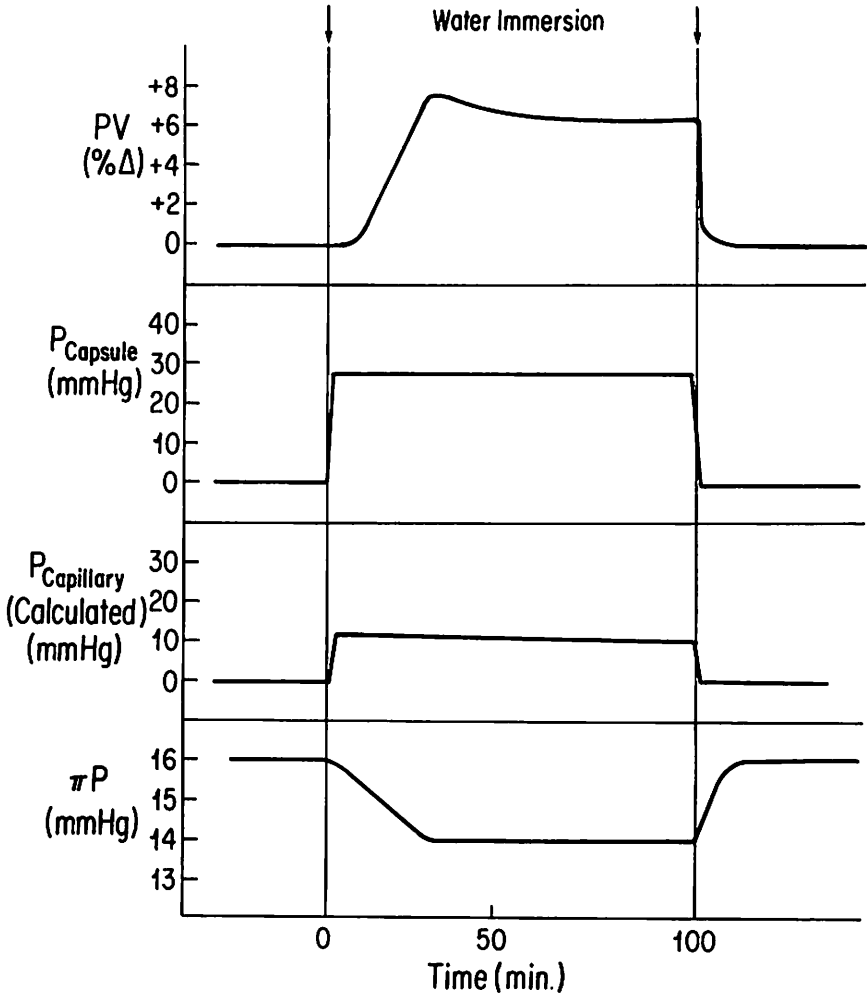


FIG. 7. Increase of plasma volume (PV) during WI in conscious, trained dogs. The estimated interstitial hydrostatic pressure (P_{capsule}) exceeds the capillary hydrostatic pressure ($P_{\text{capillary}}$) calculated from arterial and venous pressures and assumed arteriovenous resistance ratios. Plasma oncotic pressure (πP) decreases as fluid enters the plasma compartment. Data are redrawn from Miki *et al.* (1987a).

Thus, a significant hemodilution occurs in WI. These dogs were mildly hydrated prior to WI, but they were studied under volume depletion conditions. Capillary hydrostatic pressures were calculated from the arterial and venous pressure gradients. These data and the capsule data indicate that while capillary hydrostatic pressure increases in WI, the rise in interstitial fluid pressure is greater. Moreover, there is a decline of plasma oncotic pressure. Therefore, the Starling forces favor net capillary fluid reabsorption during WI.

The control of extracellular fluid volume (ECFV) during WI was studied in another group of dogs which had been instrumented chronically and splenectomized (Miki *et al.*, 1987a). These dogs were anesthetized and acutely nephrectomized on the day of the experiment and studied in the anesthetized state. ECFV was continuously estimated using ^{125}I -iothalamate which behaves similarly to inulin. In addition, plasma volume was measured continuously using ^{51}Cr as in the awake animals.

These data indicate that ECFV increases by 4% above the initial ECFV during WI and that plasma volume increases by 33% in these nephrectomized animals. Studies of the dogs for 120 min of WI indicate that plasma volume increases by as much as 40% above the pre-immersion levels. Calculations of interstitial fluid volume indicate that the volume of this compartment changes very little during WI but that the volume of the cellular compartment decreases. Therefore, WI leads to a massive shift of fluid from the intracellular compartment through the interstitial compartment into the plasma compartment. The apparent role of the kidney during this response is to eliminate the shifted fluid and minimize the increase of plasma volume. The idea that the shifted fluid comes from the cells is supported by the fact that plasma $[\text{K}^+]$ increases in the nephrectomized animal reflecting the movement of a K^+ rich fluid into the plasma compartment.

The total volume of urine which is eliminated during a WI of several hours duration is much more than could be derived from the plasma compartment alone. The transcapillary nature of the fluid shift is indicated by the fact that measurements of lymph flow in the thoracic duct in conscious dogs reveals that lymph flow more likely decreases rather than increases in WI (Miki *et al.*, 1987b).

In another experiment, dogs were instrumented with porous capsules or wick catheters to estimate interstitial fluid pressure during graded immersion. Venous pressure was measured using a catheter with its tip positioned in the foreleg cephalic vein close to the site of interstitial pressure measurement (Miki *et al.*, 1986b).

WI of the awake dog to the mid-foreleg level led to an increase only of the interstitial pressure and venous pressure did not change. WI to the

mid-chest level elicited a further increase of interstitial pressure and, at this level, the venous pressure showed a small increase. Immersion to further depths produced further increments in interstitial pressure and venous pressure but a pressure gradient persisted between the venous compartment and the interstitial compartment. The lack of elevation of venous pressure when the dogs were only immersed to the forelimb level probably reflects relative differences in the compliance of the venous compartment and the interstitial compartment. Moreover, the rise in central venous pressure with increasing WI depth was linearly correlated with the rise of the cephalic venous pressure. These data provide confirming evidence that a pressure gradient develops between the interstitial compartment and the capillary compartment which favors fluid reabsorption. The critical question which remains is how does fluid come out of the cell into the plasma during WI?

There are several possible mechanisms which might explain this response. The first one relates to the fact that the fluid shifting into the plasma from the interstitial compartment is isotonic (in disagreement with the earlier observations of Davis and Dubois) (1977). An isotonic fluid movement of this type would serve to leave behind proteins in the interstitial compartment. This in turn would elevate interstitial protein concentration which would serve to pull fluid out of the cell on an osmotic basis. In support of this idea, measurements of fluid from the porous capsules indicate that the protein concentration tends to increase (Fig. 8a).

A second mechanism might involve ion pumps located in the cell membrane. It has been known for some time that if the cell volume is increased, there will be a rapid response of the ion pumps in the cell membrane which will pump both ions and fluid out of the cell to restore volume. In WI, the hydrostatic pressure might be perceived by the cell as an increase of cell volume and this stimulus might turn on the cellular ion pumps. An effect of this nature would have to be compatible with the observation that a K^+ rich fluid apparently leaves the cells, possibly with amino acids (Miki *et al.*, 1987a) (Fig. 8b).

The third mechanism may simply be related to differences in compliances existing between the plasma compartment, the interstitial compartment, and the cellular compartment. In other words, an elevated external hydrostatic pressure might decrease cell volume more than the volume of the interstitial compartment. Thus, fluid moving out of the cell into the interstitial compartment would raise interstitial pressure without much of a change of interstitial volume and increased capillary reabsorption would then occur. Any one or a combination of these mechanisms acting in concert could account for the fluid shift out of the cells. There is a need for further studies to sort out these mechanisms (Fig. 8c).

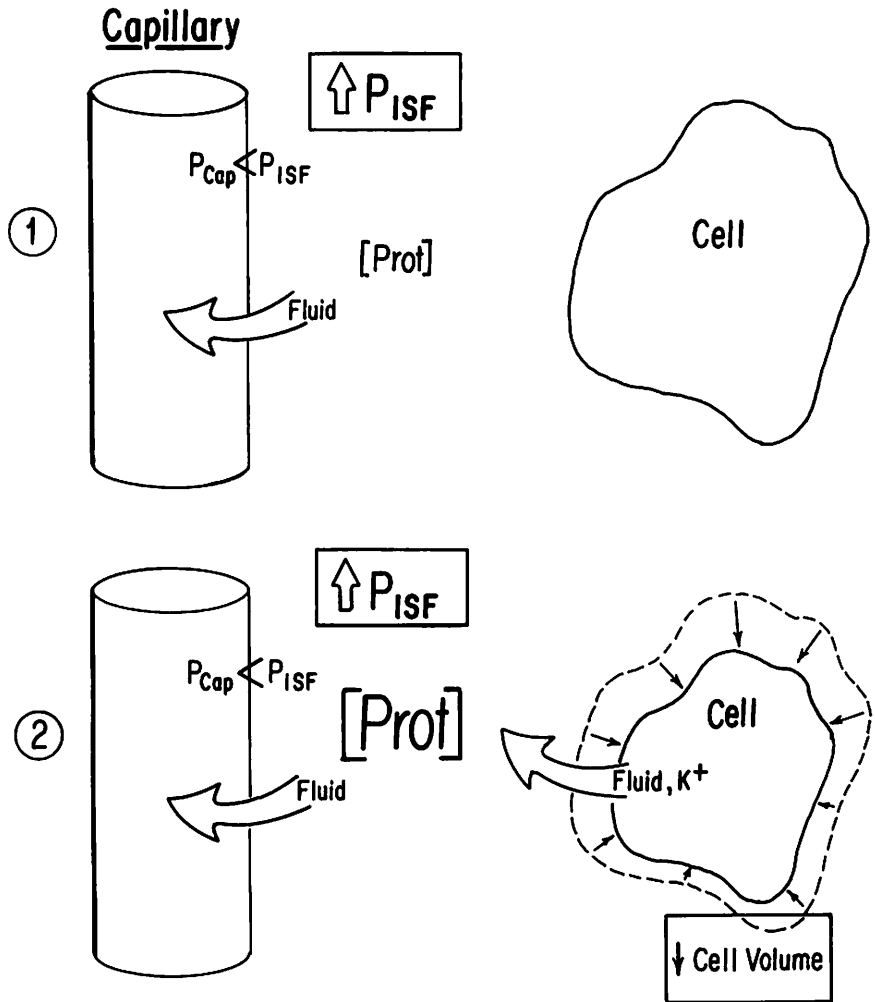


FIG. 8. Possible mechanisms for the fluid shift in WI:

(a) an initial shift of fluid out of the interstitial compartment owing to a pressure gradient $P_{isf} > P_{cap}$ may increase interstitial protein concentration $[prot]$. The rise in $[prot]$ would then draw fluid out of the cells osmotically and decrease cell volume.

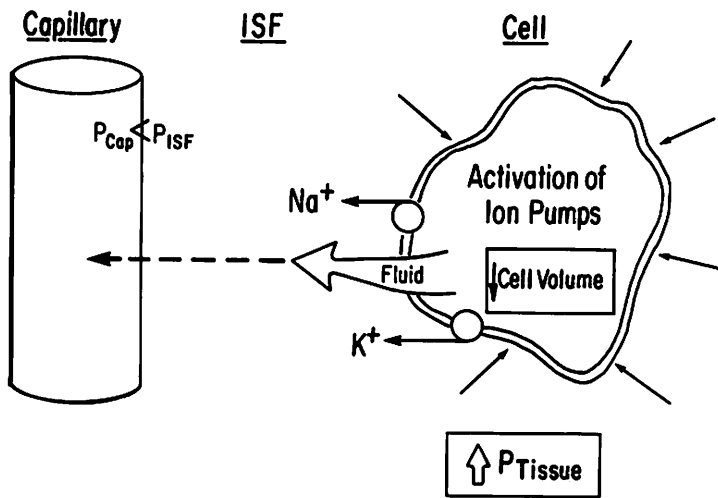


FIG. 8. Possible mechanisms for the fluid shift in WI:
 (b) An increase of cell volume activates membrane ion pumps which pump ions out of the cell to restore cell volume. The tissue compression of WI might be perceived as an increase of cell volume which could activate membrane ion pumps. The pumping of Na^+ and K^+ would then account for the reduction of cell volume in WI.

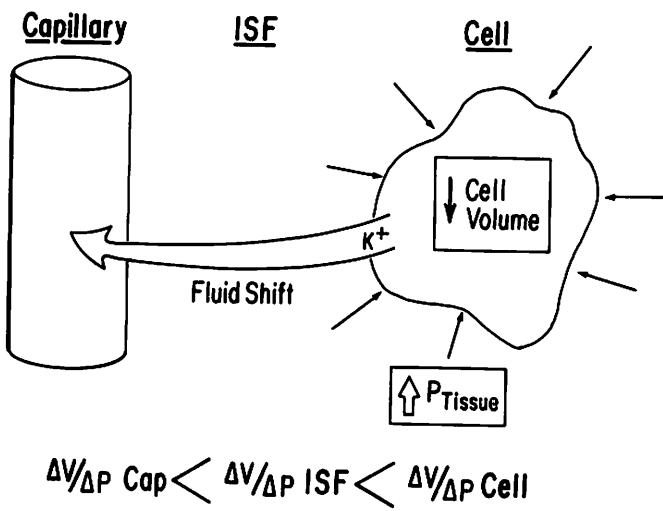


FIG. 8. Possible mechanisms for the fluid shift in WI:
 (c) The shift of fluid out of the cells and into the plasma in WI may be due to differences in compliance ($\Delta V/\Delta P$) between the cell, ISF, and capillary compartments. Thus, a given rise of external pressure elicits a greater decrease of volume in the cell than in the other two compartments.

Several laboratories have confirmed the fundamental observation that a fluid shift occurs during WI in humans using either hematocrit, Evans Blue, or plasma protein to estimate plasma volume. In addition, the time course of the fluid shift occurring in the human appears to be similar to that occurring in the dog (Greenleaf *et al.*, 1983). Khosla and Dubois reported that the interstitial fluid pressure actually decreases in the human during WI when measured by wick catheter. This apparent discrepancy can be easily explained by the fact that Khosla and Dubois used the water level as their zero pressure reference whereas we used the level of the right atrium along with a pleural balloon catheter for our zero pressure reference. If the zero reference differences are taken into account then there is no difference between the observations of Khosla and Dubois (1981) and our observations.

In terms of human performance in the sea, the fluid shift during WI is quite important. Depending on the experimental conditions, the fluid shift will lead to cellular dehydration and loss of K^+ from the cellular compartment. In addition, the magnitude of the plasma volume increase depends upon the ability of the kidneys to eliminate the shifted fluid. If inadequate renal function is present, the fluid shift could lead to a massive hypervolemia which in turn would stress the systemic and pulmonary circulations. Factors such as water temperature, positive pressure breathing, exercise, and physical fitness probably contribute both to the magnitude of the fluid shift and the ability of the body to deal with it.

V. PULMONARY EFFECTS

WI brings about two mechanical effects on the chest wall and the lungs. First, as discussed above, the central shift of fluid into the thorax leads to an increase of the pulmonary vascular volume and pressure (Begin *et al.*, 1976). The latter effect acts to displace air from the lungs and it therefore reduces the vital capacity. A major consequence of this effect is that the pulmonary artery pressure may rise to rather high levels. Second, if WI is to the level of the neck, then the hydrostatic compression of the water will load the chest wall. This influence renders it more difficult to expand the thorax. The consequence is negative pressure breathing (Hong *et al.*, 1969) associated with a greater effort in order to achieve gas exchange. This effort is usually not perceived as dyspnea, but it is accompanied by increased blood flow to the intercostal muscles and to the diaphragm (Hajduczuk *et al.*, 1976a). Thus, the work of breathing is elevated during WI. The greater inspiratory effort serves to further elevate blood into the thorax and add to the increased intrathoracic volume.

The pattern of breathing during WI resembles that which occurs during elastic loading (Hong *et al.*, 1969). In the dog, this is generally associated with a tachypnea. By definition, under thermoneutral conditions, the systemic O_2 consumption does not change. The arterial PO_2 tends to decline, although the arterial blood remains well saturated. In addition, there is a decline of the arterial CO_2 tension (Hajduczuk *et al.*, 1987a). Prefaut *et al.*, (1979) attributed the mild hypoxemia observed in man to an increase of pulmonary closing volume. This effect of the increased closing volume may be offset to some extent in man by an increase of the distribution of pulmonary blood flow to the more apical regions of the lungs (Arboreliuis *et al.*, 1972). Thus, there may be a better matching of perfusion to ventilation in WI but specific studies to address this question have not been carried out.

The neural control of breathing may be influenced to a major extent during the loaded breathing of WI because the mechanical effects on the chest wall and the engorgement of lung circulation serve to activate pulmonary and chest wall mechanoreceptors which lead to an altered breathing pattern.

In summary, the WI maneuver has dramatic effects upon the chest wall and lungs. This is an area which requires further investigation because of the important therapeutic implications of WI and because of the potential influence these adjustments may have on man in the sea.

VI. REGIONAL CIRCULATION

During thermoneutral WI, there is no change of the systemic O_2 consumption and the core temperature of the body is unchanged. In this situation, there is a sustained elevation of the cardiac output which means that there is a general elevation of the ratio of systemic blood flow to metabolism.

An understanding of the distribution of the elevated cardiac output in WI is important in order to be able to interpret the sustained systemic hyperemia response. The cardiac output is elevated during immersion and this may or may not be accompanied by an elevation of the arterial pressure (Arborelius *et al.*, 1972; Hajduczuk *et al.*, 1987a). The mechanism responsible for the elevation of the cardiac output is related to an elevation of the cardiac preload. Therefore, there is an increase of cardiac volume work during WI. In addition, the elevation of either systolic or mean arterial pressure will raise the cardiac afterload. Hence, it is also likely that there is increased cardiac pressure work during WI. The increases of cardiac volume and pressure work lead to an increase of myocardial O_2 demand. This accounts for the increase of coronary blood

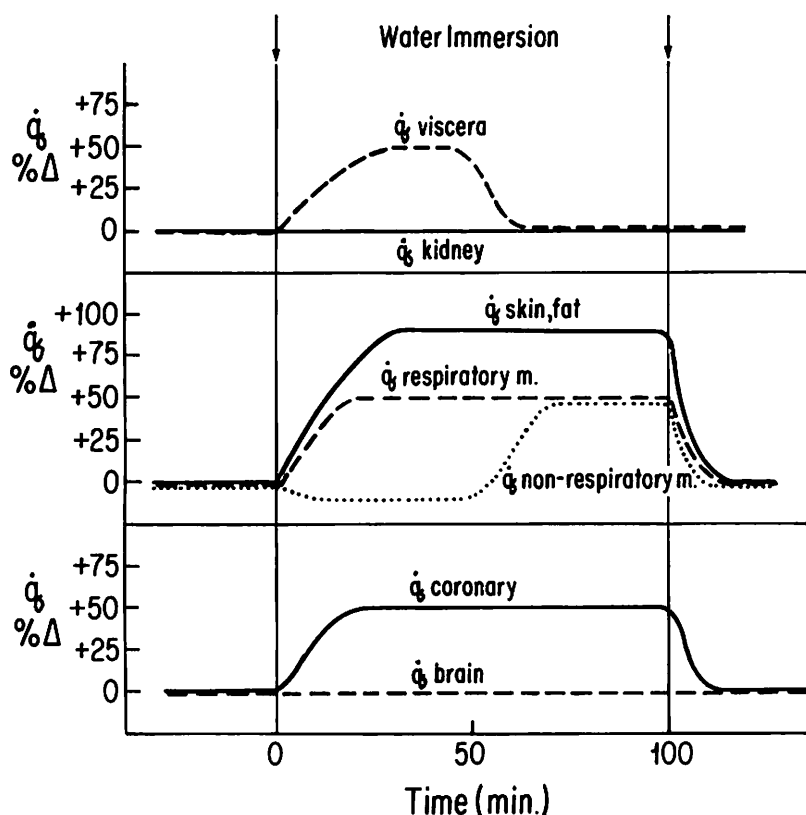


FIG. 9. Regional blood flow adjustments (\dot{q}) as measured by 15 micron radioalabeled microspheres in conscious, trained dogs during WI. Data illustrate the time course of the percent changes of blood flows from the pre-WI levels. Responses were similar in cardiac denervated dogs. Note that the cardiac output is distributed into the viscera early in WI, and then into skeletal muscle later in WI. Data are redrawn from Hajduczuk *et al.* (1987b).

flow which occurs during WI (Hajduczuk *et al.*, 1987b, Fig. 9). Measurements of regional myocardial blood flow during WI in conscious instrumented dogs indicate that, although myocardial flow is elevated, the transmural distribution of blood flow across the ventricular wall is unchanged.

In addition, as mentioned above, there are increases in blood flow to the intercostal muscles and to the diaphragm. These flow increments occur in order to provide increased O_2 delivery to accommodate the increased metabolic demand of the respiratory muscles (Hajduczuk *et al.*, 1987b).

While the core temperature of the body is unchanged during thermoneutral WI, the transition of the subject from room air to the warm water of the thermoneutral environment leads to a warming of the skin and insulative tissues. Thus, during WI there are increases of blood flow to skin and subcutaneous fat on the order of several hundred percent. The increased blood flows to the superficial layers of the body are probably related to superficial heating. These responses may be either locally mediated by the tissue warming, or they may be related to altered sympathetic neural activity (Hajduczuk *et al.*, 1987b).

Therefore, several of the regional flow adjustments which occur during WI can be accounted for on the basis of either increased local metabolic demand (heart, respiratory muscles) or thermal requirements (skin, fat).

On the other hand, there are other major blood flow adjustments which occur during WI that are less easily explained. As mentioned previously, there is no change in blood flow to the kidney during WI and GFR is unchanged. However, in terms of the other abdominal viscera, there are early major increases of blood flows to the pancreas, spleen, hepatic arterial vascular bed, and all segments of the gastrointestinal tract. These visceral flow increments are in proportion to the increase of the cardiac output which is apparently directed into the splanchnic vascular circuit. The latter flow adjustments do not persist however as they are time-dependent. The early increases in visceral flows occur during the first 30 min of WI. After 30 min, there is a striking change in the overall flow response pattern. Early in WI there are no changes observed in the blood flow distribution to non-respiratory skeletal muscles. However, later in WI, the increased visceral flow declines to pre-immersion levels and the elevated cardiac output is directed into the skeletal muscles, away from the viscera (Fig. 9).

Therefore, in early WI, since the arterial pressure is elevated, there is vasoconstriction in the skeletal muscles. Later in WI, the skeletal muscles vasodilate and they receive the increased cardiac output (Fig. 9). The physiologic basis for these regional flow readjustments occurring over time in WI is uncertain. The early vasoconstriction in skeletal muscle may be related to increased muscle sympathetic activity, or it may be mediated by autoregulation. The muscle bed may dilate later on because of changes of skeletal muscle tonus or due to local warming of deep muscle vessels. Conversely, the reason for the delayed vasoconstriction occurring in the viscera is also unclear. In any event, the sustained response to WI appears to consist of increased skeletal muscle blood flow, although specific studies to address this question have not been carried out. Other investigators have demonstrated that there is increased skeletal muscle blood flow in WI using the $^{133}\text{Xenon}$ washout method

(Balldin *et al.*, 1971). The hyperemia of WI has been suggested to be a useful tool for increasing the washout of N_2 in the treatment of decompression sickness. In addition, the fact that there appears to be a selective increase in blood flows to certain visceral organs early in WI suggests that WI might be useful as a tool to provide a non-pharmacologic means of increasing drug delivery to certain organs (Lin, 1984).

The regional vascular adjustments to WI might also contribute to the fluid shift which occurs. Clearly, the muscle vasoconstriction occurring early in WI would act to decrease capillary hydrostatic pressure in this large vascular bed and lead to increased capillary reabsorption (Mellander, 1960). Later on, the visceral vasoconstriction could also contribute to the fluid shift. Thus, the source of the fluid entering the plasma may be initially mainly skeletal muscle, and then later, certain abdominal viscera.

Although arterial pressure tends to rise during WI, there is no overall change in cerebral blood flow. Therefore, there is cerebral vasoconstriction during WI. This response may be due either to the mild hypocapnia which develops, or more likely, be representative of an autoregulation response (Hajduczuk *et al.*, 1987b). The one region of the brain which exhibits consistent flow changes appears to be in the cerebellum which shows increased blood flow. This local cerebellar response may reflect regional cerebral metabolic changes associated with an altered vestibular and postural reflex input owing to the buoyancy effect of the water. This specific response requires further investigation in view of the fact that the cerebellum has been shown to exert powerful effects upon blood pressure regulation (Hajduczuk *et al.*, 1987b).

It has been proposed by a number of investigators that cardiac receptor afferents can exert important influences upon regional blood flow (Bishop *et al.*, 1983). In this regard, the results of the cardiac denervation experiments are interesting in that, with one exception, the regional blood flow patterns observed in WI were similar to those occurring in the intact or sham-denervated animals (Fig. 9). These data indicate that the peripheral circulatory response to WI is not dependent upon reflex influences arising from cardiac receptors, in contrast to the renal functional response. The one difference occurring after cardiac denervation occurred in the pituitary. In the sham dogs, there was no change of neurohypophyseal blood flow during WI. However, after CD, there was a significant decline of blood flow during WI in parallel with the decline of vasopressin levels in the plasma. Thus, the cardiac receptor input has an important role to play in governing blood flow to the neural lobe and vasopressin secretion.

In summary, there are profound regional circulatory adjustments which are elicited during thermoneutral WI. Several of the flow responses

can be accounted for on the basis of local metabolic or thermal responses. However, the major flow adjustments to the viscera and to skeletal muscle remain unexplained along with the time-dependency of these responses. The importance of increased cerebellum blood flow in WI remains to be determined. However, the cardiac nerves make little contribution to these flow adjustments. The visceral and the muscle flow changes may contribute to the fluid shift which occurs.

VII. CONCLUSIONS

Current data indicate that the diuresis response during WI acts to minimize the hypervolemia which would otherwise be quite massive in the absence of the kidneys. The primary mechanism for the diuresis and natriuresis involves activation of cardiac mechanoreceptors which in turn reflexly reduce renal sympathetic nerve activity. Although plasma atripeptin levels rise during WI, and plasma renin activity declines, it is difficult to assign a major role to these hormonal adjustments with respect to the renal response. Changes in plasma vasopressin are difficult to correlate with the renal functional response. Thus, the primary role of the hormonal adjustments is that of modulating the renal response during conditions of altered hydration, circadian influences and physical training.

In consideration of man in the sea, it is well to keep in mind that the massive fluid shift out of the cells can lead to marked cellular dehydration and loss of intracellular K^+ . The functional consequences of the latter effects require further evaluation. Optimal renal function is necessary in order to minimize the plasma hypervolemia.

ACKNOWLEDGEMENTS

The research carried out in the author's laboratory which is described in this report was supported by a Program Project Grant from the National Heart, Lung, and Blood Institute (PO1-HL-28542).

REFERENCES

- Arborelius, M.J., U.I. Balldin, B. Lilja, and C.E.G. Lundgren. Hemodynamic changes in man during immersion with head above water. *Aerosp. Med.* 43:592-598, 1972.
- Balldin, U.I., C.E.G. Lundgren, J. Lundvall, and S. Mellander. Changes in the elimination of 133 -xenon from the anterior tibial muscle in man induced by immersion in water and shifts in body position. *Aerosp. Med.* 42:489-493, 1972.
- Begin, R., M. Epstein, M.A. Sackner, R. Levinson, R. Dougherty, and D. Duncan. Effects of water immersion to the neck on pulmonary circulation and tissue volume in man. *J. Appl. Physiol.* 40:293-299, 1976.

- Behn, C., O.H. Gauer, K. Kirsch, and P. Eckert. Effects of sustained intrathoracic vascular distension of body fluid distribution and renal excretion in man. *Pflugers Archiv.* 313:123-135, 1969.
- Bie, P., M. Mumksdorf, and J. Warburg. Renal effects of overhydration during vasopressin infusion in conscious dogs. *Am. J. Physiol.* 247:F103-F109, 1984.
- Bishop, V.S., A. Malliani, and P. Thoren. Cardiac mechanoreceptors. In: J.T. Shepherd and F.M. Abboud, eds. *Handbook of Physiology. The Cardiovascular System*. Bethesda, MD: Am. Physiol. Soc., Sec. 2, Vol. III, Chapter 15, p. 497-556, 1983.
- Blomqvist, C.G., and H.L. Stone. Cardiovascular adjustments to gravitational stress. In: J.T. Shepherd and F.M. Abboud, eds. *Handbook of Physiology. The Cardiovascular System*. Bethesda, MD: Am. Physiol. Soc., Sec. 2, Vol. III, Chapter 28, p. 1025-1063, 1983.
- Claybaugh, J.R., D.R. Pendergast, J.E. Davis, C. Akiba, M. Pazik, and S.K. Hong. The effect of training on hormonal and urinary responses to supine posture and immersion. *J. Appl. Physiol.* 61:7-15, 1986.
- Davis, J.T., and A.B. Dubois. Immersion diuresis in dogs. *J. Appl. Physiol.* 42:915-922, 1977.
- Dibona, G.F. The functions of renal nerves. *Rev. Physiol. Biochem. Pharmacol.* 94:75-181, 1982.
- Dibona, G.F. Neural regulation of renal tubular sodium reabsorption and renin secretion. *Fed. Proc.* 44:2816-2822, 1985.
- Epstein, M. Renal effects of head-out water immersion in man: implications for an understanding of volume homeostasis. *Physiol. Rev.* 58:529-581, 1978.
- Epstein, M., A.G. Denunzio, and R.D. Loutzenhiser. Effects of vasopressin administration on diuresis of water immersion in normal humans. *J. Appl. Physiol.* 51:1384-1387, 1981.
- Epstein, M., J.L. Katsikas, and D.C. Duncan. Role of mineralocorticoids in the natriuresis of water immersion in normal man. *Circ. Res.* 32:228-236, 1973.
- Gauer, O.H. and J.P. Henry. Neurohumoral control of plasma volume. In: A.C. Guyton and A.W. Cowley, eds. *Cardiovascular Physiology II*. Baltimore, MD: University Park Press, Vol. 9, p. 145-189, 1976.
- Gilmore, J.P. Neural control of extracellular volume in the human and non-human primate. In: J.T. Shepherd and F.M. Abboud, eds. *Handbook of Physiology. The Cardiovascular System*. Bethesda, MD: Am. Physiol. Soc., Sec. 2, Vol. III, p. 885-915, 1983.
- Goetz, K.L., B.C. Wang, P.G. Geer, R.J. Leadley, Jr., and H.W. Reinhard. Atrial stretch increases sodium excretion independently of release of atrial peptides. *Am. J. Physiol.* 250:R946-R950, 1986.
- Goetz, K.L. Physiology and pathophysiology of atrial peptides. *Am. J. Physiol.* 254:E1-E15, 1988.
- Greenleaf, J.E., J.T. Morese, P.R. Baines, J. Silver and L.C. Keil. Hypervolemia and plasma vasopressin response during water immersion in man. *J. Appl. Physiol.* 55:1688-1693, 1983.
- Hajduczk, G., S.K. Hong, J.R. Claybaugh, and J.A. Krasney. The role of cardiac nerves in the hemodynamic and renal responses to head-out water immersion in conscious dogs. *Am. J. Physiol.* 254:R242-R253, 1987a.
- Hajduczk, G., K. Miki, J.R. Claybaugh, S.K. Hong, and J.A. Krasney. Regional circulatory responses to head-out water immersion in conscious dogs. *Am. J. Physiol.* 253:R254-R263, 1987b.
- Hargens, A.R. *Introduction and Historical Perspectives. Tissue Fluid Pressure and Composition*, A.R. Hargens, ed. Baltimore, MD: Williams and Wilkins, 1987.
- Hong, S.K., P. Ceretelli, J.C. Cruz, and H. Rahn. Mechanisms of respiration during submersion in water. *J. Appl. Physiol.* 27:553-538, 1969.

- Khosla, S.S. and A.B. DuBois. Osmoregulation and interstitial fluid pressure changes in humans during water immersion. *J. Appl. Physiol.* 51:686-692, 1981.
- Krasney, J.A., G. Hajduczuk, C. Akiba, B.W. McDonald, D.R. Pendergast and S.K. Hong. Cardiovascular and renal responses to head-out water immersion in canine model. *Undersea Biomed. Res.* 11:169-183, 1984.
- Krasney, J.A., D.R. Pendergast, E. Powell, B.W. McDonald and J.L. Plewes. Regional circulatory responses to head-out water immersion in the anesthetized dog. *J. Appl. Physiol.* 53:1625-1633, 1982.
- Krishna, G.G. and G.M. Danovitch. Renal responses to central volume expansion in humans is attenuated at night. *Am. J. Physiol.* 244:R481-R486, 1983.
- Lin, Y.C. Circulatory functions during immersion and breath-hold dives in humans. *Undersea Biomed. Res.* 11:123-138, 1984.
- Mellander, S. Comparative studies on the adrenergic neurohormonal control of resistance and capacitance blood vessels in the cat. *Acta Physiol. Scand.* 50 (supp):176, 1960.
- Miki, K., G. Hajduczuk, S.K. Hong and J.A. Krasney. Plasma volume changes during head-out water immersion in the conscious dog. *Am. J. Physiol.* 251:R582-R590, 1986a.
- Miki, K., M.R. Klocke, G. Hajduczuk, D. Curran-Everett, and J. Krasney. Effect of water immersion in interstitial and intravascular pressures in conscious dogs. *Fed. Proc.* 45:907, 1986b.
- Miki, K., G. Hajduczuk, S.K. Hong and J.A. Krasney. Extracellular fluid and plasma volumes during water immersion in nephrectomized dogs. *Am. J. Physiol.* 252:R972-R978, 1987a.
- Miki, K., M. Pazik, E. Krasney, S.K. Hong and J.A. Krasney. Thoracic duct lymph flow during head-out immersion in conscious dogs. *Am. J. Physiol.* 252:R782-R785, 1987b.
- Miki, K., K. Shiraki, S. Sagawa, A.J. Debold and S.K. Hong. Atrial natriuretic factor during head-out immersion at night. *Am. J. Physiol.* 254:R235-R241, 1988.
- Miki, K., Y. Hayashida, S. Sagawa and K. Shiraki. Role of sympathetic nerve activity in renal responses during head-out water immersion in conscious dogs. *FASEB J.* 2:A1319, 1988b.
- Norsk, P. and M. Epstein. Effects of water immersion on arginine vasopressin release in humans. *J. Appl. Physiol.* 64:1-10, 1988.
- Pendergast, D.R., A.J. Debold, M. Pazik and S.K. Hong. Effect of head-out immersion on plasma atrial natriuretic factor in man. *Proc. Soc. Exp. Biol. Med.* 184:429-435, 1987.
- Peterson, T.V., J.P. Gilmore, and I.H. Zucker. Initial renal responses of nonhuman primate to immersion and intravascular volume expansion. *J. Appl. Physiol.* 48:243-248, 1980.
- Peterson, T.V., B.A. Benjamin, and N.L. Hurst. Effect of vagotomy and thoracic sympathectomy on responses of the monkey to water immersion. *J. Appl. Physiol.* 63:2476-2481, 1987.
- Prefaut, C., F. DuBois, C. Roussos, R. Amaral-Marques, P.T. Macklem and F. Ruff. Influence of immersion to the neck in water on airway closure and distribution of perfusion in man. *Resp. Physiol.* 37:313-323, 1979.
- Prosnitz, E.H. and G.F. DiBona. Effect of decreased renal sympathetic nerve activity on renal tubular sodium reabsorption. *Am. J. Physiol.* 235:F557-F563, 1978.
- Randall, W.C., M.P. Kaye, J.X. Thomas, Jr., M.J. Barger. Intrapericardial denervation of the heart. *J. Surg. Res.* 29:101-109, 1980.
- Rowell, L.B. *Human Circulation Regulation during Physical Stress*. New York: Oxford, 1986.
- Shiraki, K., N. Konda, S. Sagawa, J.R. Claybaugh and S.K. Hong. Cardiorenal-endocrine responses to head-out immersion at night. *J. Appl. Physiol.* 60:176-183, 1986.
- Sondeen, J.L., S.K. Hong, J. Claybaugh and J.A. Krasney. Renal responses to volume expansion in conscious dogs with and without repletion. *FASEB J.* 2:A1483, 1988.

- Thames, M.D. Contribution of cardiopulmonary baroreceptors to the control of the kidney. *Fed. Proc.* 37:1209-1213, 1978.
- Thames, M.D., B.D. Miller and F.M. Abboud. Baroreflex regulation of renal nerve activity during volume expansion. *Am. J. Physiol.* 24:H810-H814, 1982.
- Von Diringshofen, H. Die Wirkungen des hydrostatischen druckes des wasserbades auf den blutdruck in den kapillaren und die bindegewebsentwässerung. 7. *Kreislaufforsch* 37:382-390, 1948.
- Yoshino, H., D.C. Curran-Everett, S.K. Hong and J.A. Krasney. Altered heart rate-arterial pressure relation during head-out water immersion in the conscious dog. *Am. J. Physiol.* 254:R595-R601, 1988.

2

Physiological Limitations of Humans as Breath-Hold Divers

Yu-Chong Lin

| | |
|--|----|
| I. Introduction | 33 |
| II. Diving Time | 35 |
| A. Predicted Breath-Hold Time | 36 |
| B. Observed Breath-Hold Time | 36 |
| C. Breath-Hold Breaking Points | 38 |
| D. Breath-Hold Time of Professional Divers | 39 |
| III. Diving Depth | 40 |
| A. Predicted Diving Depth | 40 |
| B. Diving Depth of Professional Breath-Hold Divers | 41 |
| C. Breath-Hold Diving Depth Records | 41 |
| IV. Hypothermia | 42 |
| V. Decompression Sickness | 44 |
| VI. Drowning and Near Drowning | 45 |
| A. Shallow Water Blackout and Drowning | 45 |
| B. Near Drowning | 45 |
| VII. Cardiac Arrhythmia and Diving Bradycardia | 47 |
| A. Head-Out Immersion | 47 |
| B. Submersion | 48 |
| VIII. Summary | 50 |
| Acknowledgements | 52 |
| References | 52 |

I. INTRODUCTION

Breath-hold (BH) diving (free diving or skin diving) is an ancient means of working in the sea. Free diving offers the advantages of practicality, simplicity, and mobility. These advantages are offset, however, by the inability of humans to stay underwater for extended periods.

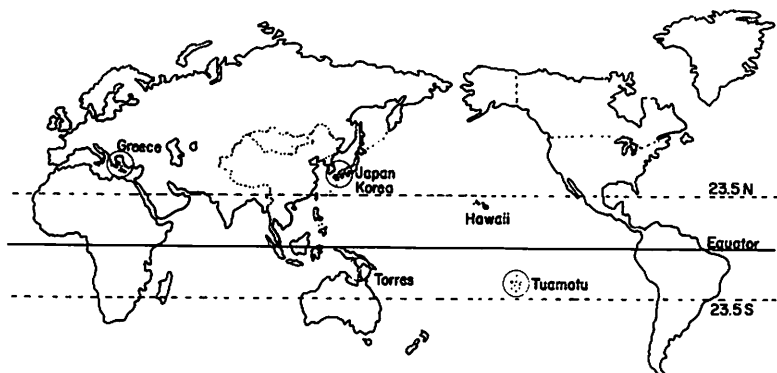


FIG. 1. Prime locations of professional breath-hold divers.

Humans have practiced breath-hold diving as a profession for centuries, and although the number of professional divers is declining, it is still exercised in a few places (Fig. 1). A significant number of professional BH divers continue to find their livelihood along the coasts of Japan and Korea. Also illustrated in Fig. 1 are locations where large numbers of professional BH divers once worked. The advent of synthetic materials rendered shell harvesting economically uncompetitive along the northern Australian coast, scuba diving displaced old ways of gathering sponges in the Aegean Sea, and the cultured pearl eliminated high-risk pearl diving in the Tuamotu Archipelago. Undoubtedly, with industrialization comes new job opportunities, pollution, depletion of marine resources, and changes in general social attitude which all contribute to the continuing decline of BH diving as a profession. Dr. Suk Ki Hong, one of the pioneers in the study of divers at work, predicted BH diving will cease to be a profession around the year 2000 (Hong, 1965). In recent years, however, the number of professional BH divers in Korea and Japan has remained steady at about 20,000 (Park *et al.*, 1988). Sensible practice of ecological principles and lucrative economic rewards have contributed to the break in the predicted decline.

In contrast to the dwindling of professional BH diving, sports diving has been gaining popularity worldwide since World War II. Commercial air transport has greatly extended the traditional summer diving period. However, with increasing popularity of recreational diving, accidents occur more frequently (Craig, 1961a; Craig, 1961b). Accidents associated with skin diving are mostly physiological and usually involve drowning or near drowning and, by and large, may be avoided by exercising sensible diving practices and respecting physiological limits of the human as a diver. This review is pertinent in this regard.

The distinctly unremarkable ability of humans to stay underwater, in some respects, is advantageous. Diving with respiratory aids, as in scuba or surface supplied diving, prolongs the time of submergence, making possible extended underwater exploration, harvesting, inspection, installations, repairs and the like. However, this type of diving brings the danger of decompression sickness which could be manifested immediately or some 20 years after inappropriate diving practices. Because of the biologically imposed limits on underwater durations, BH divers generally avoid decompression sickness. Only rarely and under extreme circumstances can decompression sickness result from BH diving (Cross, 1965; Paulev, 1965).

Teruoka (1932) can be credited with bringing these age-old practices of BH diving into the light of modern scientific scrutiny. In 1932, he was the first to publish a scientific description of diving equipment, diving pattern, and gas exchange in professional BH divers in Japan. His paper offered a glimpse of human limits in imitating diving mammals. In 1965, a symposium held in Tokyo called attention to this original work and has attracted worldwide interest since (Rahn and Yokoyama, 1965). Studies on divers in Japan (called *ama* for sea-women; *katsugi* for sea-men) and Korea (called *hae-nyo* for sea-women) during the past 30 years have contributed significantly to our knowledge of the physiology of BH diving. Combined results from these studies and laboratory investigations represent the current understanding of human limits in diving. The extent of humans imitating diving mammals has been reviewed recently (Lin, 1986; Lin, 1987a; Lin, 1987b; Lin, 1988).

There are many natural and man-made conditions that impede or are detrimental to, human activity in water. For example, locomotion in water requires a great deal more energy than on land, vision is distorted, communication requires special efforts, humans are ineffective in dealing with large waves, strong water currents, biological or chemical contamination, toxic animals and plants, and certainly, sharks. The majority of these conditions are not under the divers' control, and avoidance of these depends on human intelligence and common sense, not on physical ability. This review focuses on the following physiological limitations for humans: diving time, depth, temperature regulation, and associated problems of drowning and cardiac arrhythmia.

II. DIVING TIME

As mentioned, humans can stay underwater for only a brief period. Professional divers who submerge repetitively limit their individual dives to less than 60 sec in Korea (Hong *et al.*, 1963; Park *et al.*, 1983a) and Japan

(Teruoka, 1932; Shiraki *et al.*, 1985). In the laboratory, where BH, exercise, and water immersion can be studied separately, much longer BH times have been demonstrated.

A. Predicted Breath-Hold Time

Theoretically, BH time can be calculated from oxygen supply-demand relationships (Klocke & Rahn, 1959), as:

$$\text{BH with O}_2, \text{ BH time} = \frac{\text{VC(BTPS)}}{\dot{V}_{\text{O}_2(\text{STPD})}} \times \frac{P_B - 47}{863}$$

$$\text{BH with air, BH time} = \frac{\text{TLC(BTPS)} \times F_{\text{AO}_2}}{\dot{V}_{\text{O}_2(\text{STPD})}} \times \frac{P_B - 47}{863}$$

where BH time is expressed in minutes; VC (vital capacity), and TLC (total lung capacity) in milliliters at BTPS (body temperature and pressure, saturated with water vapor); \dot{V}_{O_2} , the oxygen consumption in ml/min at STPD (standard temperature and pressure, dry) F_{AO_2} is the fractional alveolar concentration of oxygen; $P_B - 47$ is the barometric pressure (in torr) less the water vapor pressure at 37°C; and 863 is the constant for converting a gas volume from BTPS in liters to STPD in milliliters. VC instead of TLC is used in the oxygen BH experiment. The assumption here is that residual volume (RV) limits the shrinkage of the lung, and that in BH with air, $\text{TLC} \times F_{\text{AN}_2}$ is greater than RV regardless of BH duration. Estimated from the oxygen supply in the lung alone, a BH time of 4 min with air and 16 min with oxygen should be possible for an average man resting at sea level (VC, 4.78L; RV, 1.19L; and \dot{V}_{O_2} , 250 ml/min).

B. Observed Breath-Hold Time

In fact, BH times similar to predicted values have been demonstrated in subjects breathing air (Hong *et al.*, 1970; Hong *et al.*, 1971) and breathing oxygen (Klocke and Rahn, 1959; Schneider, 1930). BH time should be even longer if the calculation includes usable oxygen in the blood. The record for BH with oxygen without prior hyperventilation is 6 min 29 sec (Table 1). Intense hyperventilation with oxygen or air prior to BH dramatically increases BH time. BH times ranging from 13 to 14 min following hyperventilation with oxygen have been reported by Klocke and Rahn (1959). A student of Schneider's held his breath for 15 min 13 sec after a period of hyperventilation with air followed by 3 deep breaths of

TABLE 1.
Published Records of Breath-Hold Time

| Conditions | BH Time (min:sec) | Subject | Reference |
|------------------------------------|----------------------|-------------|---------------------------|
| Air | 4'30" | Subject SKH | Hong <i>et al.</i> , 1970 |
| | 4'00" | Student | Hong <i>et al.</i> , 1971 |
| O ₂ | 6'29" | Pouliquen | GBWR ^a , 1985 |
| O ₂ , HV ^b | 13'00" | Subject SH | Klocke and Rahn, 1959 |
| | 13'48" | Subject MT | Klocke and Rahn, 1959 |
| | 14'00" | Subject HR | Klocke and Rahn, 1959 |
| | 15'13" | Student | Schneider, 1930 |
| | 20'05" | Frechette | Mithoefer, 1965 |
| O ₂ , HV-W ^c | 13'43" | Foster | GBWR ^a , 1989 |

^a, Guinness Book of World Records

^b, Hyperventilation

^c, Hyperventilation and submergence in water

pure oxygen (Schneider, 1930). One of the most shocking BH records was noted by Mithoefer (1965). Under the same conditions as Schneider's a student at the same university held his breath for 20 min and 5 sec (Table 1). Under simulated "field conditions," Foster held his breath for almost 14 min in a motel pool (heated) following hyperventilation for 30 min with oxygen (Table 1). One should note that these long BH times were achieved under laboratory conditions at rest and that the BH time is inversely proportional to oxygen consumption (Lin *et al.*, 1974). Therefore, such long BH durations cannot be achieved in an actual dive, in which oxygen consumption is elevated. However, these findings show humans can survive without breathing much longer than once thought if oxygen supply is increased and/or oxygen consumption is reduced.

Although oxygen supply-demand relationships accurately predict breath-hold breaking points, these limits can only be reached by using all factors known to prolong BH time and by overcoming physical and psychological discomfort. Factors that prolong BH time have been reviewed by Mithoefer (1965) and Lin (1987a). The most effective means of extending BH time is prior hyperventilation as noted above, which lowers alveolar P_{CO₂} and hence delays the urge to resume breathing. Unfortunately, vigorous

hyperventilation prior to submergence is the single most dangerous practice in open water, causing blackout or shallow-water drowning during ascent (see "DROWNING AND NEAR DROWNING" section).

C. Breath-Hold Breaking Points

Mechanisms leading to termination of a voluntary BH are complex. However, it has long been known the breaking point does not coincide with the sum of chemical stimuli (Godfrey and Campbell, 1968; Kelman and Wann, 1971). Researchers have identified two BH breaking points. The *Conventional Breaking Point*, or simply the *Breaking Point*, is reached when one feels BH can no longer be continued. The time taken to reach this point is the BH time mentioned above. However, BH time varies widely within as well as between individuals by using this criterion, mainly because of the involvement of subjective judgment. *Breaking point* varies from less than 20 sec (Schneider, 1930) to 270 sec (Hong *et al.*, 1970) when subjects are told to expire once as deeply as possible, then inspire fully and hold the breath as long as possible (i.e. BH without prior hyperventilation). Psychological factors are recognized as the major determinants of the conventional breaking point (Hill and Flack, 1908; Schneider, 1930; White, 1920), and undoubtedly contribute to the wide range of BH times reported in the literature.

In contrast to the *conventional breaking point*, the *physiological breaking point*, is sharply defined by chemical stimulus alone, that is the desire to breathe returns. The *physiological breaking point*, in contrast to the *conventional breaking point*, varies within a narrow range (Lin, 1987a). Involuntary ventilatory activities occur when the *physiological breaking point* is reached, but voluntary inhibition of glottis opening is still possible (Lin *et al.*, 1974). A number of terms have been used to identify this stage, such as the end of the easy-going phase, the desire to breathe, want of oxygen, diaphragm contraction, involuntary ventilatory activity, or simply the sensation of air hunger.

Divers should pay close attention to the *physiological breaking point* in order to avoid shallow water drowning. Although BH can still be continued by conscious effort, drowning may occur during the ascent phase of the dive as a result of extreme hypoxia. The desire to breathe occurs at an alveolar P_{CO_2} of the brain stem has been estimated to be between 47 and 48 torr (Table 2) and is reached after 30 to 40 sec of BH following a full expiration and inspiration (Lin, 1987a).

TABLE 2.

Alveolar P_{CO_2} at Physiological Breaking Point^a

| BH sec | P_{ACO_2} mmHg | $P_{ACO_2}^b$ mmHg | Note | Reference |
|-----------|---------------------|-----------------------|--------------------|----------------------------|
| 80 | 48.8 | — | | Kobayashi and Sasaki, 1967 |
| 37 | 49.3 | 47.6 | | Agostoni, 1963 |
| 68 | 48.3 | 46.9 | High \dot{V}_E^c | Agostoni, 1963 |
| 62 | 48.4 | 47.1 | 80% VC | Agostoni, 1963 |
| 66 | 49.3 | 47.7 | 20% VC | Agostoni, 1963 |
| 104 | 48.6 | 47.9 | | Lin <i>et al.</i> , 1974 |
| — | 48.6 | — | | Douglas and Haldane, 1909 |

^a BH with oxygen at rest^b Estimated from P_{ACO_2} with correction of sampling delays^c Minute ventilation in L/min

TABLE 3.

General Patterns of Diving to 5- and 10-m Depth by Wet Suit Divers

| Dive Time Sec | Korean Women ^a | | Japanese Men ^b | |
|---------------------|---------------------------|------------|---------------------------|--|
| | 10-m dive | 5-m dive | 5-m dive | |
| Single dive time | 43 (100%) | 32 (100%) | 39 (100%) | |
| Descent time | 19 (44%) | 9.3 (29%) | 8 (20%) | |
| Ascent time | 12 (28%) | 6.0 (19%) | 8 (20%) | |
| Bottom time | 12 (28%) | 16.5 (52%) | 23 (60%) | |
| Single surface time | 85 | 46 | 42 | |
| No. of dives/hr | 28.1 | 46.2 | 44.4 | |

^a Park *et al.*, 1983a^b Shiraki *et al.*, 1985

D. Breath-Hold Time of Professional Divers

Professional BH divers in Japan and Korea accomplish much useful work in brief periods of less than 60 sec each (Table 3). These divers are disciplined to heed the physiological warning. The actual usable time at the bottom is indeed very short. An earlier survey (Hong *et al.*, 1963) of

women divers who wore simple cotton suits, and 2 new studies (Park *et al.*, 1983a; Shiraki *et al.*, 1985) of divers in wet-suits show that only about half of the diving time is available for bottom activity during a 5-m dive, while only a quarter of the time is available in a 10-m dive, with the remainder spent on ascent and descent (Table 3). Typically, in a 5-m dive the divers use 30 to 40 sec in a single dive and spend 40 to 50 sec on surface. They repeat a similar pattern about 45 times an hour (Table 3). For a 10-m dive, both dive and surface times are lengthened with an increase in surface-to-dive ratio. Number of dives per hour is reduced. Comparing the 5-m dives, men appear to have a slightly longer bottom time than women, because of more forceful descent (Table 3).

The diving pattern described above pertains to a sustained period of repeated dives. Longer dive times of 90 sec (Scholander *et al.*, 1962), 118 sec (Teruoka, 1932), and 155 sec (Cross, 1965) in open sea working conditions were observed occasionally.

III. DIVING DEPTH

The short BH time is one obvious factor preventing divers from reaching great depths. Professionals dive most frequently to a depth of less than 10 m. The BH diving depth rarely exceeds 20 meters even with assist. The term "assist" is used in the sense that descent can be accelerated with minimal energy by using a large counterweight which is lifted on ascent by an assistant on the boat. Two types of professional divers are most common in Japan: *kachido* is the unassisted ama who works attached to a float on the surface and descends under her own power; *funado* is the assisted ama who descends rapidly with a counterweight, releases the weight and works at the bottom, and then is pulled up by a partner on a boat. There are many variations used to lessen energy expenditure on descent, ascent, or both (Yokoyama, 1987), thus increasing both bottom time and depth.

A. Predicted Diving Depth

It was predicted the depth limit in atmospheres for humans should be about equal to the TLC/RV ratio. If TLC is allowed to be compressed down to RV during a dive, the depth limit (D, in meters) should be:

$$D = P_B \times (\text{TLC/RV} - 1) \times 10$$

where P_B is the barometric pressure expressed in atmospheres and 10 represents the seawater equivalent depth (in meters) of 1 atmosphere.

The depth limit should be in the range of 30 to 40 m, according to this prediction. By and large, this is the depth within human reach if no extraordinary procedures are employed. However, this is not the case when extraordinary procedures are used for the purpose of seeking new records. See "*Breath-Hold Diving Depth Records*" section).

B. Diving Depth of Professional Breath-Hold Divers

Teruoka (1932) was the first to describe the diving pattern of Japanese professional divers who dive to around 5-m depths unassisted. A survey in 1963 by Hong *et al.* (1963) showed a similar pattern in unassisted Korean women divers. These divers both descended and ascended at a rate of slightly less than 0.6 m/s. In assisted dives, ascent and descent could be 3 times the rate of unassisted dives and to depths of around 20 m. In general, assisted divers descend quickly by carrying counterweights and ascend at an accelerated rate with help from an assistant utilizing mechanical leverage on a boat.

Recent studies of Korean divers (Park *et al.*, 1983a) and Japanese men divers (Shiraki *et al.*, 1985) showed no drastic changes in traditional diving patterns. Although modern divers wear wet suits, four- to five-m dives are still predominant (Table 3).

Tuamotu pearl divers (men) were the deepest working BH divers known, regularly reaching depths greater than 30 meters (Cross, 1965). They also had a lot of diving accidents, indicating that increased depth is in fact counterproductive. They are the only BH divers to experience a decompression sickness known as *taravana*.

C. Breath-Hold Diving Depth Records

Just as humans can achieve astonishingly long BH time under unusual circumstances without special assist devices, humans also can reach great depths with a single breath. When Craig (1968) reviewed BH diving depths up to 1967, he found that the record of 64.8 m considerably exceeds predicted values. This is possible, as was found later, because RV diminishes (increasing TLC/RV) during BH dive by chestward displacement of blood. Over the years, depth records have increased (Table 4). By early 1976, Mayol reached 86 m or 284 ft (Ricci and Marroni, 1976). Later Maiorca made a new record at 87 m, and his latest record is now 91 m (E. Maiorca, personal com., 1987). In November 1976, Mayol dove to 100 m in 3 min and 39 sec with a single breath (Triton, 1977a). Descent was accelerated by using a counterweight and ascent by a self-inflatable balloon (Triton, 1977b). To prolong BH time, one prerequisite was to lower

TABLE 4.

Depth Records of Breath-Hold Dive

| Year | Divers | Depth (m) | Reference |
|------|-------------------|-----------|-------------------------------|
| 1951 | Felco and Novelli | 35 | Craig, 1968 |
| 1952 | Bucher | 39 | Craig, 1968 |
| 1956 | Felco and Novelli | 41 | Craig, 1968 |
| 1960 | Santarelli | 43 | Craig, 1968 |
| | Maiorca | 45 | Craig, 1968 |
| | Santarelli | 46 | Craig, 1968 |
| | Maiorca | 49 | Craig, 1968 |
| 1966 | Mayol | 60.4 | Craig, 1968 |
| 1967 | Croft | 64.8 | Craig, 1968 |
| 1968 | Croft | 68.6 | Schaefer <i>et al.</i> , 1968 |
| 1974 | Maiorca | 87 | Maiorca, 1987 |
| 1976 | Mayol | 86 | Ricci and Marroni, 1976 |
| 1974 | Maiorca | 94 | Pers. Com., Maiorca, 1987 |
| 1983 | Mayol | 105 | Missiroli and Rizzato, 1984 |

the alveolar P_{CO_2} as much as possible before breath holding. Technique varies on how to lower it. Hyperventilation is common, but in Mayol's case, special posture, acupuncture, and yoga techniques, among other things were used (Triton, 1977a).

The World Underwater Federation in 1960 and the Italian Underwater Federation in 1981 ceased to acknowledge these records judging they were dangerous and added no new knowledge. The race continues, regardless. An unofficial record of 105 m (more than 340 ft) was said to have been made by Mayol in 1983 (Missiroli and Rizzato, 1984). It looks as though the depth limit is yet to be reached (Fig. 2).

IV. HYPOTHERMIA

Another limitation on human in-water activity is that hypothermia inevitably follows prolonged immersion. The water temperature at which the human body neither loses nor gains heat, called *thermoneutral temperature*, is 35°C. This temperature is substantially higher than that of

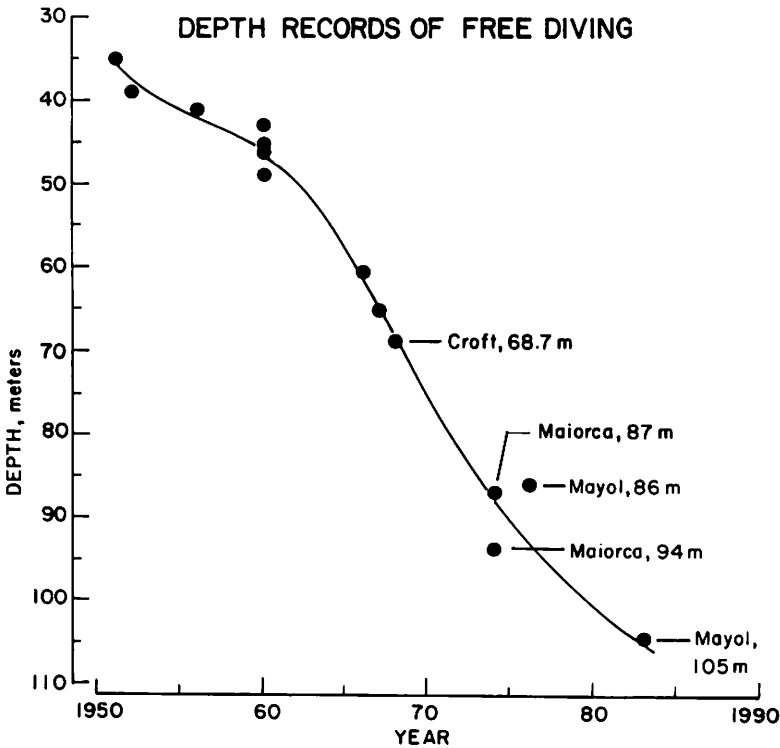


FIG. 2. Depth records of human BH diving. Records made before 1967 were reviewed by Craig (1968).

ocean water even in midsummer, and thermal drain continues as long as a person is in water. Hypothermia affects the performance of all organ systems, and its contribution to drowning is well known (Keatinge, 1969).

Insulation is the only effective protection against hypothermia, because heat flow is determined by the thermal gradient and the properties of the environment. The thermal conductivity of water is 25 times that of air, causing rapid heat transfer along the thermal gradient. In addition to the high thermal conductivity of water, muscular activity in water reduces the thermal insulation of the body shell, thus facilitating heat loss from body core to water during diving (Keatinge, 1961; Park *et al.*, 1984; Rennie *et al.*, 1980; Veicsteinas and Rennie, 1982). Cardiac irregularities, impairment of rational thinking, sensory and motor degradation,

loss of consciousness, and drowning could occur when body temperature falls below 35°C (Webb, 1976). Because of this, professional BH divers stop harvesting, by experience, when body temperature reaches 35°C (Kang *et al.*, 1965) and allow their bodies to warm up before resuming diving. With use of a wet suit, hypothermia is effectively prevented (Kang *et al.*, 1983; Park *et al.*, 1983b; Shiraki *et al.*, 1986). Consequently, the previous practice of breaking up dives into 1-hour shifts in summer, or much shorter shifts in winter, has been replaced by 2- to 3-hour shifts. Termination of diving is no longer dictated by hypothermia.

Besides increasing the risk of hypothermia, cold water shortens BH time. Sterba and Lundgren (1985) and Hayward *et al.* (1984) reported a direct correlation between BH time and water temperature. The reduction in BH time corresponds closely to elevated oxygen consumption (Lin *et al.*, 1974). Lessons on the thermal properties of water and their consequences should therefore be an integral part of the education of sports divers.

V. DECOMPRESSION SICKNESS

Asian professional BH divers by experience stop working before they encounter decompression problems. Decompression sickness does not appear to place constraints on BH diving under ordinary conditions. It becomes significant only under severe diving stress. There was a group of male pearl divers in the Tuamotu Archipelago who encountered the most stressful diving condition — deep and long. They dove to 20 to 30 m regularly. Their dive times usually exceeded 2 min. In order to do this, they hyperventilated heavily. Many divers showed decompression symptoms. They called it *taravana* (tara, means “to fall” and vana means “crazily”). Dizziness, vertigo, vomiting, and paralysis were the most frequently experienced symptoms (Cross, 1965).

Paulev (1965) of Denmark proved in a “free escape” tank at the Norwegian Naval Base that indeed decompression sickness can result from BH diving. He showed decompression sickness can be induced when the dive time-depth product reaches a critical value. He dove to the bottom (20 m) of a submarine escape tower and repeated the dive 60 times over a 5 hour period. He showed progressive symptoms of nausea, dizziness, visual disturbance, and paresis of the right arm during the last 2 hours. The symptoms disappeared upon recompression. Later, two other submarine escape training instructors showed similar symptoms and were also cured by recompression.

VI. DROWNING AND NEAR DROWNING

A. Shallow Water Blackout and Drowning

Alveolar P_{CO_2} rises as a BH dive proceeds and normally, hypercapnic stimuli serve as a signal for surfacing. Although oxygen concentration falls, this provides no stimulus to resume breathing because P_{O_2} remains high at depth by hydrostatic compression. When a person hyperventilates before a dive, the alveolar P_{CO_2} is low at the onset of BH. Under such conditions, before the arterial P_{CO_2} can build up to a level that normally signals the desire to resume breathing, oxygen depletion can occur without the diver's knowledge while at depth (P_{O_2} is high because of compression). Blackout often occurs without warning during ascent due to the extreme hypoxia on re-expansion of the lung. This so-called shallow water blackout still occurs at an alarmingly high rate (Approx. 7,000 per year in the U.S.). It continues despite the publication of detailed scientific findings attributing the blackout to hyperventilation before diving (Craig, 1961a; Hong *et al.*, 1963; Lanphier and Rahn, 1963) and the publication of repeated warnings (Hong and Rahn, 1967; Hong, 1976; Craig, 1976). The perils of excessive hyperventilation should be emphasized in all swimming and diving programs.

B. Near Drowning

Although there is no evidence that humans have the ability to conserve oxygen while diving, it is important to note that diving reflexes that follow submergence enhance the chances of revival, even after what seems to be a hopelessly long apnea. One component of diving reflexes is apnea itself, which prevents water inhalation, greatly reducing complications of resuscitation. The cardiovascular response that follows immersion is also important because preferential redistribution of blood to the heart and brain protects these vital systems. The odds for revival improve when conditions favor rapid chilling of the body, such as cold water, and a thin-bodied or small body mass. Most importantly, hypothermia reduces metabolic requirements, further minimizing heart and brain damage.

In a December 1987 newspaper account, an 11-year-old North Dakota boy survived after 45 min in icy water. His body temperature upon arrival at the hospital was 26.7°C (Table 5). One year after the incident, his physical and mental performance were normal for his age.

Siebke *et al.* (1975) reported the revival of a 5-year-old Norwegian boy who regained full cerebral function after having been under ice water for more than 40 minutes (Table 5). Elliott (1978) reported the revival of an 18-year-old male by Dr. M.J. Nemiroff at the University of Michigan.

TABLE 5.
Once Dead

| Time min | BT ^a °C | WT ^b °C | Age yr | Sex | Remarks | Reference |
|-------------|-----------------------|-----------------------|-----------|-----|---|-----------------------------------|
| 45 | 26.7 | Ice | 11 | M | N. Dakota, Dec. 1987 | News account |
| 40 | 24.0 | Ice | 5 | M | Norway, Feb. 1974 | Siebke, 1975 |
| 40 | | Ice | 18 | M | Minnesota, subm. auto CPR 2 hr, 3.2 GPA ^c | Elliott, 1978 |
| 30 | | 0 | 5 | M | Canada, HR 5-10 bpm | Hunt, 1974 |
| 25 | 31.0 | <20 | 6 | | Denmark Normal after 24 days | Andersen and Theilade, 1978 |
| 25 | | Ice | 2 | M | Nevada, Dec. 1987 | News |
| 22 | 24.0 | -10 | 5 | M | Norway, Mar. 1962 CPR 2 hrs. | Kvittingen and Naess 1963 |
| 20 | 27.8 | 11 | 1 | M | Spain, Feb. 1973 | Dominguez <i>et al.</i> , 1973 |
| 20 | | Ice | 4 | M | Jan. 1984 | Daviss, 1985 |
| 17 | 32.0 | | 21 | M | Melbourne, Apr. 1963 Open-heart massage | King and Webster, 1964 |
| 15 | 32.2 | -1 | 9 | F | New York, Feb. 1987 | Ola and D'Aulaire, 1987 |
| 15 | | <20 | 15 | M | Poland, Drinking alcohol | Jaklewicz <i>et al.</i> , 1980 |
| 10 | | 4 | 25 | F | Diving at 60 fsw | Nemiroff, 1979 |

^a Body temperature

^b Water temperature

^c Grade point average

Rescuers pulled the student from a car in a frozen pond after nearly 40 min. of submergence. The student was revived by 2 hours of cardio-pulmonary resuscitation (CPR) followed by a respiratory assist for 18 hours. The student not only showed no brain damage but also resumed schooling and maintained a 3.2 grade point average.

Other records of lesser degree, but nevertheless involving seemingly hopeless submergence, are listed in Table 5. These records emphasize that aggressive and persistent CPR should be applied in drowning cases, especially when cold water is involved. Heroic efforts to revive near drowning victims have been successful. In two cases, CPR was administered for 2 hours (Elliott, 1978; Kvittingen and Naess, 1963), and open-

heart massage was carried out in another case (King and Webster, 1964). It was said about cold-water drowning: "One is not dead until warm and dead!" (Martin, 1984).

Humans also exhibit diving bradycardia and vasoconstriction, though in a somewhat attenuated form (Hong *et al.*, 1971; Lin, 1983a; Lin *et al.*, 1983a; Lin *et al.*, 1983b; Song *et al.*, 1969). Their circulatory responses lack the intensity and promptness required for effective conservation of oxygen during diving (Hong *et al.*, 1971). Therefore, survival may depend on a reduced body temperature, which in turn lowers the oxygen requirement. This type of survival has, however, not resulted from the oxygen conservation that is so pronounced and effective in diving mammals (Andersen, 1966; Scholander, 1963).

VII. CARDIAC ARRHYTHMIA AND DIVING BRADYCARDIA

It should not be surprising to find that cardiac arrhythmias are found in BH diving because significant cardiac arrhythmias are found, even in common respiratory maneuvers such as deep inspiration, prolonged inspiration, BH, and release of BH (Lamb *et al.*, 1958). Furthermore, arrhythmias are prominent in cold-water exposure and bradycardia occurs during apnea. The question is whether abnormal heart responses pose any problems or potential limits for humans during diving. By and large, cardiac arrhythmias are rare in laboratory experiments. In the field however, there seems to be a high frequency of arrhythmias in association with immersion and BH (Hong *et al.*, 1967; Sasamoto, 1965; Scholander *et al.*, 1962). Diving bradycardia is commonly observed in the laboratory as well as in the field (Hong, 1987; Song *et al.*, 1969; Lin *et al.*, 1983a). Heart rate can be alarmingly low at times, but fatality due to these extreme bradycardias is not substantiated.

A. Head-Out Immersion

Heart rate changes little during immersion in thermoneutral water. Review of 13 reports involving 100 male subjects indicates it is either unchanged or decreases only slightly (Lin, 1984), although cold water depresses it (Keatinge and Evans, 1961; Knight and Horvath, 1987, Rennie *et al.*, 1971). Several opposing factors act together to determine heart rate during immersion. Other factors being equal, the rising central blood volume and pressure should trigger a tachycardial response through the Bainbridge reflex, but concurrent elevation of arterial pressure, by the increased stroke volume and cardiac output, acts against an increase in heart rate. Cold water reduces heart rate, but increased metabolic demands during diving (staying afloat in place, replacing heat

loss, and exercise) cause it to rise. The pre-dive heart rate in open sea is higher than the resting value in air (Hong *et al.*, 1967; Irving, 1963; Scho-lander *et al.*, 1962).

B. Submersion

The phenomenon of *diving bradycardia* is by far the most frequently studied of circulatory changes, both in the field and in the laboratory. Bradycardia develops promptly upon BH in humans but is slow to reach its lowest point (Lin *et al.*, 1983a). In comparison, maximal bradycardia is attained within a few cardiac cycles following the onset of apnea in diving mammals (Elsner, 1968), as well as nondiving mammals such as dogs (Elsner *et al.*, 1966; Lin, 1983b; Lin *et al.*, 1983c) and rats (Lin, 1974; Lin and Baker, 1975).

Fig. 3 summarizes a survey of breath-hold induced bradycardia in humans, which may be very intense in some instances. It is very evident bradycardia occurs during BH both at rest and during exercise. The response is greatly increased in humans exercising while holding their breath. Those indicated by an "a" in Fig. 3 are single subject records. Bradycardia is dramatized during underwater swimming. Jung and Stolle (1981) demonstrated a swimmer's heart rate levels off at 55 beats/minute during a 50-m underwater swim. Such activity requires between 5 to 10 times the resting oxygen consumption. The heart rate would have been about 180 beats/min on land. In some cases, the decrease may be as much as 90% of the pre-BH heart rate (Fig. 3, Table 6). A sinus bradycardia of 5.6 beat/min was recently reported by Arnold (1985) in a man BH with face immersion in ice water. When this point was added to the previously published figure (Lin, 1982), it fits on the line of a 90% reduction in heart rate (lower left-hand corner in Fig. 3).

Surprisingly, no discomfort or unusual sensation was reported by these subjects with extreme bradycardia, though the low heart rate persisted for more than 20 sec (Arnold, 1985). It is unlikely either diving-induced cardiac arrhythmias or BH sinus bradycardia limits human activity underwater. However, there is a cautionary note put forward after diving reflex bradycardia was suspected in the death of an elderly man while he was washing his face (Wolf, 1964). The relevance of this type of abnormal response to diving is yet to be assessed.

Elevation of vagal tone is responsible for the diving bradycardia in humans just as in aquatic mammals, as was deduced by differential autonomic nervous blockade (Berk and Levy, 1977; Finley *et al.*, 1979; Heistad *et al.*, 1968), while the sympathetic branch of the autonomic nervous system plays a minor role. However, more convincing evidence

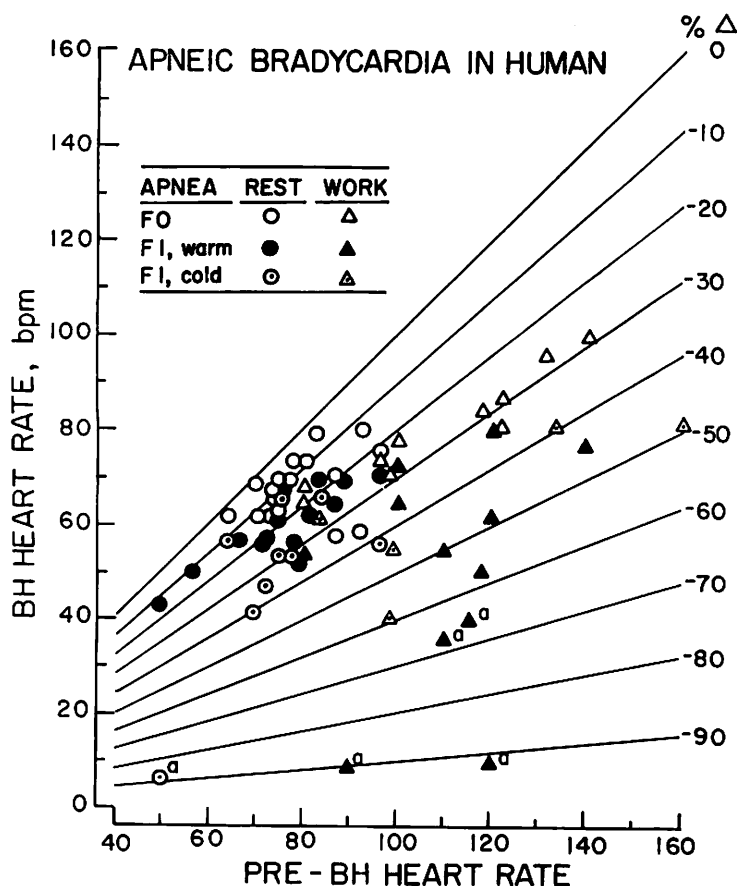


FIG. 3. Bradycardial response in humans at rest and during exercise (modified from Lin, 1982).

is derived from animal data, both in diving species (Andersen, 1966; Murchaugh *et al.*, 1961) and nondiving species (Gooden *et al.*, 1974; Lin, 1974; Lin *et al.*, 1983c).

There may be a combination of conditions which will favor more severe levels of bradycardia. As seen in Table 6, the greatest reduction in HR may occur when exercise is combined with cold water. It is known that diving bradycardia is mediated via the vagus nerve. This cardiodepressor

TABLE 6.

Diving Bradycardia in Man

| WT ^a °C | Heart Rate (b/min) | Conditions | Reference |
|-----------------------|-----------------------|------------------|---------------------------------|
| — | 25 | Swimming | Irving, 1963 |
| 0 | 25 | Rest | Whayne and Killip, 1967 |
| 25 | 24 | Rest | Landsberg, 1975 |
| 15 | 22 | Rest | Kawakami <i>et al.</i> , 1967 |
| — | 20 | Rest | Ryan <i>et al.</i> , 1976 |
| — | 20 | Shell harvesting | Scholander <i>et al.</i> , 1962 |
| 25 | 17 | Bicycling | Stromme and Blix, 1976 |
| — | 13 | Resting | Elsner <i>et al.</i> , 1966 |
| — | 13 | Resting | Scholander, 1963 |
| — | 12 | Resting | Elsner, 1963 |
| 10 | 10 | Resting | Hughes <i>et al.</i> , 1978 |
| — | 10 | Exercising | Frey and Kenney, 1974 |
| 10 | 10 | Swimming | Asmussen and Kristiansson, 1968 |
| — | 9.4 | Climbing stairs | Frey and Kenney, 1977 |
| — | 8.8 | Swimming | Stromme <i>et al.</i> , 1970 |
| 1 | 5.6 | Resting | Arnold, 1985 |

^a Water temperature

activity on the heart is enhanced when there is an elevated background level of sympathetic stimulation (Levy, 1971). It is reasonable to assume this so-called "accentuated antagonism" is present during BH diving as the circulating catecholamine level is elevated during cold exposure and during exercise.

VIII. SUMMARY

In comparison to marine mammals, humans are poor divers because of severe physiological constraints which limit breath-hold time, diving depth, and ability to conserve body heat. Physiological limits of humans as breath-hold (BH) divers are grossly underestimated, leading to premature abandoning of resuscitation efforts in drowning cases. Therefore, understanding human limitations, or survivability, is of paramount importance, both in medical teaching and clinical practice.

Diving Time. Theoretically, BH time can be predicted from O_2 supply-demand relationships. Based on the O_2 supply in the lung, a BH time of 4 min with air and 16 min with O_2 should be possible. Laboratory results proved these predictions correct in humans at rest. Breath-holding time can be substantially lengthened by prior hyperventilation. A student at Wesleyan University held his breath with O_2 for 20.1 min after hyperventilation. Furthermore, humans can survive lengthy apnea when it is coupled with hypothermia, such as in cold-water drowning.

Diving Depth. Just as humans can achieve an astonishingly long BH time, they also can reach great depths with a single breath. Although it was predicted that the depth limit for humans should be about equal to the TLC/RV ration (in atm), in the range of 30 to 40 m, this calculation falls far short of attained depths because the TLC/RV ratio increases during diving. In Craig's review, Croft holds the record at 64.8 m. The prediction is exceeded because RV shrinks during diving by the chestward displacement of blood. Over the years, depth records have escalated. By 1976, Mayol reached 86 m, or 284 ft, with a single breath. Maiorca recorded 878 m in 1974 and his latest record is 94 m. An unofficial record of 105 m (more than 340 ft) was said to have been made by Mayol in 1983.

Hypothermia. The thermoneutral water temperature for humans is 35°C which is substantially higher than that of ocean water even in mid-summer. Thermal drain continues as long as a person is immersed. Korean professional BH divers stop harvesting when body temperature falls to 35°C , thus avoiding the onset of potentially life-threatening functional deterioration while in water. However, just as diving time and depth can be surprising, humans can survive astonishingly low body temperature (24°C), under unusual circumstances.

Diving Reflex and Near Drowning. It is important to note diving reflex causes apnea when a person is accidentally submerged, preventing inhalation of water. This, in addition to the cardiovascular response that follows, enhances the chance of revival, even after hopelessly long apnea. The odds of survival improve when conditions favor a quick chilling of the body. Hypothermia reduces demand for O_2 , and redistribution of blood flow minimizes damage to the brain and heart. In addition, apnea-induced bradycardia (as low as 6 beats/min) during submergence further protects the heart from hypoxic damage. We have now learned successful revival is possible after apnea of 40 min or longer. In light of this, aggressive CPR should always be practiced in an attempt to revive drowning victims, especially in cases involving cold water.

ACKNOWLEDGEMENTS

This study was supported in part by the U.S. Department of Commerce, Office of Sea Grant, NA85AA-D-SG082 R/HP-5 and M/PM-2, and the Pioneer Imin Cultural Foundation of Hawaii. The assistance of Dr. K.K. Shida Pang and Mr. M. Sanders in the preparation of this manuscript is gratefully acknowledged.

REFERENCES

- Agostcni, E. Diaphragm activity during breath-holding: Factors related to its onset. *J. Appl. Physiol.* 8:30-36, 1963.
- Andersen, H.T. Physiological adaptations in diving vertebrates. *Physiol. Rev.* 46:212-243, 1966.
- Andersen, P.K. and D. Theilade. Survival after submersion for 25 minutes. *Ugeskr. Laeg.* 140:1620-1621, 1978.
- Arnold, R.W. Extremes in human breath-hold, facial immersion bradycardia. *Undersea Biomed. Res.* 12:183-190, 1985.
- Asmussen, E. and N.G. Kristiansson. The "diving bradycardia" in exercising man. *Acta Physiol. Scand.* 73:527-535, 1968.
- Berk, J.L. and M.N. Levy. Profound reflex bradycardia produced by transient hypoxia or hypercapnia in man. *Eur. Surg. Res.* 9:75-84, 1977.
- Craig, A.B. Causes of loss of consciousness during underwater swimming. *J. Appl. Physiol.* 16:583-586, 1961a.
- Craig, A.B. Underwater swimming and loss of consciousness. *J. Am. Med. Assoc.* 176:255-258, 1961b.
- Craig, A.B. Depth limits of breath-hold diving. *Respir. Physiol.* 5:14-22, 1968.
- Craig, A.B. Summary of 58 cases of loss of consciousness during underwater swimming and diving. *Med. Sci. Sports* 8:171-175, 1976.
- Cross, E.R. Taravana. Diving syndrome in the Tuamotu diver. In: H. Rahn and T. Yokoyama, eds. *Physiology of Breath-Hold Diving and the Ama of Japan*. Washington, D.C.: Nat. Acad. Sci., publ. #1341, p. 207-219, 1965.
- Daviss, B. Cold water to the rescue. *Science* 85, p. 72, 1985.
- Dominquez De Villota, E., G. Barat, P. Peral, A. Juffe, J.M. Fernandez De Miguel, and F. Avello. Recovery from profound hypothermia with cardiac arrest after immersion. *Brit. Med. J.* 4:394-395, 1973.
- Douglas, C.G. and J.S. Haldane. The regulation of normal breathing. *J. Physiol.* 38:420-440, 1909.
- Elliott, J.C. New hope for cold-water drowning victims. *NOAA* 8:46-47, 1978.
- Elsner, R. Reduced limb blood flow in man during breath hold dives. *Fed. Proc.* 22:179, 1963.
- Elsner, R. Cardiovascular adjustments to diving. In: H.T. Andersen, ed. *The Biology of Marine Mammals*. New York, NY: Academic press. p. 117-145, 1968.
- Elsner, R., D.L. Franklin, R. Van Citters, and D.W. Kenney. Cardiovascular defense against asphyxia. *Science* 153:941-949, 1966.
- Frey, M.A. and R.A. Kenney. Systolic time intervals during face immersion bradycardia. *Fed. Proc.* 33:327, 1974.
- Frey, M.A. and R.A. Kenney. Changes in left ventricular activity during apnea and face immersion. *Undersea Biomed. Res.* 4:27-37, 1977.

- Finley, J.P., J.F. Bonet, and M.B. Waxman. Autonomic pathways responsible for bradycardia on facial immersion. *J. Appl. Physiol.* 47:1218-1222, 1979.
- Godfrey, S., and E.J.M. Campbell. The control of breath holding. *Resp. Physiol.*, 5:385-400, 1968.
- Gooden, B.A., H.L. Stone, and S. Young. Cardiac responses to snout immersion in trained dogs. *J. Physiol.* 242:405-415, 1974.
- Guinness Book of World Records*. N. McWhiter *et al.*, eds. New York, NY: Sterling Publ. Co., Inc. p. 31, 1985.
- Guinness Book of World Records*. D. McFarlan *et al.*, eds. New York, NY: Sterling Publ. Co., p. 30, 1989.
- Hayward, J.S., C. Hay, B.R. Mathews, C.H. Overwhiel, and D.D. Radford. Temperature effects on the human dive response in relation to cold-water near drowning. *J. Appl. Physiol.* 56:202-206, 1984.
- Heistad, D.D., F.M. Abboud, and J.W. Eckstein. Vasoconstrictor response to simulated diving in man. *J. Appl. Physiol.* 25:542-549, 1968.
- Hill, L. and M. Flack. The effect of excess carbon dioxide and want of oxygen upon the respiration and the circulation. *J. Physiol.* 37:77-111, 1908.
- Hong, S.K. Hae-Nyo, the diving women of Korea. In: H. Rahn and T. Yokoyama, eds. *Physiology of Breath-Hold Diving and the Ama of Japan*. Washington, DC: Nat. Acad. Sci., publ. #1341, p. 99-111, 1965.
- Hong, S.K. The physiology of breath-hold diving. In: R.H. Strauss, ed. *Diving Medicine*, New York, NY: Grune and Stratton, p. 269-286, 1976.
- Hong, S.K. Breath-hold bradycardia in man: an overview. In: C.E.G. Lundgren and M. Ferrigno, eds. *The Physiology of Breath-hold Diving*. Bethesda, MD: Undersea & Hyperbaric Med. Soc. p. 158-171, 1987.
- Hong, S.K., H. Rahn, D.H. Kang, S.H. Song, and B.S. Kang. Diving pattern, lung volumes, and alveolar gas of the Korean diving woman (ama). *J. Appl. Physiol.* 18:457-465, 1963.
- Hong, S.K. and H. Rahn. The diving women of Korea and Japan. *Sci. Am.* 216:34-43, 1967.
- Hong, S.K., S.H. Song, P.K. Kim, and C.S. Suh. Seasonal observations on the cardiac rhythm during diving in the Korean ama. *J. Appl. Physiol.* 23:18-22, 1967.
- Hong, S.K., T.O. Moore, G. Seto, H.K. Park, W.R. Hiatt, and E.M. Bernauer. Lung volume and apneic bradycardia in divers. *J. Appl. Physiol.* 29:172-176, 1970.
- Hong, S.K. Y.C. Lin, D.A. Lally, B.J.B. Yim, N. Kominami, P.W. Hong, and T.O. Moore. Alveolar gas exchanges and cardiovascular functions during breath-holding with air. *J. Appl. Physiol.* 30:540-547, 1971.
- Hughes, T., J. Carter, and S. Wolf. Changes in cardiac rate, rhythm, and conduction in man during dive reflex. *Clin. Res.* 26:A548, 1978.
- Hunt, N.G. D.K. Whitaker, and N.J. Willmott. Water temperature and the "diving reflex". *Lancet* 1:972, 1975.
- Irving, L. Bradycardia in human divers. *J. Appl. Physiol.* 18:489-491, 1963.
- Jaklewicz, H., D. Lipinska, W. Rynkowska, M. Bogdanowicz and B. Lewandowska. Compensatory possibilities of the central nervous system: a case of "near drowning" in a 15-year-old boy. *Psychiatr. Pol.* 14:303-306, 1980.
- Jung, K. and W. Stolle. Behavior of heart rate and incidence of arrhythmia in swimming and diving. *Biotelem. Pat. Monitg.* 8:228-239, 1981.
- Kang, D.H., P.K. Kim, B.S. Kang, S.H. Song, and S.K. Hong. Energy metabolism and body temperature of the ama. *J. Appl. Physiol.* 20:46-50, 1965.
- Kang, D.H., Y.S. Park, I.S. Lee, D.S. Yeon, S.H. Lee, Y.S. Hong, D.W. Rennie, and S.K. Hong. Energetics of wet-suit diving in Korean women divers. *J. Appl. Physiol.* 54:1702-1707, 1983.

- Kawakami, Y., B.H. Natelson, and A.B. DuBois. Cardiovascular effect of face immersion and factors affecting diving reflex in man. *J. Appl. Physiol.* 23:964-970, 1967.
- Keatinge, W.R. The effect of work and clothing on the maintenance of body temperature in water. *Q'ty J. Expt'l. Physiol.* 46:9-82, 1961.
- Keatinge, W.R. *Survival in Cold Water*. Oxford, England: Blackwell Scientific Publ. 1969.
- Keatinge, W.R. and M. Evans. The respiratory and cardiovascular response to immersion in cold and warm water. *Q'ty J. Expt'l Physiol.* 46:83-94, 1961.
- Kelman, G.R. and K.T. Wann. Mechanical and chemical control of breath holding. *Q'ty J. Expt'l Physiol.* 56:92-100, 1971.
- King, R.B. and I.W. Webster. A case of recovery from drowning and prolonged anoxia. *Med. J. Australia* 1:919-920, 1964.
- Klocke, F.J., and H. Rahn. Breath-holding after breathing of oxygen. *J. Appl. Physiol.* 14:689-693, 1959.
- Knight, D.R. and S.M. Horvath. Effect of hydrostatic pressure on plasma concentrations of norepinephrine during cold water immersion. *Undersea Biomed. Res.* 14:1-10, 1987.
- Kobayashi, S. and C. Sasaki. Breaking point of breath holding and tolerance time in re-breathing. *Japn. J. Physiol.* 17:43-56, 1967.
- Kvittingen, T.D. and A. Naess. Recovery from drowning in fresh water. *Brit. Med. J.* 1:1315-1317, 1963.
- Lanphier, E.H. and H. Rahn. Alveolar gas exchange during breath-hold diving. *J. Appl. Physiol.* 18:471-477, 1963.
- Landsberg, P.G. Bradycardia during human diving. *S. Afr. Med. J.* 49:626-630, 1975.
- Lamb, L.E., G. Dermksian, and C.A. Sarnoff. Significant cardiac arrhythmia induced by common respiratory maneuvers. *Am. J. Cardiol.* 2:563-571, 1958.
- Levy, M.N. Cardiac sympathetic-parasympathetic interactions. *Federation Proc.* 43:2598-2602, 1984.
- Lin, Y.C. Autonomic nervous control of cardiovascular response during diving in the rat. *Am. J. Physiol.* 227:601-605, 1974.
- Lin, Y.C. Breath-hold diving in terrestrial mammals. In: R.L. Terjung, ed. *Exercise and Sport Science Reviews*, Vol. 10. Philadelphia, PA: Franklin Press. p. 270-307, 1982.
- Lin, Y.C. Cardiopulmonary physiology of nondiving mammals during breath-hold dives. In: K. Shiraki and S. Matsuoka, eds. *Hyperbaric Medicine and Underwater Physiology*. Kitakyushu, Japan: Univ. of Occupational and Environmental Health, p. 25-35, 1983a.
- Lin, Y.C. Hemodynamic changes during voluntary snout immersion in chronically instrumented dogs. *Chinese J. Physiol.* 26:1-10, 1983b.
- Lin, Y.C. Circulatory functions during immersion and breath-hold dives in humans. *Undersea Biomed. Res.* 11:123-138, 1984.
- Lin, Y.C. Breath-hold diving: human imitation of aquatic mammals. In: A.O. Brubakk, J.W. Kanwisher, and G. Sundnes. *Diving in Mammals and Man*. Trondheim, Norway: Tapir Publ. p. 81-89, 1986.
- Lin, Y.C. Effect of O₂ and CO₂ on breath-hold breaking point. In: C.E.G. Lundgren and M. Ferrigno, eds. *The Physiology of Breath-Hold Diving*. Bethesda, MD: Undersea & Hyperbaric Med. Soc. p. 75-86, 1987a.
- Lin, Y.C. Human imitation of marine mammals and its clinical significance. In: A.A. Bore, A.J. Bachrach and L.J. Greenbaum, eds. *Underwater Physiology IX*. Bethesda, MD: Undersea and Hyperbaric Med. Soc., p. 29-45, 1987b.
- Lin, Y.C. Applied physiology of diving. *Sports Med.* 5:41-56, 1988.
- Lin, Y.C., D.A. Lally, T.O. Moore, and S.K. Hong. Physiological and conventional breath-hold breaking points. *J. Appl. Physiol.* 37:391-296, 1974.

- Lin, Y.C. and D.G. Baker. Cardiac output and its distribution during diving in the rat. *Am. J. Physiol.* 228:733-737, 1975.
- Lin, Y.C., T.O. Moore, J.J. McNamara, and S.K. Hong. O₂ consumption and conservation during apnea in the anesthetized dog. *Respir. Physiol.* 24:313-324, 1975.
- Lin, Y.C., K.K. Shida, and S.K. Hong. Effects of hypercapnia, hypoxia, and rebreathing on heart rate response during apnea. *J. Appl. Physiol.* 54:166-171, 1983a.
- Lin, Y.C., K.K. Shida, and S.K. Hong. Effect of hypercapnia, hypoxia, and rebreathing on circulatory response to apnea. *J. Appl. Physiol.* 54:172-177, 1983b.
- Lin, Y.C., E.L. Carlson, E.P. McCutcheon, and H. Sandler. Cardiovascular functions during voluntary apnea in dogs. *Am. J. Physiol.* 245:R143-R150, 1983c.
- Maiorca, E. Depth records: practical considerations. In: C.E.G. Lundgren and M. Ferrigno, eds. *The Physiology of Breath-hold Diving*. Bethesda, MD: Undersea and Hyperbaric Med. Soc. p. 291-294, 1987.
- Martin, T.G. Near-drowning and cold water immersion. *Ann. Emerg. Med.* 13:263-273, 1984.
- Missiroli, F.M. and B. Rizzato, Mayol a 105 metri. *Mondo Sommerso*, No. 272, p. 32-37, 1984.
- Mithoefer, J.C., Breath-holding. In: W.O. Fenn and H. Rahn, eds. *Handbook of Physiology, Respiration II*. Washington, DC: Am. Physiol. Soc. p. 1011-1925, 1965.
- Murdaugh, H.V., J.C. Seabury, and W.L. Mitchell. Electrocardiogram of the diving seal. *Circulation Res.* 9:358-361, 1961.
- Nemiroff, M.J. The girl with everything. A diving incident. *South Pac. Undersea Med. Soc. J.* Jan.-Mar.:27-28, 1979.
- Ola, P. and E. D'Aulaire. Drama in real life: Chain of circumstance. *Reader's Digest*, p. 143-48, Dec. 1987.
- Park, Y.S., H. Rahn, I.S. Lee, S.I. Lee, D.H. Kang, S.Y. Hong, and S.K. Hong. Patterns of wet suit diving in Korean women breath-hold divers. *Undersea Biomed. Res.* 19:203-215, 1983a.
- Park, Y.S., D.W. Rennie, I.S. Lee, Y.D. Park, K.S. Paik, D.H. Hang, D.J. Suh, S.H. Lee, S.Y. Hong, and S.K. Hong. Time course of deacclimatization to cold water immersion in Korean women divers. *J. Appl. Physiol.* 54:1708-1716, 1983b.
- Park, Y.S. D.R. Pendergast, and D.W. Rennie. Decrease in body insulation with exercise in cool water. *Undersea Biomed. Res.* 11:159-168, 1984.
- Park, S.Y., K. Shiraki, and S.K. Hong. Energetics of breath-hold diving in Korean and Japanese professional divers. In: Y.C. Lin and K.K. Shida, eds. *Man in the Sea*. Best Publishing Co., Vol. II, p. 75-87, 1990.
- Paulev, P.E. Decompression sickness following repeated breath hold dives. *J. Appl. Physiol.* 20:1028-1031, 1965.
- Rahn, H. and T. Yokoyama, eds. *Physiology of Breath-Hold Diving and the Ama of Japan*. Washington, DC: Nat. Acad. Sci., Publ. #1341, 1965.
- Ryan, C.M. Hollenberg, D.B. Harvey, and R. Gwynn. Impaired parasympathetic responses in patients after myocardial infarction. *Am. J. Cardiol.* 37:1013-1018, 1976.
- Rennie, D.W., P. DiPrampo, and P. Cerretelli. Effect of water immersion on cardiac output, heart rate, and stroke volume of man at rest and during exercise. *Med. Sport.* 24:223-228, 1971.
- Rennie, D.W., Y.S. Park, A. Veicsteinas, and D. Pendergast. Metabolic and circulatory adaptation to cold water stress. In: D. Cerretelli and B. Whipp, eds. *Exercise Bioenergetics and Gas Exchange*. Amsterdam, Netherlands: Elsevier/North-Holland, p. 315-321, 1980.
- Ricci, G., and H. Marroni. Physiological observations during deep breath-hold diving. *Cinesiologie.* 60:187-193, 1976.

- Sasamoto, H. The electrocardiogram pattern of the diving ama. In: H. Rahn and T. Yokoyama, eds. *Physiology of Breath-Hold Diving and the Ama of Japan*. Washington, DC: Nat. Acad. Sci., Publ. #1341, p. 271-280, 1965.
- Schaefer, K.E., R.D. Allison, J.H. Dougherty, Jr., C.R. Carey, R. Walker, F. Yost, and D. Parker. Pulmonary and circulatory adjustments determining the limits of depths in breathhold diving. *Science*, 162:1020-1023, 1968.
- Schneider, E.C. Observations on holding the breath. *Am. J. Physiol.* 94:464-470, 1930.
- Scholander, P.F. The master switch of life. *Sci. Am.* 209:92-106, 1963.
- Scholander, P.F., H. Hammel, H. LeMessurier, E. Hemingsen, and W. Garey. Circulatory adjustment in pearl divers. *J. Appl. Physiol.* 17:184-190, 1962.
- Siebekke, H., T. Rod, H. Breivik, and B. Lind. Survival after 40 minutes' submersion without cerebral sequelae. *Lancet* 1:1275-1277, 1975.
- Shiraki, K., S. Sagawa, N. Konda, Y.S. Park, T. Komatsu, and S.K. Hong. Diving pattern of Tsushima male breath-hold divers (Katsugi). *Undersea Biomed. Res.* 13:439-452, 1985.
- Shiraki, K., S. Sagawa, N. Konda, Y.S. Park, T. Komatsu, and S.K. Hong. Energetics of wet-suit diving in Japanese male breath-hold divers. *J. Appl. Physiol.* 61:1475-1480, 1986.
- Song, S.H., W.K. Lee, Y.A. Chung, and S.K. Hong. Mechanism of apneic bradycardia in man. *J. Appl. Physiol.* 27:323-327, 1969.
- Sterba, J.A. and C.E.G. Lundgren. Diving bradycardia and breath-holding time in man. *Undersea Biomed. Res.* 12:139-150, 1985.
- Stromme, S.B. and A.B. Blix. Indirect evidence for arterial chemoreceptor reflex facilitation by face immersion in man. *Aviat. Space Environ. Med.* 47:597-599, 1976.
- Stromme, S.B., O. Kerem, and R. Elsner. Diving bradycardia during rest and exercise and its relation to physical fitness. *J. Appl. Physiol.* 28:614-621, 1970.
- Teruoka, G. Die ama und ihre arbeit. *Arbeitsphysiol.* 5:239-251, 1932.
- Triton. 3 mins 39 secs. *Triton* 22:1148-149, 1977a.
- Triton. More about that dive. *Triton* 22:201, 1977b.
- Veicsteinas, A. and D.W. Rennie. Thermal insulation and shivering threshold in Greek sponge divers. *J. Appl. Physiol. Respirat. Environ. Exercise Physiol.* 52:845-850, 1982.
- Webb, P. Thermal stress in undersea activity. In: C.J. Christiansen, ed. *Underwater Physiology V*. Bethesda, MD: FASEB, p. 705-724, 1976.
- Wayne, T.F. and T. Killip III. Simulated diving in man: comparison of facial stimuli and response in arrhythmia. *J. Appl. Physiol.* 22:800-807, 1967.
- White, P.D. Observations on some tests of physical fitness. *J. Med. Sci.* 159:866-874, 1920.
- Wolf, S. The bradycardia of the dive reflex: a possible mechanism of sudden death. *Trans. Am. Climat. Assoc.* 76:192-200, 1964.
- Yokoyama, T. Energy expenditure by the diving ama in Japan. In A.A. Bove, A.J. Bachrach, and L.J. Greenbaum, Jr., eds. *Underwater and Hyperbaric Med. Physiology IX*. Bethesda, MD: Undersea and Hyperbaric Med. Soc., p. 17-28, 1987.

3

Arterial Gas Tensions and Hemoglobin Concentrations of the Free Diving Antarctic Weddell Seal

Warren M. Zapol, Roger D. Hill, Jesper Qvist, Konrad Falke, Robert Schneider, Graham C. Liggins, and Peter W. Hochachka

| | |
|---|----|
| I. Introduction | 57 |
| II. Techniques | 59 |
| A. Flush Solution | 60 |
| B. Blood Sampling System | 60 |
| C. Nitrogen Tension Measurement | 61 |
| D. Hemoglobin and Hematocrit | 61 |
| III. Results | 62 |
| A. Arterial Hemoglobin | 62 |
| B. Arterial Oxygen Tensions | 63 |
| C. Arterial Nitrogen Tensions | 64 |
| IV. Discussion | 65 |
| A. Nitrogen | 65 |
| B. Hemoglobin | 67 |
| Acknowledgements | 70 |
| References | 70 |

I. INTRODUCTION

Diving physiology has interested scientists for over a century (Bert, 1870). The mammals and birds which dive to great depths for long periods to exploit food sources deep in the ocean have developed remarkable

evolutionary adaptations to optimize their diving ability. Some of the respiratory accommodations are obvious to casual inspection, such as a small lung volume to body size ratio, thoracic cage mobility and circular bronchial cartilages (Kooyman, 1981). Some respiratory and circulatory adaptations have been observed in the laboratory; captive seals have been forced to dive while monitored by invasive instrumentation (Zapol *et al.*, 1979) (Swan Ganz catheters, left ventricular catheters) or have been subjected to the pressure of depth (Kooyman *et al.*, 1972) in a hyperbaric chamber. However, it has been clear for over ten years that laboratory diving forces an abnormally profound diving reflex, including intense bradycardia and marked regional arterial vasoconstriction (Blix and Folkow, 1983; Bryden, 1971). This intense bradycardia is far slower than recorded in free swimming seals with an electrocardiogram (ECG) and breakoff leads (Kooyman and Campbell, 1972).

In 1976 and 1977 our research group at McMurdo Station (168°E, 77°S) studied the Antarctic Weddell seal (*Leptonychotes weddelli*), a champion pinniped diver, capable of diving over one hour to depths of over 500 meters (Kooyman, 1981). Our studies were performed in the Station's biological laboratory and we examined organ biochemistry (Hochachka *et al.*, 1977), regional blood flow with microspheres (Zapol *et al.*, 1979), and fetal physiology (Liggins *et al.*, 1980). Often during those laboratory studies we considered the importance of repeating the experiments in the field realizing that many measurements during forced diving might not reflect the values in freely exercising, free swimming seals, but we were sobered by the difficulties of monitoring heart rate and velocity, and of sampling the arterial blood of a free swimming Weddell seal. There were three obvious problems: pressure causing seawater to leak into instrumentation, since seawater pressure at 500 meters is 750 lbs/square inch, low temperature causing blood samples to freeze, McMurdo Sound has a nearly constant temperature of -1.9°C , and how to reliably retrieve blood samples or data records from a free swimming seal.

Each member of our team contributed toward solving these problems. The central developments were the design, construction and field testing of both a submersible 64K RAM microprocessor monitor and a blood sampling system by Hill (1986). Also important was the development of a safe anesthetic technique for arterial cannulation and a simple, percutaneous procedure for implanting a fetal ECG electrode requiring only local anesthesia. In 1978, Kooyman (1981) demonstrated for us a captive diving technique at McMurdo which was vital to our field research and allowed us to reliably recover both monitoring data and blood samples. This chapter will summarize a small segment of those results on diving oxygen

and nitrogen exchange which have been reported in greater detail elsewhere (Falke *et al.*, 1985; Qvist *et al.*, 1986).

II. TECHNIQUES

Adult male seals weighing 300-400 kg were selected from seal colonies on the annual ice near the shore of Ross Island, Antarctica, and sledged to a site at which two holes of one meter diameter had been drilled through the three-meter-thick annual sea ice of McMurdo Sound. The field site was chosen to be sufficiently distant from any natural ice cracks that an instrumented seal released at this site would be obliged to return to the drilled holes to breathe. During general anesthesia (consisting of intramuscular 0.3 mg/kg Ketamine for induction, followed by mask inhalation via a to-and-fro circuit of 1-4% halothane in oxygen), a small skin incision over a fore-flipper artery allowed us to place an aortic catheter (3.2 mm O.D.) via an arteriotomy. Following catheterization, ECG leads were placed subcutaneously in the equivalent of 'right arm' and 'left leg' positions. In the 16 to 24 hours allowed for recovery from anesthesia, two neoprene patches were glued to the seal's dorsal fur with a fast-setting cyanoacrylate adhesive (Loctite 422). A microcomputer monitor and blood sampler, both described in detail elsewhere (Hill, 1986) were bolted to these patches and connected to the ECG electrodes and aortic catheter, respectively.

The monitor sampled and stored records of heart rate, depth, swimming activity and aortic blood temperature, and controlled the blood sampling roller pump. Either a single or a set of sequential blood samples could be initiated at any part of the dive by appropriate programming of the microcomputer monitor.

After full recovery from general anesthesia, the instrumented seal was released into the water through one of the drilled holes, which was subsequently blocked. A portable fish hut, with a hole in its floor, was placed over the other hole to act as a field laboratory. The seal was thus forced to surface inside the field laboratory between dives to breathe. The laboratory contained a small computer (Zenith Z-90 with 192 KBytes of memory and two 640-KByte floppy disk drives) to which physiological and environmental data were transmitted through fiber-optic cables during a brief connection when the seal surfaced. At this time the instructions for obtaining the next blood sample were transmitted from the laboratory computer to the microcomputer monitor.

At the conclusion of an experiment the second hole was re-opened, and when the seal hauled out onto the ice the aortic catheter and ECG leads were removed and the monitor and blood sampler were removed. Finally the seal was sledged back to its original capture site and released.

A. Flush Solution

Clotting was inhibited by flushing all the blood collection lines (and the aortic catheter) with a solution containing heparin (100 U/ml), and bonding heparin to the inside surfaces of the sample bags and collection lines (TDMAC) (Leininger *et al.*, 1966). Freezing was a problem because the normal saline flush solution froze after several hours immersion in seawater. Blood samples, however, did not freeze for up to an hour. A concentrated (5 M) sodium chloride solution was added to the normal saline in the flush bag to raise its salinity to 0.6 M. Since contamination of blood samples with the high molarity flush solution rendered them useless for P_{O_2} , P_{CO_2} , hematocrit and pH analysis, it was essential to detect inadvertent dilution with flush solution. Hematocrit measurements provided the most useful field indicator of dilution as the high osmolarity of flush solution caused dehydration of the red blood cells and a concomitant increase of the ratio of hemoglobin to hematocrit. The plasma sodium concentration of the blood samples was measured after returning from Antarctica and those samples with an elevated sodium concentration were not included in the gas tension analysis.

B. Blood Sampling System

Arterial blood was sampled at predetermined depths during descent, ascent or after certain dive durations had elapsed. Blood was collected with two different techniques (Hill, 1986) each with its own advantages. One technique provided a 50 ml blood sample which was used for biochemical sampling and arterial P_{N_2} measurements (Falke *et al.*, 1985). Twenty-two single arterial samples were obtained using this technique. A second blood collecting system provided up to eight sequential blood samples during a single dive. This device, described by Hill (1986), collected blood sequentially into a series of nine 10-ml syringes. The first syringe was filled with flush solution from the sample line and was discarded. The optimum pumping speed for uninterrupted serial sampling was found to be 20 ml/min, or 30 sec for each 10-ml syringe and produced eight sequential samples. When sampling was discontinuous, for example with a 5-min collection interval, two syringes were filled every 5 min. Blood remaining in the sample line filled the first of the two syringes and was discarded. This blood collection protocol reduced the number of useful samples but provided four undiluted blood samples drawn at flexible intervals during a single dive. With this technique 31 blood samples were drawn during nine free dives of up to 37-min duration.

An insulated field hut was erected on the sea ice near the diving hut to provide a stable thermal environment. P_{aO_2} , P_{aCO_2} and pH_a were measured and recorded at 37°C with an automated, self-calibrating blood gas laboratory (ABL-30, Radiometer, Copenhagen), barometric pressure was frequently recorded. As aortic temperatures during diving were near 37°C (Hill *et al.*, 1987) we report all blood gas tensions at this temperature for consistency. Hemoglobin measurements were made with another automated and frequently calibrated device (OSM-2, Radiometer). All data presented are mean \pm S.D.

C. Nitrogen Tension Measurement

Five ml of each blood sample was transferred to a glass syringe and, within two hours of sampling, injected into a Van Slyke apparatus for the determination of inert gas content (Klocke and Rahn, 1961). The few blood samples that could not be analyzed immediately were stored at 0°C. Serial determinations of inert gases from single samples revealed a maximum loss of inert gas content of 8 to 10 percent after two hours, presumably due to diffusion of nitrogen through the 1.0-mm thick sampling bag wall.

The solubility of inert gases in seal blood was determined as a function of the hemoglobin concentration by tonometry at 37°C with mixtures of air and CO₂ containing a known inert gas partial pressure. The solubility coefficient (α) for inert atmospheric gases (97.4 percent N₂ and 2.3 percent argon) at 37° in Weddell seal blood varies with the hemoglobin concentration (Hb) in grams per 100 ml of blood according to the linear regression: $y = 0.01126 + 0.00017 \text{ Hb}$ (standard error of the slope, 3.3×10^{-5} , $n = 15$). After appropriate correction for hemoglobin concentration and dilution by flushing fluid, the inert gas tensions of arterial blood (that is, the nitrogen tension P_{aN_2}) were calculated.

D. Hemoglobin and Hematocrit

Dives were partitioned into short duration (< 17 min, probably feeding dives) and longer periods (> 17 min, probably exploratory dives) based upon finding a normal post-dive arterial pH (pH_a) and arterial base excess (BE_a) value in all dives of less than 17 min duration, whereas all dives of 17 min duration or greater had significant decreases of pH_a and BE_a and an increased blood lactate in the early surface recovery period.

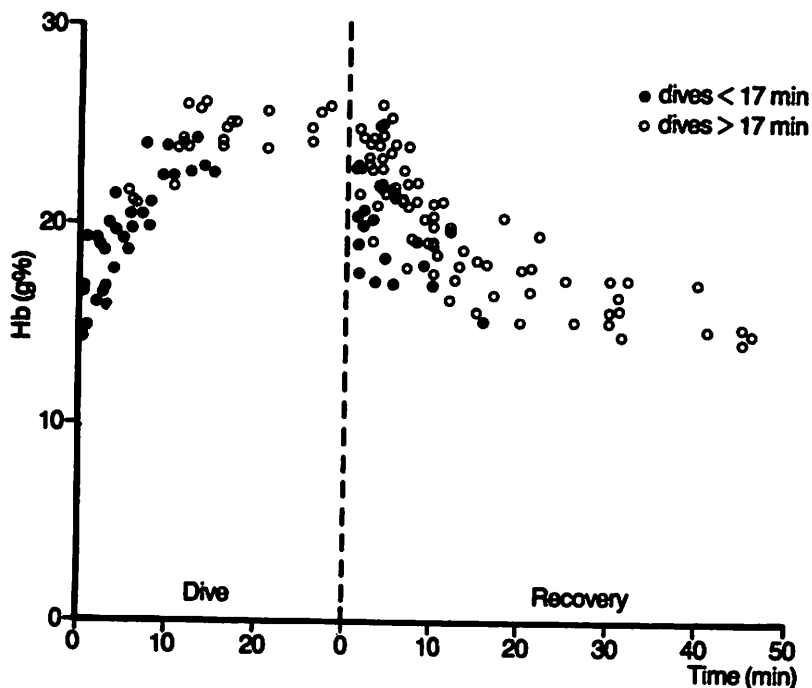


FIG. 1. Arterial hemoglobin changes during diving and after resurfacing. Dives were divided into short (<17 min) and long dives (>17 min). Serial blood sampling during long dives showed that Hb concentration stabilized within 10-12 min. The rate of rise of Hb was 0.85 g% per min per dl during the first 10 min. The rate of decrease during recovery was similar.

III. RESULTS

A. Arterial Hemoglobin

The 53 arterial hemoglobin concentration measurements obtained in 32 dives are displayed in Fig. 1. During diving the arterial hemoglobin concentration (Hb) increased rapidly from a resting value of 15.1 ± 1.1 g% (mean \pm SD). The rate of hemoglobin increase was approximately $0.85 \text{ g\%} \times \text{min}^{-1}$ during the first 10 min of diving in all dives. This remarkably consistent increase in arterial hemoglobin concentration was accompanied by an increase of the arterial hematocrit (Hct), from 38 ± 3 to approximately 55%. After 10-15 min of diving there was little further increase of Hb. The highest values of Hb and Hct were sampled late

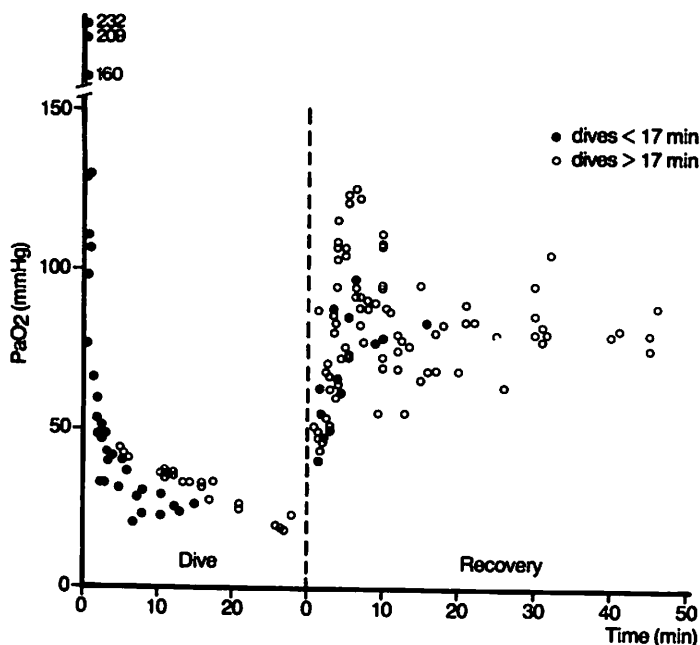


FIG. 2. Changes of arterial oxygen tensions (PaO_2) during diving and after resurfacing. Early diving compression hyperoxia is apparent. The lowest PaO_2 recorded was 18.2 mm Hg at the end of a 27 min dive. Similar low PaO_2 values were recorded at the end of short dives, i.e. dives that ended before 17 min. The highest post-dive PaO_2 values were recorded after dives of long duration.

during long dives: after a dive of 26.3 min the Hb and Hct were 25.4 g% and 59.3% respectively. Both values were significantly higher ($p < 0.01$) than the values obtained late in dives lasting less than 17 min. The Hb and Hct returned to their resting values within 12-16 min after surfacing if the seal did not immediately undertake a further long dive.

B. Arterial Oxygen Tensions

The 55 arterial PO_2 (PaO_2) measurements we obtained from 33 dives are presented in Fig. 2. The surface PaO_2 ranged from 56 to 100 mm Hg (78.1 ± 12.9 mm Hg) with the highest values recorded during brief resting periods between sequential dives. Blood samples that were collected

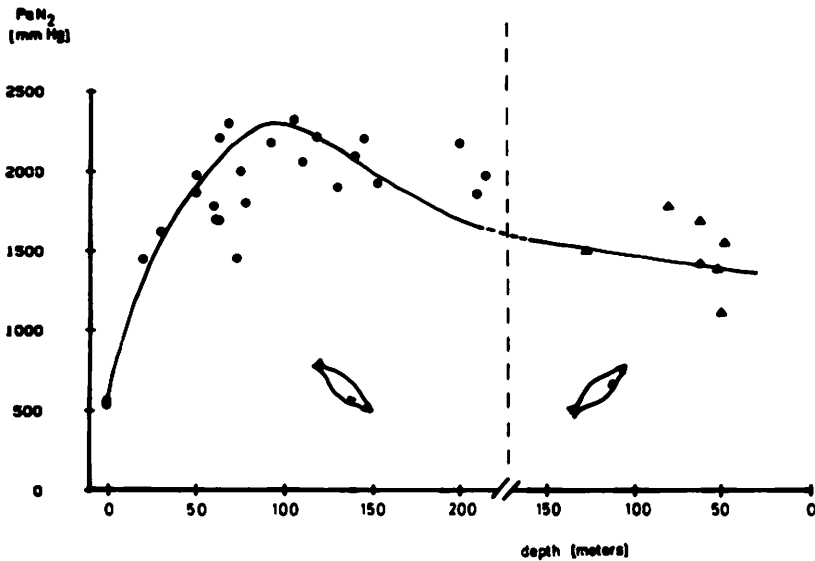


FIG. 3. Arterial blood nitrogen tensions (P_{aN_2}) in free-diving Weddell seals. Each 90-second sample was obtained during a single dive. P_{aN_2} is plotted at the mean depth of the sampling period over various depth ranges, depending upon the seal's rate of descent or ascent.

within the first minute of descent gave oxygen tensions as high as 232 mm Hg, well above the atmospheric P_{O_2} of 130-140 mm Hg (atmospheric pressure was 718 to 740 mm Hg) evidencing compression hyperoxia. In seven short dives (8 to 17 min duration) the P_{aO_2} of blood sampled up to 2 min before surfacing was as low as 20.4 mm Hg (mean 24.5 ± 2.9 mm Hg). During these short dives to depths of 200-300 m the heart rate (HR) at sampling time was high (average 40 bpm, range 30-65). The lowest P_{aO_2} value we measured was 18.2 mm Hg (28% saturation) in a blood sample obtained 1 min before the conclusion of a 27-min dive.

C. Arterial Nitrogen Tensions

Values of P_{aN_2} from 29 blood samples obtained during dives by four seals (Fig. 3) show that by the time the seal has descended to 50 to 70 m, the P_{aN_2} peaked at approximately 2300 mm Hg, then decreased to a range of 1100 to 1800 mm Hg as the dive progressed. A comparison of early (<7 minutes) with later (7 to 23 minutes) P_{aN_2} values revealed a statistically significant decline (early, 1983 ± 51 mm Hg, $n=19$; late 1521 ± 76 mm Hg, $n=8$).

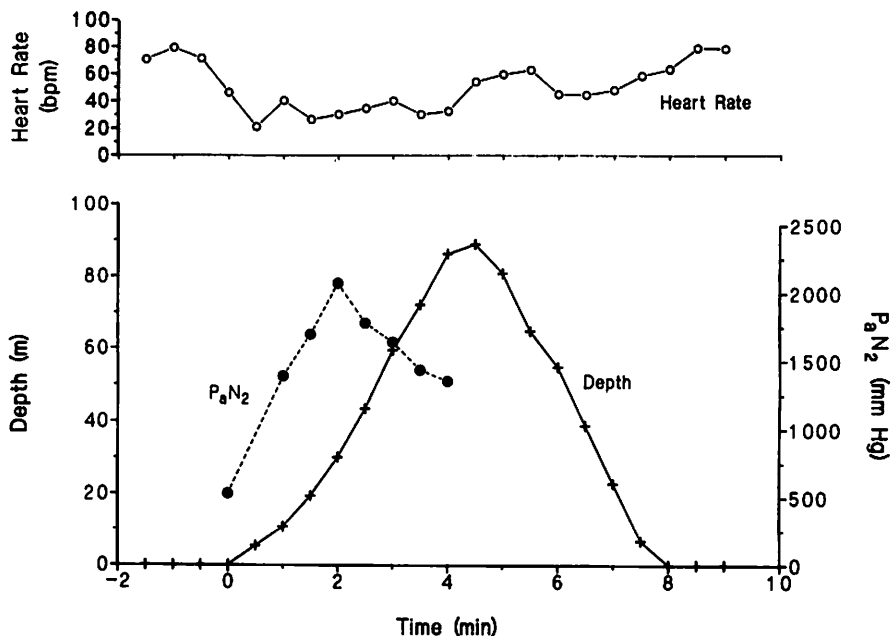


FIG. 4. Heart rate and depth combined with serial determinations of P_{aN_2} . The sampling time was 30 seconds for each sample. P_{aN_2} values are early during a dive when pulmonary gas exchange spaces are collapsing.

Sequential 30-second blood samples taken together with heart rate and depth during single dives show that the maximum P_{aN_2} of 2079 mm Hg at approximately 30 m (Fig. 4) was followed by a sharp decline of P_{aN_2} to 1200 mm Hg during further descent to a maximum depth of 89 m. P_{aN_2} continues to fall during descent, and a greater diving depth was not associated with a higher P_{aN_2} value. Three sets of four sequential samples taken during late phases of dives showed declining P_{aN_2} values between 1,700 and 1,200 mm Hg.

IV. DISCUSSION

A. Nitrogen

Our results provide direct evidence that the free-swimming Weddell seal protects itself from nitrogen narcosis and decompression sickness by both limiting N_2 uptake from the lungs and redistributing N_2 during the dive. Nitrogen uptake is effectively stopped by the collapse of gas-exchanging alveoli, and the maximum P_{aN_2} is then reduced by redistribution of

N₂ to organs and tissues. Shunting of venous blood through collapsed alveoli will also reduce the peak P_{AN_2} . The peak P_{AN_2} of 2,079 mm Hg (Fig. 4) indicates alveolar collapse occurs at approximately 28 m (3.7 atmospheres absolute multiplied by 550 mm Hg). However, because of the 30-second sampling period, the P_{AN_2} values represent averages of blood sampled at depths ranging up to 35 m taking into account the Weddell seals' maximum observed rate of descent or ascent of 70 m/min. Our observation that the P_{AN_2} in samples drawn between 200 and 230 m (21 to 24 atmospheres absolute) never rose above 2,329 mm Hg, explains how the seal escapes nitrogen narcosis during deep diving. The P_{AN_2} does not rise to levels that produce narcosis.

Seals usually exhale before diving. Kooyman *et al.* measured the average lung volume during diving of adult Weddell seals to be 11.6 liters ($n=4$), of which 9.2 liters was N₂ (Kooyman *et al.*, 1971). Assuming that perfusion during free diving is similar to that in forced diving and is restricted to vital organs, such as the brain, heart, and lungs, only approximately 7.5 kg of tissues and up to 56 liters of blood are available for N₂ distribution in the 350-kg diving Weddell seal (Zapol *et al.*, 1979). Thus, the peak P_{AN_2} of 2,400 mm Hg corresponds to absorption of only 2.5 liters of N₂ from the lungs. This provides further evidence that during diving the major fraction of the Weddell seal's lung gas is compressed into non-gas exchanging segments of the respiratory tract. At the end of the dive this residual gas can expand to open collapsed alveoli.

The decline in P_{AN_2} from the early peak values during the dive is due to mixing within the blood and delivery of N₂ to perfused tissues. During our studies we measured a consistent increase of arterial hemoglobin content from 15 to 25 g/dl during the first 17 minutes of diving (Fig. 1). The higher hemoglobin concentration increases N₂ solubility in blood by 8 to 10 percent and thus contributes to the decrease of P_{AN_2} during the dive. If other major tissues, such as muscle and blubber (the latter with an approximately 4.5 times higher capacity than blood to dissolve inert gases; the solubility coefficient for homogenized seal blubber and muscle was determined by Van Slyke analysis after tonometry at 37°C: $\alpha_{\text{blubber}} = 0.065$ and $\alpha_{\text{muscle}} = 0.018$) are perfused during the dive they too will be available for N₂ redistribution and can lower the P_{AN_2} to safer levels for surfacing.

The P_{AN_2} of 1200 to 1800 mm Hg reached near the end of ascent but before breathing allows the seal to return to the surface without risking the bends despite rapid decompression. Although arterial blood samples obtained at depth and left for several hours at ambient pressure often released bubbles, in the swimming seal this gas probably remains in solution long enough to be transported to the lungs and exhaled upon surfacing.

TABLE 1.
Splenic Scaling for Terrestrial and Marine Mammals

| Species | Body weight (kg) | Spleen autopsy weight (g) | Spleen autopsy weight % body weight | <i>In vivo</i> spleen weight % body weight | RBC storage capacity % |
|------------------------|------------------|---------------------------|-------------------------------------|--|------------------------|
| Weddell seal n = 13 | 440 ± 70 | 3900 ± 2000 | 0.89 ± 0.214 | 7 | 60* |
| Harbor seal n = 12 | 24 ± 19.2 | — | 0.40 ± 0.152 | — | — |
| Horse n = 15 | 554 ± 37 | — | 0.3* | 1.9 | 54 |
| Sheep n = 20 | 45 ± 4.5 | 91 ± 12.5 | 0.20 ± 0.031 | 1.2 | 26 |
| Dog | — | — | 0.22 | 1.9* | 20 |
| Man | 70 | 150 - 200 | 0.25 - 0.29 | — | <10 |

*Author's estimate

The PaN_2 values measured 4 and 7 minutes after surfacing from a dive to 105 m were 616 and 628 mm Hg, respectively.

We do not know all the mechanisms that allow the Weddell seal to dive safely to 500 m and rapidly return to the surface. However, microcomputer-controlled blood sampling has provided direct evidence that, by limiting blood N_2 uptake through alveolar collapse and by redistributing blood N_2 to blood and tissues during the dive the Weddell seal reduces the risk of nitrogen narcosis and decompression sickness.

B. Hemoglobin

As we have shown that arterial Hb values increase by nearly 60% during the first 10-12 min of both long and short dives (Fig. 1), we now suggest that the source of this influx of oxygenated red blood cells (RBC) is the spleen. Table 1 provides the spleen weight as a percent of body weight for selected terrestrial and marine mammals. The Weddell seal has the highest spleen weight as a percentage of body weight of any reported mammal including the southern Elephant seal, which is also believed to be a prolonged diving seal (Bryden, 1971). In contrast, Baleen whales and porpoises have small spleens, comprising only 0.02% of body weight (Zwillenberg, 1955; Zwillenberg, 1959).

In terrestrial species, such as the sheep (Turner and Hodgetts, 1959) and the horse (Persson *et al.*, 1973), the spleen has been shown to serve as a dynamic RBC reservoir containing respectively, 26% and 54% of the total RBC mass during rest. Excitement, exercise, or the injection of epinephrine caused the ovine spleen to contract and increased the hematocrit by 25% or more (Turner and Hodgetts, 1959). There was no indication that other organs or tissues provide a reversible red blood cell storing capacity after splenectomy. The pathologically enlarged human spleen contracts markedly after epinephrine injection producing a leukocytosis (Schaffner *et al.*, 1985) but there is little increase of either the circulating hemoglobin or hematocrit (Schaffner, personal communication).

In seals the diving reflex is characterized by profound sympathetic vasoconstriction of the peripheral vasculature (Blix and Falkow, 1983). We suggest that during diving the Weddell seal capitalizes upon this sympathetic response to induce constriction of its very large spleen, thus injecting large quantities of oxygenated red cells into the central circulation. Splenic smooth muscle might also be stimulated to contract by low arterial oxygen tensions or due to increased circulating catecholamine levels. Since the spleen is an active storage vessel, determinations of its weight at autopsy are probably misleading, e.g. the spleen weight of the sheep at autopsy is less than a fifth of the *in vivo* weight obtained by clamping the vascular pedicle during general anesthesia (Turner and Hodgetts, 1959). Thus, the autopsy weight of this organ markedly underestimates its red cell storage capacity *in vivo*. A splenic red cell storage capacity of 54% of the total red cell volume has been recorded in the horse (Persson *et al.*, 1973).

We estimate that more than 60% of the red cell mass is stored in the resting Weddell seal spleen based upon two assumptions: (1) the circulating blood volume of the Weddell seal during rest is approximately 10% of total body weight, although laboratory dilution studies during stress have measured values of 15% of body weight or more, and (2) the seal's plasma volume remains constant during diving. This is partly justified because the horse's plasma volume decreased less than 4% after adrenaline injection, despite complete contraction of the spleen, while the measured circulating blood volume increased from 36 to 48 liters (Persson *et al.*, 1973). In a 350 kg Weddell seal with a resting hematocrit of 38% and a diving hematocrit of 60%, the circulating blood volume during diving could increase (due to red cell injection) from 35 to 55 liters. This corresponds to an increase of the circulating blood volume from 10 to 15.7% of the body weight. The latter value is close to the values measured in seals having a high hematocrit during laboratory blood volume investigations (Kooyman *et al.*, 1981; Lenfant *et al.*, 1969). This hypothesis is shown

Dynamic spleen function of Weddell seals

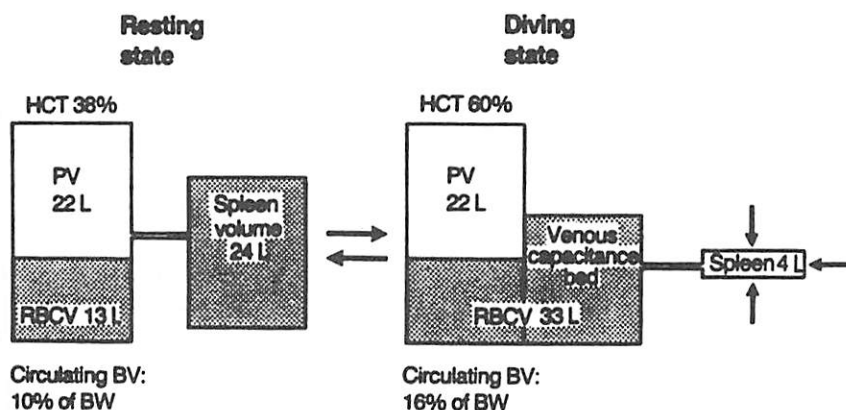


FIG. 5. One hypothesis explaining the events leading to the marked increase of circulating arterial hemoglobin concentration during diving by the Weddell seal. Red blood cells (RBC) stored in the spleen at near resting O_2 and CO_2 tensions are released into the portal circulation and the subdiaphragmatic capacitance veins and then enter the central circulation via the inferior vena caval sphincter. The effects of RBC release are to maintain or increase the aortic arterial oxygen content until the reservoir is depleted (usually the first 10-12 min of long dives), reduce the buildup of CO_2 until the reservoir is emptied (a dilution effect). The splenic storage capacity of red cells may amount to 60% or more of the RBC mass and the spleen's weight during rest probably totals at least 7% of the body weight.

diagrammatically in Fig. 5. Although measurements of *in vivo* spleen weight have not been reported for any seal, we can estimate the *in vivo* resting spleen weight of the Weddell seal weighing 350 kg at 24 kg or 7% of body weight. This estimate is conservative because Hct values of 65% have been measured and we have assumed that the splenic venous effluent has a Hct of 100%, which is unlikely. After spleen contraction the increased circulating blood volume could be accommodated in the subdiaphragmatic venous capacitance system described in the Weddell seal and other species of seals (Blix and Folkow, 1983). During diving with splenic emptying, oxygenated splenic red blood cells would transit the portal circulation, liver and large venous cavities before entering the central circulation. Blix and Folkow (1983) reported inferior vena cava PO_2 was higher than aortic PO_2 during laboratory studies of an unnamed seal species. Splenic contraction may have elevated the caval PO_2 .

During diving, the injection of large amounts of red blood cells into the central circulation with near atmospheric tensions of nitrogen may explain why arterial nitrogen tensions decrease from 2,400 mm Hg to 1,600

mm Hg (Falke *et al.*, 1985). If a large proportion of the total red cell mass is stored in the spleen it would not be exposed to a high alveolar P_{N_2} during descent. During the dive diluting circulating blood with stored red cells having a near atmospheric P_{N_2} would reduce the $P_{a_{N_2}}$ considerably.

Scientists studying seal physiology should strive to take measurements in free swimming and unstressed seals. The remarkable increase of arterial hemoglobin concentration we recorded with each dive was not observed in laboratory diving. Blood sampling of freely diving Weddell seals suggest they remain aerobic and do not increase plasma lactate concentration for diving periods up to 17 minutes (Guppy *et al.*, 1986; Kooyman *et al.*, 1983). In contrast, a laboratory dive as short as 5 minutes is followed by a recovery lactate washout into circulating blood. The difference may well rest upon selective perfusion of exercising muscles in unstressed short free dives. Perfusing exercising muscles would account for the rapid reduction of aortic $P_{a_{O_2}}$ we measured in short dives (Fig. 2). A complete explanation of this metabolic puzzle awaits the continuous measurement of regional blood flow during free diving, just as the precise anatomic identification of the source of the diving increase of arterial hemoglobin concentration, presumably the spleen, awaits future studies upon free swimming seals.

ACKNOWLEDGEMENTS

This work was supported by the National Science Foundation, Division of Polar Programs Grant No. 8100212, and grants from the Danish Medical Research Council and the NATO Science Fellowship Scheme. The work required field support from the National Science Foundation, ITT Antarctic Services and U.S. Naval Support Force Antarctica. The studies were performed in accordance with Permit No. 394 granted by the National Marine Fisheries Service, U.S. Department of Commerce, NOAA.

REFERENCES

- Bert, P. *Lecons sur la physiologie comparee de la respiration*. Paris: Bailliere. p. 526-553, 1870.
- Blix, A.S. and B. Folkow. Cardiovascular adjustments to diving in mammals and birds. In: Shepherd J., Abboud, F., eds. *Handbook of Physiology — The Cardiovascular System III*, Baltimore, MD, Williams and Wilkins, p. 917-946, 1983.
- Bryden, M.S. Size and growth of viscera in the southern elephant seal, *Mirounga leonina*. *Aust. J. Zool.* 19:103-120, 1971.
- Falke, K.J., R.D. Hill, J. Qvist, R.C. Schneider, M. Guppy, G.C. Liggins, P.W. Hochachka, R.E. Elliot, and W.M. Zapol. Seal lungs collapse during free diving: evidence from arterial nitrogen tensions. *Science* 229:556-558, 1985.

- Guppy, M., R.D. Hill, R.C. Schneider, J. Qvist, C.G. Liggins, W.M. Zapol, and P.W. Hochachka. Microcomputer-assisted metabolic studies of voluntary diving of Weddell seals. *Am. J. Physiol.* 19:R175-187, 1986.
- Hill, R.D. Microcomputer monitor and blood sampler for freely diving Weddell seals. *J. Appl. Physiol.* 61:1570-1576, 1986.
- Hill, R.D., R.C. Schneider, G.C. Liggins, A.H. Schuette, R.L. Elliott, M. Guppy, P.W. Hochachka, K.J. Falke, and W.M. Zapol. Heart rate and body temperature during free diving of the Weddell seal. *Am. J. Physiol.* 253:R344-351, 1987.
- Hochachka, P.W., G.C. Liggins, J. Qvist, R.C. Schneider, M.T. Snider, T.R. Wonders, and W.M. Zapol. Pulmonary metabolism during diving: conditioning blood for the brain. *Science* 198:831-834, 1977.
- Klocke, F.J. and H. Rahn. A method for determining inert gas ("N₂") solubility in urine. *J. Clin. Invest.* 40:279-285, 1961.
- Kooyman, G.L., D.H. Kerem, W.B. Campbell and J.J. Wright. Pulmonary function in freely diving Weddell seals, *Leptonychotes weddelli*. *Respiration Physiol.* 12:271-282, 1971.
- Kooyman, G.L. and W.B. Campbell. Heart rates in freely diving Weddell seals, *Leptonychotes weddelli*. *Comp. Biochem. Physiol.* 43A:31-36, 1972.
- Kooyman, G.L., J.P. Schroeder, D.M. Denison, D.D. Hammond, J.J. Wright, and W.P. Bergman. Blood nitrogen tensions of seals during simulated deep dives. *Am. J. Physiol.* 223:1016-1020, 1972.
- Kooyman, G.L., *Weddell Seal: Consummate Diver*. Cambridge: Cambridge University Press, 1981.
- Kooyman, G.L., M.A. Castellini, R.W. Davis and R.A. Maue. Aerobic diving limits of immature Weddell seals. *J. Comp. Physiol.* 151:171-174, 1983.
- Leininger, R.I., M.M. Epstein, R.D. Falb, and G.A. Grode. Preparation of non-thrombogenic plastic surfaces. *Trans. Am. Soc. Artif. Int. Organs.* 12:151, 1966.
- Lenfant, C., R.E. Elsner, G.L. Kooyman and C.M. Drabek. Respiratory function of blood of the adult and fetal Weddell seal, *Leptonychotes weddelli*. *Am. J. Physiol.* 216:1595-1597, 1969.
- Liggins, G.C., J. Qvist, P.W. Hochachka, B.J. Murphy, R.K. Creasy, R.C. Schneider, M.T. Snider, and W.M. Zapol. Fetal cardiovascular and metabolic responses to simulated diving in the Weddell seal. *J. Appl. Physiol.* 49:424-430, 1980.
- Persson, S.G.B., L. Ekman, G. Lydin, and G. Tufvesson. Circulatory effects of splenectomy in the horse I-IV: II. Effect on plasma volume and total and circulating red-cell volume. *ABL Vet. Med. A.* 20:456-468, 1973.
- Qvist, J., R.D. Hill, R.C. Schneider, K.J. Falke, C.G. Liggins, M. Guppy, R.L. Elliott, P.W. Hochachka, and W.M. Zapol. Hemoglobin concentrations and blood gas tensions of free-diving Weddell seals. *J. Appl. Physiol.* 61:1560-1569, 1986.
- Schaffner, A., M.D. Augustiny, C.O. Rainer and J. Fehr. The hypersplenic spleen, a contractile reservoir of granulocytes and platelets. *Arch. Intern. Med.* 145:651-654, 1985.
- Turner, A.W., and U.E. Hodgetts. The dynamic red cell storage function of the spleen in sheep. Relationship to fluctuations of jugular haematocrit. *Aust. J. Expl. Biol.* 37:339-420, 1959.
- Zapol, W.M. G.C. Liggins, R.C. Schneider, J. Qvist, M.T. Snider, R.K. Creasy, and P.W. Hochachka. Regional blood flow during simulated diving in the conscious Weddell seal. *J. Appl. Physiol.* 47:968-973, 1979.
- Zwillenberg, H.H.L. Die mikroskopische Anatomie der Milz der Furchenwale. *Acta. Anat.* 32:24-39, 1955.
- Zwillenberg, H.H.L. Über die Milz des Braunfisches. *Zeitschr. Anat. Entw.* 121:9-18, 1959.

4

Energetics of Breath-Hold Diving in Korean and Japanese Professional Divers

Y. S. Park, K. Shiraki, and S.K. Hong

| | |
|--|----|
| I. Introduction | 73 |
| II. Swimming Suit Divers | 75 |
| A. Heat Exchange During Diving Work | 75 |
| B. Limited Diving Time | 76 |
| III. Wet-Suit Divers | 77 |
| A. Heat Exchange During Diving Work | 77 |
| B. Thermal Insulation During Diving Work | 82 |
| IV. Summary | 85 |
| Acknowledgements | 86 |
| References | 86 |

I. INTRODUCTION

Over the last 2,000 years professional breath-hold divers (Ama) in Korea and Japan have harvested the ocean floor for shellfish, sea urchins, seaweeds and other marine organisms. It is estimated that in Korea and Japan today more than 20,000 divers are engaged in this profession. Interestingly, while only women are engaged in this type of diving work in Korea, both men and women are involved in Japan. Traditionally, these divers wore only cotton diving suits, but in recent years (1960s and 1970s) they adopted wet suits to protect against eventual hypothermia during diving work. The present paper summarizes energetics of diving documented for Japanese male (Shiraki *et al.*, 1986 and Korean female (Kang *et al.*, 1983) unassisted breath-hold divers.

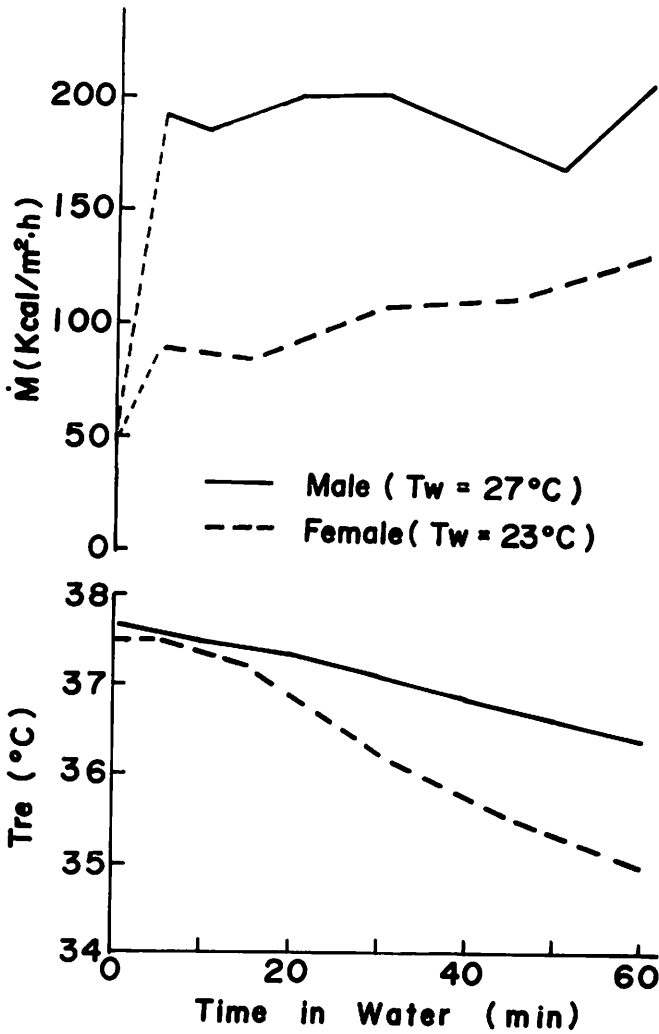


FIG. 1. Changes in metabolic rate (\dot{M}) and rectal temperature (T_{re}) of male and female divers during diving work with swimming suits in summer. Each curve represents the mean of 4 divers. Based on data of Kang *et al.* (1983) and Shiraki *et al.* (1986).

II. SWIMMING SUIT DIVERS

A. Heat Exchange During Diving Work

In each of 4 Korean females (average age 37 years, height 159 cm, weight 51 kg, surface area 1.5 m², skinfold thickness 12.6 mm) and 4 Japanese male divers (average age 28 years, height 166 cm, weight 61 kg, surface area 1.7 m², skinfold thickness 8.6 mm), thermal exchange between the body and water was studied while they were working (slow swimming plus intermittent diving) in the sea. The male divers wore swimming trunks and the female divers wore their traditional cotton swimming suits, and none of them used fins.

Fig. 1 depicts the average metabolic rate (\dot{M}) and rectal temperature (T_{re}) of male and female divers as a function of working time in summer. The resting \dot{M} before diving work was approximately 50 kcal/m² in both groups. Upon initiation of the work the \dot{M} immediately increased to about 190 kcal/m² in the male and 90 kcal/m² in the female divers. After this initial rise, the \dot{M} in the male diver remained unchanged, whereas that in the female diver increased gradually, probably due to shivering. Overall, the extra \dot{M} due to diving work appeared to be 2-3 times higher in the male than in the female diver, indicating that the workload of diving was much greater in the former than in the latter. This may be, in part, due to the difference in their diving patterns. During repetitive dives, the average rate of descent was observed to be 17% faster in the male (0.63 m/sec) than in the female (0.54 m/sec) divers (Park *et al.*, 1983 and Shiraki *et al.*, 1985).

The T_{re} of the subjects declined gradually during diving work in both groups. However, the rate of T_{re} fall was much greater in the female than in the male divers, such that the total reduction in T_{re} during the 1-h diving work was approximately 1.3°C in the male and 2.5°C in the female divers.

Fig. 2 illustrates the cumulative extra heat production (ΔM) and the reduction in body heat content (ΔS) during diving work. The sum of these two values for any time period gives the extra heat loss due to diving (i.e., thermal cost of diving). The ΔM was calculated from the extra $\dot{V}O_2$ over the resting value ($\Delta M = 4.83 \Delta \dot{V}O_2$) and the ΔS from the change in mean body temperature in water ($\Delta S = 0.83 \Delta T_b \times \text{body weight}$, $\Delta T_b = 0.6 \Delta T_{re} + 0.4 \Delta \bar{T}_{sk}$, \bar{T}_{sk} = mean skin temperature). In the male diver, the ΔM over 1 h was 125 kcal/m² and the ΔS was 85 kcal/m²; thus the total extra heat loss was calculated to be 210 kcal/m². In the female diver, the total amount of extra heat loss over 1 h (212 kcal/m²) was similar to that in the male diver, but the ΔS (165 kcal/m²) appeared to be 3 times greater than the ΔM (47 kcal/m²). In other words, most of the thermal loss in the

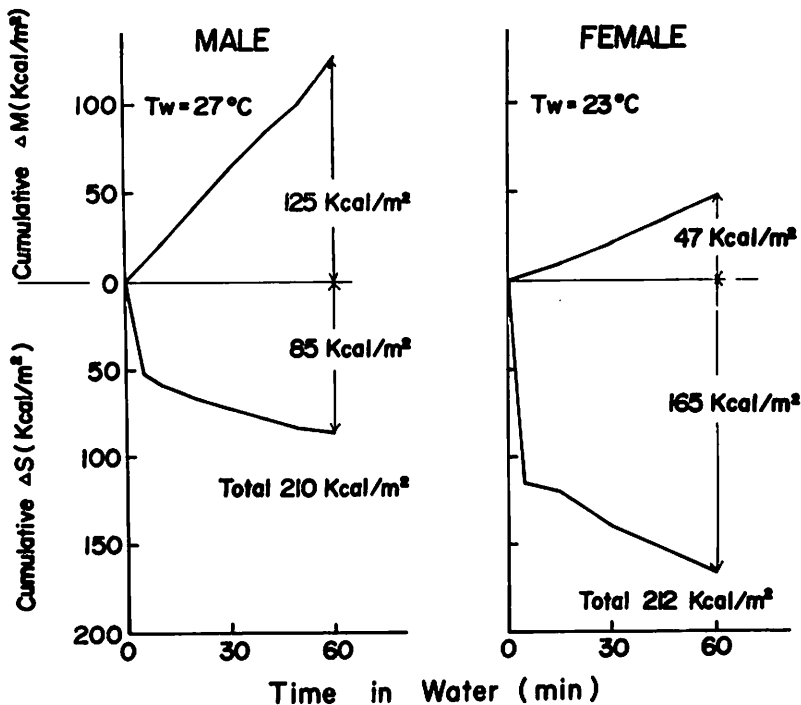


FIG. 2. Cumulative extra heat production (ΔM) and body heat content change (ΔS) in male and female divers during diving work with swimming suits. Each curve represents the mean of 4 divers. Based on data of Kang *et al.* (1983) and Shiraki *et al.* (1985).

female diver was from the body heat storage. It may be that because of a relatively low workload of diving in the female diver, the heat production that compensated for the heat loss is inadequate and, consequently, the body heat content decreased more than in the male diver.

B. Limiting Diving Time

Earlier studies on Korean women cotton-suit divers (Kang *et al.*, 1963; Kang *et al.*, 1965) indicated that the most important factor determining the working time was deep body cooling rather than the absolute amount of body heat loss. Divers voluntarily terminated their diving work when the rectal temperature fell to about 35°C , the time to reach this point being proportional to sea water temperature. The amount of heat loss

during a work shift was much greater in cold seasons than in warm seasons.

Exactly the same result was obtained in contemporary Korean women divers during diving work with cotton swimming suits. As depicted in Fig. 3, divers terminated the work after 60 min in summer ($T_w = 23^\circ\text{C}$) and at 30 min in winter ($T_w = 10^\circ\text{C}$). However, the final T_{re} was about 35°C in both seasons. The mean skin temperature (\bar{T}_{sk}) dropped to 24°C in summer and 13°C in winter at the end of work shift; hence the reduction in the mean body temperature was much greater in winter (8.4°C) than in summer (6°C). The loss of body heat content was calculated to be 165 and 242 kcal/m^2 in summer and winter, respectively. These results confirm once again that the core temperature fall is the most critical factor determining the working time in unprotected divers.

Since the T_{re} of 35°C appeared to be the maximal degree of hypothermia that divers voluntarily tolerated, we defined the theoretical limit of diving work in terms of deep body cooling. In any season, the rate of T_{re} fall after the initial 5-10 min was proportional to the initial $T_{re}-T_w$ difference; thus the cooling rate per unit $T_{re}-T_w$ gradient was about 0.21°C/h per $^\circ\text{C}$ ($T_{re}-T_w$). Using this value and the initial delay in T_{re} change (t_i) the duration of diving work (t_{limit}) in which the T_{re} would decline to 35°C was calculated.

$$t_{limit} \text{ (h)} = \frac{37^\circ\text{C} - 35^\circ\text{C}}{0.21 (37^\circ\text{C} - T_w)} + t_i$$

Such estimations for a wide range of T_w are illustrated in Fig. 4.

III. WET-SUIT DIVERS

A. Heat Exchange During Diving Work

In the same divers involved in the above mentioned study of swimming suit diving, the heat exchange in wet-suit diving was studied during their natural work shifts in summer and winter. The female divers (Korean) wore the same type of suits (5 mm neoprene jacket, pants, boots, hood, and cotton gloves) in all seasons, but the male divers (Japanese) wore different types of suits depending upon the season. That is, in summer they wore 5 mm neoprene wet-suits (jacket and pants) with cotton gloves and socks, whereas in winter they wore thicker (5.5 - 6.0 mm) wet-suits with gloves, boots, and hoods of neoprene. Thus, the external insulation of the male diver was higher in winter than in summer.

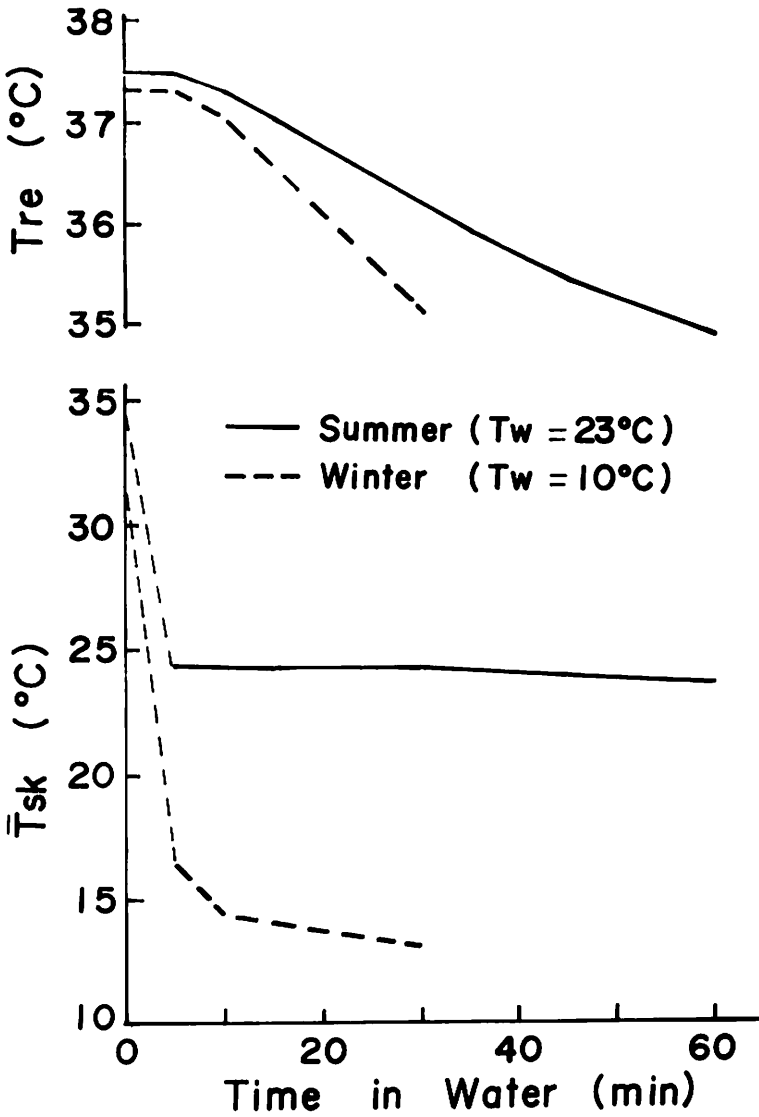


FIG. 3. Changes in rectal (T_{re}) and mean skin (T_{sk}) temperatures of Korean women cotton-suit divers during diving work in summer and winter. Each curve represents the mean of 4 divers. Based on Kang *et al.* (1983).

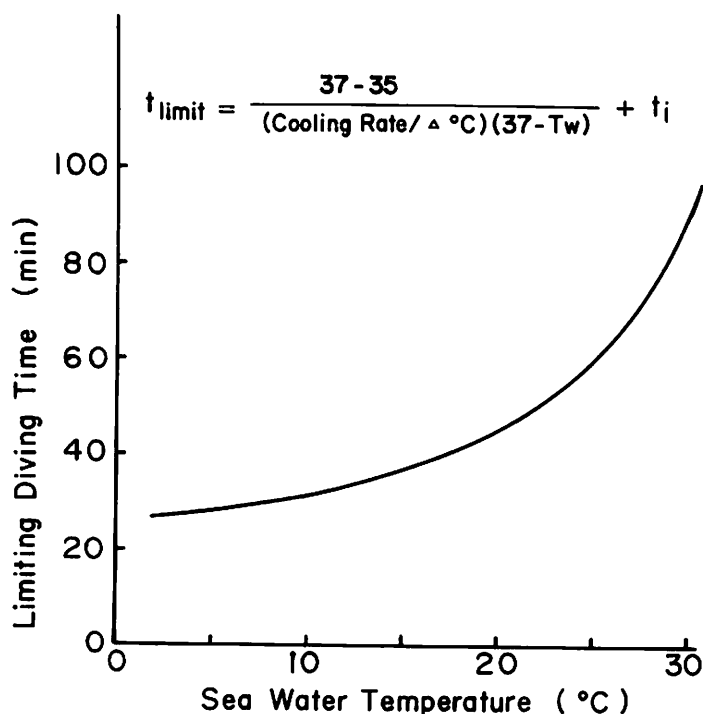


FIG. 4. Prediction of limiting diving time in Korean women cotton-suit divers.

Fig. 5 summarizes changes in T_{re} , \dot{M} and cumulative extra heat loss (ΔH) during diving work observed in female divers. Unlike in swimming suit diving, the T_{re} in the wet-suit diving was not appreciably altered over the 2-h work period. The reduction in T_{re} in 2 h was only 0.4°C in the summer ($T_w = 23^\circ\text{C}$) and 0.6°C in winter ($T_w = 10^\circ\text{C}$). Thus, the T_{re} was of no major importance in the determination of work period. The \dot{T}_{sk} (and hence T_b) was also maintained at a level significantly higher than that in swimming suit diving (data not shown).

The \dot{M} of the subjects increased rapidly during the first few minutes of diving work and remained unchanged during the rest of the work period in all seasons. However, the net increase in \dot{M} appeared to be 3 times greater in winter (approximately $100 \text{ kcal/m}^2\text{h}$) than in summer (approximately $33 \text{ kcal/m}^2\text{h}$). Since the work load of diving may not be significantly

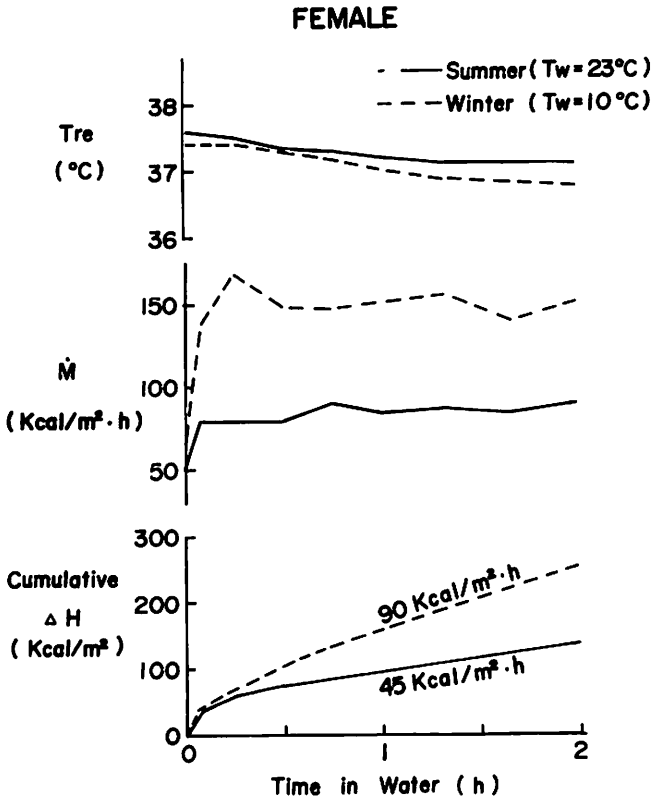


FIG. 5. Changes in rectal temperature (T_{re}), metabolic rate (\dot{M}) and cumulative extra heat loss (ΔH) during diving work in Korean women wet-suit divers. Each curve represents the mean of 4 divers. Based on the data of Kang *et al.* (1983).

different between the two seasons, the higher level of \dot{M} in winter than in summer indicates that even with wet-suits, shivering takes place in the cold season. In this connection, it is important to point out that contemporary Korean women divers do not wear protective gloves even in the cold of winter. Thus, the exposure of hands and face to cold water may have evoked shivering response in the cold season. In fact, Van Someron *et al.*, (1982) have observed that local cooling of hands and feet stimulated heat production in nude subjects immersed in lukewarm water.

The cumulative extra heat loss (ΔH) during diving work is depicted in the bottom panel of Fig. 5. In both summer and winter the cumulative ΔH increased rapidly during the initial 20-30 min period, followed by a

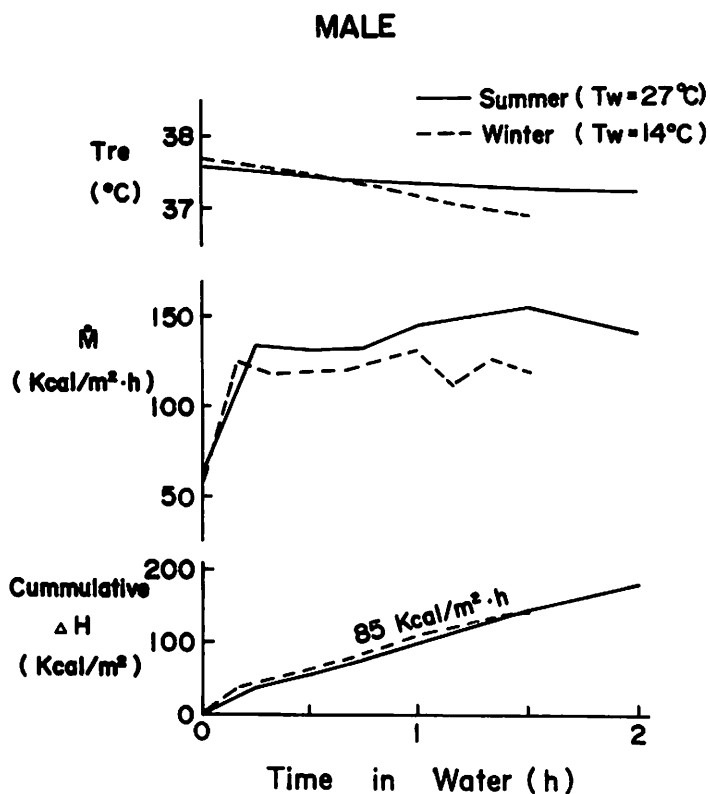


FIG. 6. Changes in rectal temperature (T_{re}), metabolic rate (\dot{M}) and cumulative extra heat loss (ΔH) during diving work in Japanese male wet-suit divers. Each curve represents the mean of 4 divers. Based on the data of Shiraki *et al.* (1986).

steady, slow increase. Thus, from the slope of the steady portion of the curve we estimated the rate of net thermal loss of approximately $45 \text{ kcal/m}^2 \cdot \text{h}$ in summer and $90 \text{ kcal/m}^2 \cdot \text{h}$ in winter. These values are less than 20% of those values observed in cotton-suit divers.

Fig. 6 illustrates courses of T_{re} , \dot{M} and cumulative ΔH of male wet-suit divers during diving work in summer ($T_w = 27^\circ\text{C}$) and winter ($T_w = 14^\circ\text{C}$). Average values during the entire work period are summarized in

Table 1, along with values for female wet-suit divers for comparison. In general, changes in T_{re} were similar to those in female divers. The final T_{re} at the end of a work shift was 37.2°C in summer (2-h shift) and 36.8°C in winter (90 min shift).

The \dot{M} was maintained at a more or less constant level during the entire work period. Interestingly, however, the average \dot{M} in winter (121 kcal/m²h) was not higher but slightly lower than that in summer (141 kcal/m²h) in males, in contrast to those values observed in female divers (Table 1). In the latter case, the \dot{M} in winter (148 kcal/m²h) was significantly higher than that in summer (84 kcal/m²h) due to shivering. Presumably shivering also occurred in male divers, since their T_{re} and \bar{T}_{sk} in winter were comparable to female divers' (see Table 1). Evidently, the diving work load in the male diver was lower in winter than in summer.

In both summer and winter the cumulative ΔH increased steadily after an initial rapid rise. Surprisingly, however, the value at a given work period was not significantly different between the two seasons, indicating that the thermal cost of diving did not vary with the season. The rate of diving heat loss estimated from the steady portion of the curve was approximately 85 kcal/m²h in both summer and winter.

TABLE 1.

Average Body Temperature and Heat Production during Wet-suit Diving

| | | Male ^a | | Female ^b | |
|----------------|-------------------------|-------------------|--------|---------------------|--------|
| | | Summer | Winter | Summer | Winter |
| T_w | (°C) | 27 | 14 | 23 | 10 |
| T_{re} | (°C) | 37.2 | 36.8 | 37.1 | 36.8 |
| \bar{T}_{sk} | (°C) | 33.1 | 28.4 | 31.5 | 27.5 |
| \dot{M} | (Kcal/m ² h) | 141 | 121 | 84 | 148 |

^abased on data of Shiraki *et al.* (1986)

^bbased on data of Kang *et al.* (1983)

B. Thermal Insulation During Diving Work

Since the heat loss in water is determined mostly by the magnitude of skin heat flux, which is proportional to the body core to water temperature gradient and inversely proportional to the peripheral insulation, $\dot{H}_s = [T_{core} - T_w]/I$, an equal diving heat loss (hence skin heat loss) between the two seasons in male divers requires that their thermal insulation

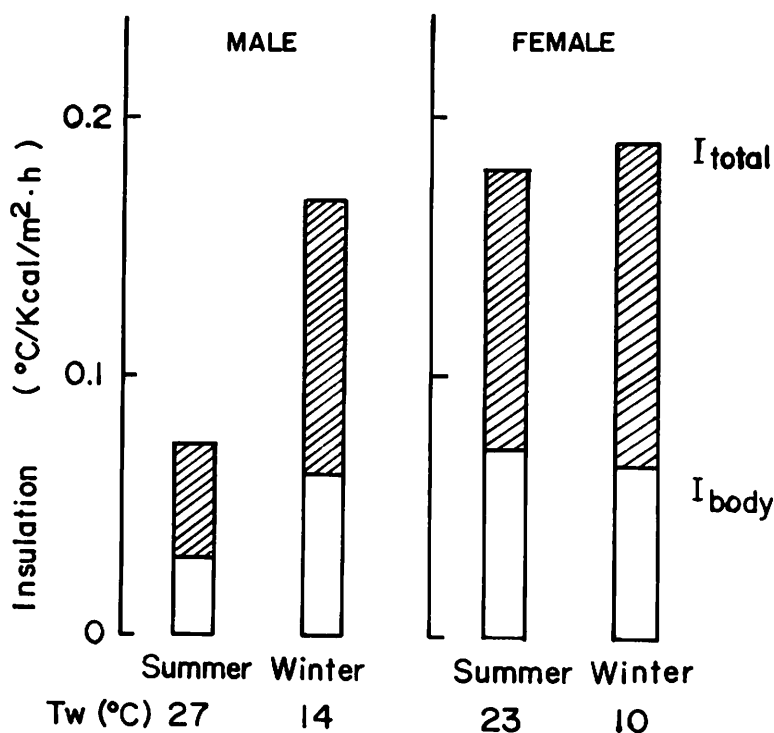


FIG. 7. Apparent thermal insulation of male (Japanese) and female (Korean) wet-suit divers during diving work in summer and winter. Each value represents the mean of 4 divers. Based on data of Kang *et al.* (1963) and Shiraki *et al.* (1986).

increased in the cold season. In fact, calculation of thermal insulation using the T_{re} , \bar{T}_{sk} , T_w and \dot{M} during diving work indicated that overall insulation ($I_{total} = [T_{re} - T_w]/\dot{H}_s$, $\dot{H}_s = 0.92 \dot{M} + \Delta S$) of male divers in winter ($0.168^\circ\text{C/kcal/m}^2\cdot\text{h}$) was 2-fold greater than in summer (0.074). This was due to changes in internal ($I_{body} = [T_{re} - \bar{T}_{sk}]/\dot{H}_s$) as well as external ($I_{suit} = I_{total} - I_{body}$) insulation (Fig. 7). The value of I_{body} and I_{suit} increased from 0.030 and $0.044^\circ\text{C/kcal/m}^2\cdot\text{h}$ in summer to 0.062 and $0.106^\circ\text{C/kcal/m}^2\cdot\text{h}$ in winter. Such a marked seasonal variation of thermal insulation has not been observed in female divers of Korea (Fig. 7).

The change in I_{suit} between warm and cold seasons in male divers was partly accounted for by the change in attire. As mentioned above, the subjects wore 5 mm wet-suits in summer, but they wore 5.5 - 6.0 mm wet-

suits and had additional protection with hoods, gloves, and boots in winter. Thus, the physical insulation external to the body was obviously increased in winter. However, this may not fully account for the observed change in I_{suit} . According to Goldman *et al.*, (1966), physical insulation of 4.76 and 6.35 mm neoprene wet-suits are only slightly different (0.128 vs 0.139°C/kcal/m²·h). Perhaps the more important reason for the change in I_{suit} was a change in diving pattern between summer and winter. As shown in Table 2, the surface to dive time ratio of male divers increases in winter. The ratio for the subjects serving in the thermal study was on the average 50% higher in winter (1.97) than in summer (1.30). Since the physical insulation of the wet-suit is lower at depth than at the surface due to compression of the trapped air (Beckman, 1963; Park *et al.*, 1988; Suh *et al.*, 1987), and the physiological insulation by wet-suits decreases with exercise due to increase in effective surface area for heat exchange (Yeon *et al.*, 1987) as well as heat convection under the suit (Wolff *et al.*, 1985), the average I_{suit} during breath-hold diving will increase as the surface to dive time ratio increases. The apparently high I_{body} in winter might also be attributed to the increase in surface to dive time ratio in winter. Since the body insulation in water undergoes a dramatic reduction during exercise (Park *et al.*, 1984; Suh *et al.*, 1987; Yeon *et al.*, 1987), I_{body} of divers would increase as the length of surface rest increases. In fact, observations in two Korean women divers (Table 3) indicated that the average I_{body} increased from 0.062 to 0.075°C/kcal/m²·h and I_{suit} increased from 0.126 to 0.153°C/kcal/m²·h as their surface to dive time ratio was voluntarily increased from 1 to 2. It is therefore speculated that

TABLE 2.

Average Dive and Surface Time during Typical Diving Work (5-10 m depth)

| | Dive Time (sec) | Surface Time (sec) | Surface Time / Dive Time |
|----------------------|--------------------|-----------------------|-----------------------------|
| Japanese Male Divers | | | |
| Summer | 39 | 42 | 1.08 |
| Winter | 33 | 47 | 1.42 |
| Korean Female Divers | | | |
| Summer and Winter | 32 | 46 | 1.44 |

*based on data of Shiraki *et al.* (1985)*based on data of Park *et al.* (1983)

a significant fraction of the increase in thermal insulation of male divers in the cold season was a consequence of a behavioral adjustment of diving pattern, a mechanism with which divers conserve body temperature during diving work in cold water. This notion may be indirectly supported by the fact that in Korean women wet-suited divers, who do not change diving pattern throughout the year, there is no apparent seasonal variation of thermal insulation (see Fig. 7). The reason why such an adaptation has been acquired only in male divers is not clear at present.

TABLE 3.

Effect of Surface to Dive Time Ratio (S/D) on Thermal Insulation of Wet-Suit Divers

| | S/D = 1 | S/D = 2 |
|---|---------|---------|
| I_{body} ($^{\circ}\text{C}/\text{kcal}/\text{m}^2\cdot\text{h}$) | 0.062 | 0.075 |
| I_{suit} ($^{\circ}\text{C}/\text{kcal}/\text{m}^2\cdot\text{h}$) | 0.126 | 0.153 |
| I_{total} ($^{\circ}\text{C}/\text{kcal}/\text{m}^2\cdot\text{h}$) | 0.187 | 0.230 |

Data represent mean of two Korean female wet-suit divers during 4-5 m diving work in summer ($T_w = 22^{\circ}\text{C}$).

IV. SUMMARY

In the present paper we compared energetics of breath-hold diving between Japanese male and Korean female professional divers. In unprotected (swimming suited) divers, the total amount of extra heat loss during diving work in summer ($23 - 27^{\circ}\text{C}$ water) was similar (approximately $210 \text{ kcal}/\text{m}^2\cdot\text{h}$), but the thermal balance was substantially different between male and female divers. That is, in male divers the amount of extra heat production was significantly greater (125 compared with $47 \text{ kcal}/\text{m}^2\cdot\text{h}$) and the reduction in body heat content was smaller (85 compared with $165 \text{ kcal}/\text{m}^2\cdot\text{h}$) than in female divers; consequently the degree of deep body cooling was relatively smaller in male than in female divers. In both male and female wet-suit divers, the amount of heat production and the fall in body heat content during diving work were significantly smaller than the corresponding values in unprotected divers. The rate of diving heat loss in female divers changed from $45 \text{ kcal}/\text{m}^2\cdot\text{h}$ in summer (23°C water) to $90 \text{ kcal}/\text{m}^2\cdot\text{h}$ in winter (10°C water), whereas in male divers, there was no apparent seasonal variation (approximately $80 \text{ kcal}/\text{m}^2\cdot\text{h}$ in both summer, 27°C water, and winter, 14°C water). The overall thermal insulation (tissue and wet-suit) during diving work in female

divers was approximately constant at all seasons ($0.18 - 0.19^{\circ}\text{C}/[\text{kcal}/\text{m}^2\cdot\text{h}]$), but that in the male diver changed significantly from $0.074^{\circ}\text{C}/[\text{kcal}/\text{m}^2\cdot\text{h}]$ in summer to 0.168 in winter. Such a difference in seasonal variation of diving heat loss and thermal insulation between male and female divers was attributed in part to the difference in their diving patterns.

ACKNOWLEDGEMENT

This report is based on studies supported from grants from the Korea Science and Engineering Foundation (1981), National Science Foundation (INT 79-18378) and the Ministry of Labor of Japan (1984).

REFERENCES

- Beckman, E.L. Thermal protection during immersion in cold water. In: C.J. Lambertsens, ed. *Proceedings of Second Symposium on Underwater Physiology*. Washington, DC: NAS-NRC, Pub. 1811, p. 247-266, 1963.
- Goldman R.F., J.F. Breckenridge, E. Reeves, and E.L. Beckman. "Wet" versus "dry" suit approaches to water immersion protective clothing. *Aerospace Med.* 37:485-487, 1966.
- Kang, B.S., S.H. Song, C.S. Suh and S.K. Hong. Changes in body temperature and basal metabolic rate of the ama. *J. Appl. Physiol.* 18:483-488, 1963.
- Kang, D.H., P.K. Kim, B.S. Kang, S.H. Song, and S.K. Hong. Energy metabolism and body temperature of the ama. *J. Appl. Physiol.* 20:46-50, 1965.
- Kang, D.H., Y.S. Park, Y.D. Park, I.S. Lee, D.S. Yeon, S.H. Lee, S.Y. Hong, D.W. Rennie and S.K. Hong. Energetics of wet-suit diving in Korean women breath-hold divers. *J. Appl. Physiol.* 54:1702-1707, 1983.
- Park, Y.H., J. Iwamoto, F. Tajima, K. Miki, Y.S. Park, and K. Shiraki. Effects of pressure on thermal insulation in humans wearing wet suits. *J. Appl. Physiol.* 64:1916-1922, 1988.
- Park, Y.S., D.R. Pendergast, and D.W. Rennie. Decrease in body insulation with exercise in cold water. *Undersea Biomed. Res.* 11:159-168, 1984.
- Park, Y.S., H. Rahn, I.S. Lee, S.I. Lee, D.H. Kang, S.Y. Hong, and S.K. Hong. Patterns of wet suit diving in Korean women breath-hold divers. *Undersea Biomed. Res.* 3:203-215, 1983.
- Shiraki, K., N. Konda, S. Sagawa, Y.S. Park, T. Komatsu, and S.K. Hong. Diving pattern of Tsushima male breath-hold divers (Katsugi). *Undersea Biomed. Res.* 12:439-452, 1985.
- Shiraki, K., S. Sagawa, N. Konda, Y.S. Park, T. Komatsu, and S.K. Hong. Energetics of wet-suit diving in Japanese male breath-hold divers. *J. Appl. Physiol.* 61:1475-1480, 1986.
- Suh, D.J., D.S. Yeon, H.J. Kim, J.K. Choi, Y.S. Park, Y.H. Park, and S.K. Hong. Thermal balance of wet-suit divers during exercise in cold water at 1, 2 and 3 ATA. In: A.A. Bove, A.J. Bachrach, and L.J. Greenbaum, Jr., eds. *Underwater and Hyperbaric Physiology IX*, Bethesda, MD: Undersea and Hyperbaric Medical Society, p. 121-129, 1987.
- Van Someron, R.N.M., S.R.K. Coleshaw, P.J. Mincer, and W.R. Keatinge. Restoration of thermoregulatory response to body cooling by cooling hands and feet. *J. Appl. Physiol.* 53:1228-1233, 1982.
- Wolff, A.H., S.R.K. Coleshaw, C.G. Newstead, and W.R. Keatinge. Heat exchanges in wet suit. *J. Appl. Physiol.* 58:770-777, 1985.
- Yeon, D.S., Y.S. Park, J.K. Choi, J.S. Kim, I.S. Lee, D.H. Kang, S.H. Lee, S.Y. Hong, D.W. Rennie, and S.K. Hong. Changes in thermal insulation during underwater exercise in Korean female wet-suit divers. *J. Appl. Physiol.* 62:1014-1019, 1987.

5

Safety of Sports Diving: Comparison of Novice and Expert Divers

*Y. Mano, M. Shibayama, T. Mizuno and
J. Ohkubo*

| | |
|--|-----|
| I. Introduction | 87 |
| II. Fatal Diving Accidents | 88 |
| III. Non-Fatal Diving Accidents | 89 |
| IV. Inner Ear Barotrauma | 94 |
| V. Comparison of Diving Profiles Between Novice and Expert Divers | 95 |
| A. Characteristics of the Diving Recorder | 95 |
| B. Subjects | 95 |
| C. Procedures | 96 |
| D. Results | 96 |
| VI. Discussion | 100 |
| VII. Conclusion | 102 |
| References | 103 |

I. INTRODUCTION

The sports diver population in Japan has increased tremendously in recent years. There are now over 400,000 people who are engaged in recreational diving. Of this number, three quarters have obtained their Certificates (C-card) in the last few years and one fourth are novice divers who have been diving for less than one year. If the index for the number of C-cards issued in 1981 is set at 100 (11,000 cards), the figure is 172 in 1983, 527 in 1985 (58,000 cards), and over 800 in 1987, indicating that the number of C-cards issued is about 90,000 per year in recent years (Japanese Association of Underwater Exploitation, 1988).

The number of diving accidents has also risen and is becoming a social issue as a result of the sudden upsurge in the sports diver population. Consequently, the regulations on diving lag behind and are currently under intense study at the national level. The Ministry of Labor and others are beginning to formulate safety policies related to sports diving. Regulations and policies on diving accidents are being set under the auspices of the Maritime Safety Agency and the National Police Agency, which engage in rescue operations. The Ministry of International Trade and Industry is doing the same from the perspective of industrial standards for diving equipment in Japan. The Ministry of Education is cooperating by supervising the compilation of guidelines for the qualification system for diving instructors, while the Labor Department is doing the same from the perspective of labor regulations governing the job of instructor (Japan Maritime Safety Board, 1988).

An examination of scuba diving accidents reveals that novice divers encounter an extremely high rate of accident. One of the reasons is that novice divers tend to panic easily. This stems mainly from incomplete adjustment of pressure in the ears (Kitajima *et al.*, 1980; Mano and Shibayama, 1987; Mano *et al.*, 1988). Another reason for the difference in accident rates may be that the diving profiles differ between them. We tried to ascertain this by accurately recording and comparing their diving profiles by using the Adonis Diving Recording (ADR) Computer System (Mano *et al.*, 1987; Ohkubo *et al.*, 1987). We also analyzed the differences in diving experience and techniques between expert and novice divers.

The occurrence of ear barotrauma, which is the most common diving injury among divers, tends to cause panic particularly in novice divers. We made a comparison between novice and expert divers by conducting Eustachian tube opening and closing function tests and hearing tests on those divers suffering from ear and sinus barotrauma at the Tokyo Medical and Dental University Hospital (Ohkubo and Watanabe, 1984; Ohkubo and Watanabe, 1985).

The results of our studies showed distinct differences in the diving profiles between experts and novices. There is also evidence that the cause of ear barotrauma differs depending on the extent of diving experience and skill.

II. FATAL DIVING ACCIDENTS

Accurately determining the conditions that lead to diving accidents, is fundamental and cannot be ignored when considering safe diving. However, diving accidents, as long as they do not result in death, are often not reported and it is therefore almost impossible to determine the number of

TABLE 1.
Diving Experience and Fatal Diving Accidents

| Diving Experience | Persons | % |
|--------------------|---------|-------|
| first dive | 32 | 30.8 |
| within one year | 15 | 14.4 |
| within three years | 32 | 30.8 |
| within five years | 6 | 5.8 |
| over five years | 19 | 18.2 |
| Total* | 104 | 100.0 |

* In 26 additional cases, diving experiences were unknown.

non-lethal accidents or the rate of their occurrence. A system such as the Diving Alert Network (DAN) is yet to be established in Japan.

Taking these points into account, we have conducted a diving accident survey, studying only accidents which resulted in death since 1975 (Kitajima *et al.*, 1980; Mano and Shibayama, 1987). Cases of novice divers who died in accidents, represents nearly half, and 30 percent of the total cases of death occurred when they dived in the ocean for the first time (Table 1). These victims had received their diving training less than 1 year previously. The number of divers who encountered fatal accidents diving at depths of less than 10 m accounted for two-thirds of the total. One-fourth of the victims died in full scuba diving equipment before even starting to dive, while still moving on the ocean surface (Table 2). We can say that in diving accidents, those who most easily fall victim to the worst possible cases are novice divers. Although difficult to survey epidemiologically, based on the previous findings, we predict a relatively high rate of novice diver accidents, even if these are not serious to the point of death.

III. NON-FATAL DIVING ACCIDENTS

The most common accident among novice divers is ear damage. When considering the safety of sports divers, the first issue must be policies that deal with ear and sinus pressure adjustment during diving.

Of the 49 scuba divers (29 men, 20 women; 23 novices, 26 experts) who were treated at the Tokyo Medical and Dental University Hospital for ear

TABLE 2.

Depth at Diving Accidents

| Depth | Number | % |
|--------------------|--------|-------|
| surface | 54 | 24.3 |
| 5 m | 47 | 21.2 |
| 10 m | 45 | 20.3 |
| 15 m | 19 | 8.5 |
| 20 m | 25 | 11.3 |
| > 20 m | 32 | 14.4 |
| Total ^a | 222 | 100.0 |

^a In additional 20 cases, the depth at which accidents occurred was unknown.

damage in the last 1 and 1/2 years, 44 complained of ear problems and 5 had squeeze of the sinus. In terms of the type of diving experience and the occurrence of damage, all 23 of the novice divers had complained of pressure damage while diving during practice in a pool or diving for the first time in the ocean. The type of damages included ruptured eardrum, earache while diving, and incomplete ear pressure adjustment.

Among experienced divers, 7 instructors suffered ear barotrauma during their diving instruction while they also had a slight cold. There were many cases of ruptured eardrums and sensory neural high tone loss among occupational divers (all fishermen).

Functional loss of hearing differs among experienced and novice divers, even in cases with similar ear damage. Results of a hearing test indicate that compared with experts who had sensory neural loss of high tones, the hearing problems of novice divers were hearing loss of low tone, high tone, or both (Fig. 1). In general, diving barotrauma mostly affects hearing loss of high tones, but some divers complained of low tone loss as well. This was particularly common among novice divers with little diving experience. However, the trend among experienced divers is for a greater degree of high tone damage.

In order to study the Eustachian tube function in these types of damage, we used sonotubometry to detect the opening and closing of the Eustachian tube in natural swallowing movements, and a sonometric Valsalva test with nasal internal pressure as the index (Fig. 2, Fig. 3) (Ohkubo

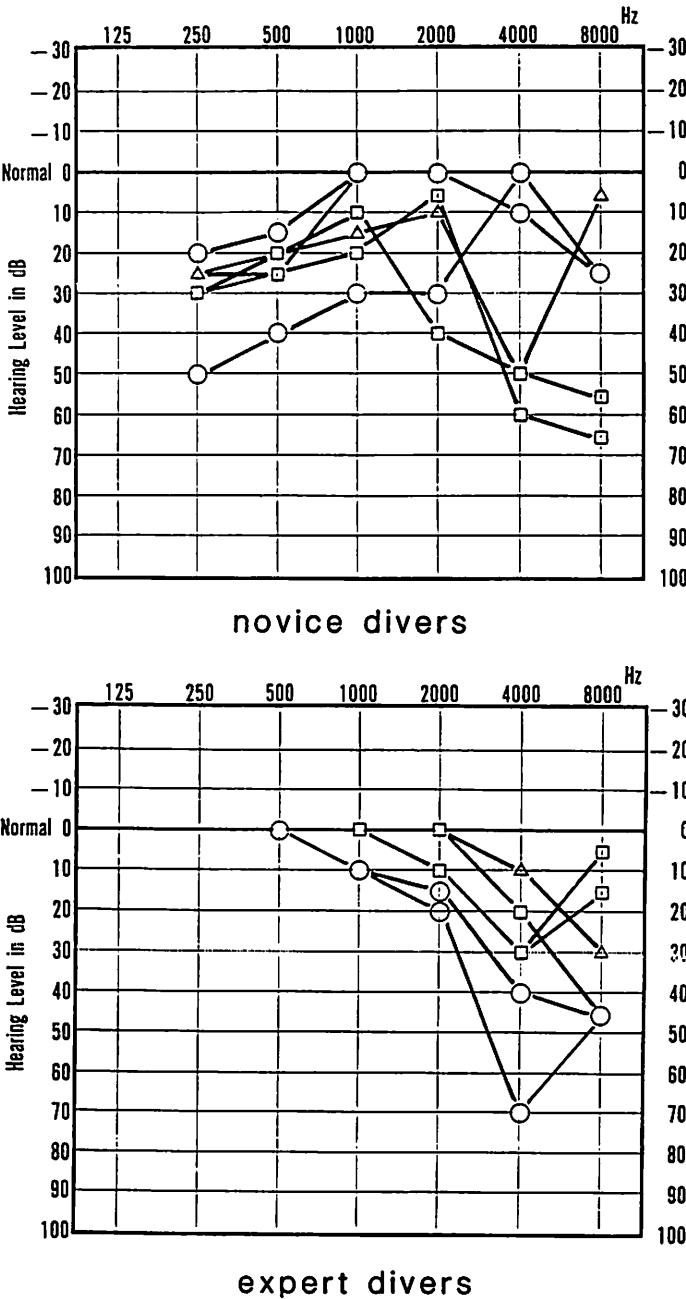


FIG. 1. Comparison of sonotubometry between novice and expert divers.

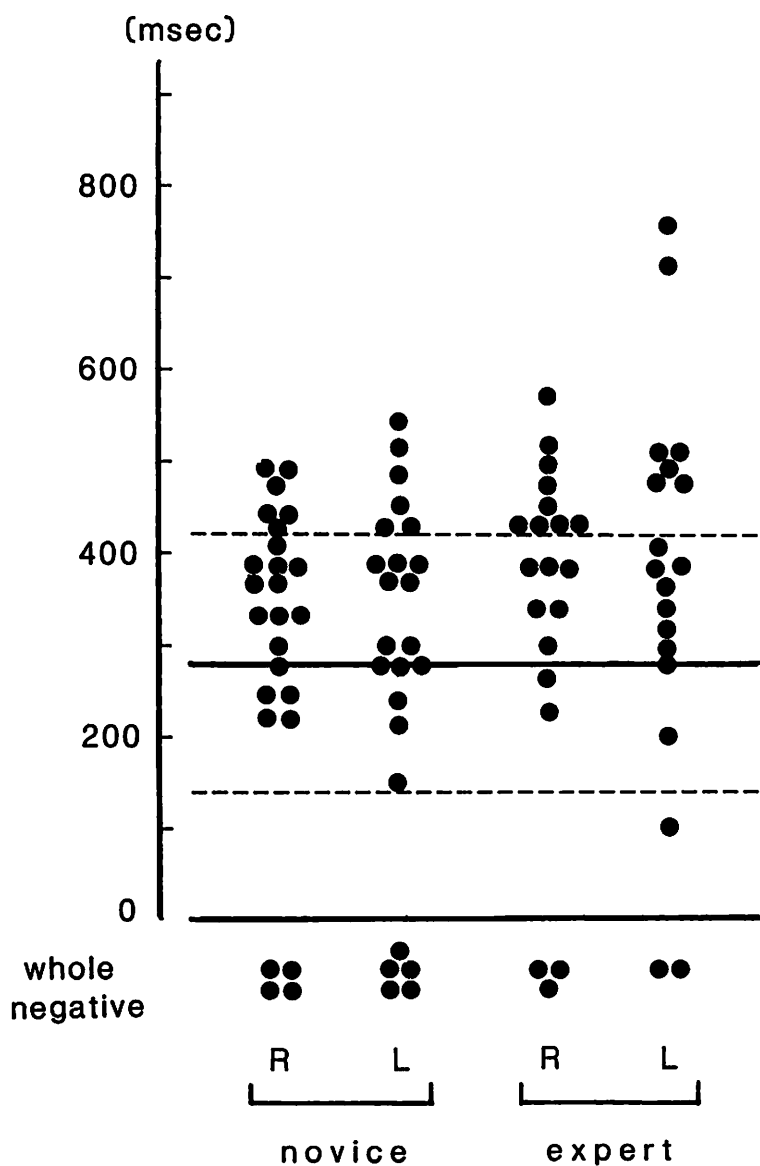


FIG. 2. Comparison of sonotubometry between novice and expert divers.

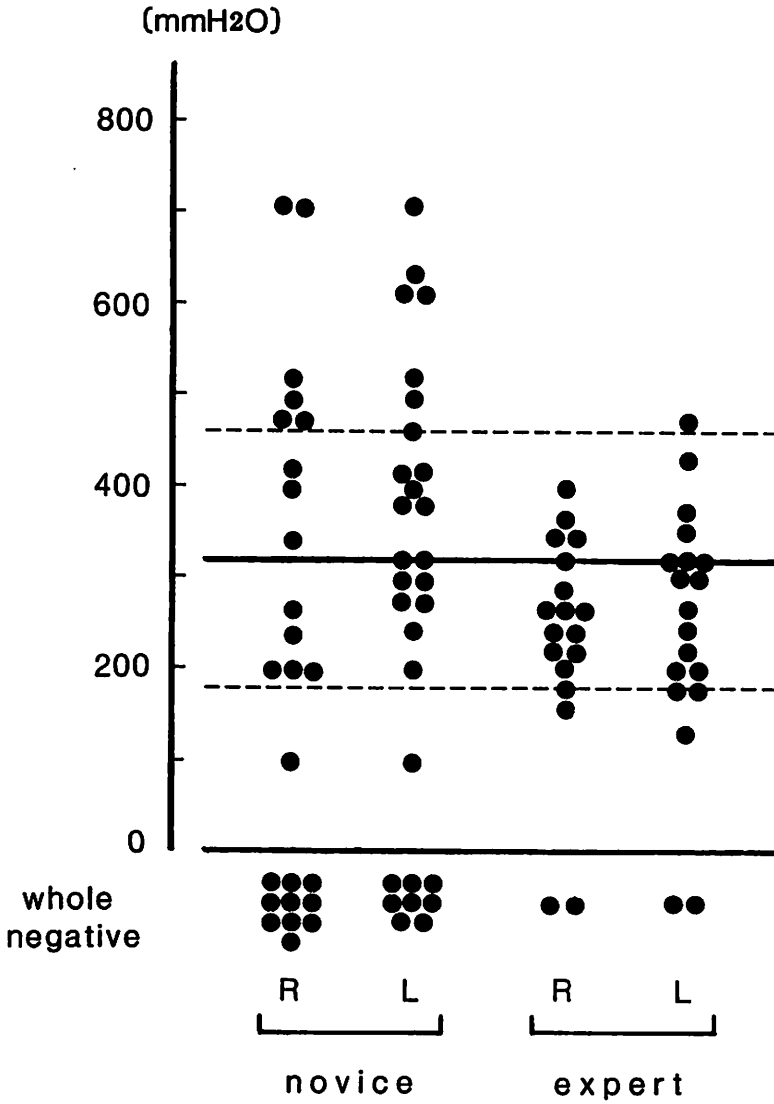


FIG. 3. Comparison of pressure required to open the Eustachian tube by Valsalva between novice and expert divers.

and Watanabe, 1984; Ohkubo and Watanabe, 1985). Among expert divers, instructors and occupational divers, the time required for continuous opening and closing of the Eustachian tube tended to be long and the same trend in swallowing movement was thought to be due to the result of acclimatization due to experience in opening the Eustachian tube while swallowing when diving.

The Eustachian tube opening rate by Valsalva was low in novice divers (52%). In comparison, the rate of opening for the experienced divers was much higher, 81%. Furthermore, even if the Eustachian tube opens in novice divers, they require a high pressure in the nasal cavity. In comparison, among many of the expert divers, the Eustachian tube opened at relatively low pressure and there was less variation, compared to novices (Table 3, Fig. 3).

TABLE 3.

Auditory Tubal Function of Barotraumatized Ears.

| | Control | Novice | Expert |
|---|------------------|------------------|------------------|
| Sonotubometry Positive | 28/32 (87.5%) | 16/23 (69.6%) | 9/11 (81.8%) |
| Intranasal Opening Time, Msec. | 288.5 \pm 34.6 | 369.3 \pm 42.5 | 402 \pm 39.9 |
| Valsalva Test Positive | 23/26 (88.5%) | 12/23 (52.2%) | 9/11 (81.8%) |
| Intranasal Opening Pressure, mmH ₂ O | 324.9 \pm 35.0 | 383.9 \pm 42.8 | 325.6 \pm 38.4 |

There were 7 novice divers who were unable to open the Eustachian tube with Valsalva and 3 who could not in 1 ear. Many novice divers found it difficult to open the Eustachian tube with Valsalva, and this is a cause for panic in water.

IV. INNER EAR BAROTRAUMA

If divers find it difficult to open their Eustachian tube with Valsalva, it is not just a matter that diving is hindered but there is a fear that this could lead to panic. This can be said to be a cause of the particularly high rate of diving accidents in less than 10 m of water.

To further understand how pressure in the middle ear adjusts to the changing environmental pressure, we conducted an experiment in monkeys.

We used a monkey and implanted a pressure sensor in the temporal bone (on the outside of the auricle up to the mastoid), and repeatedly added and reduced pressure in a hyperbaric chamber. When the surrounding pressure increases, the middle ear can withstand a pressure difference of up to 2,800 mmH₂O, but when surfacing, the maximum tolerance of pressure difference was narrowed to only 400 mmH₂O. Therefore, rather than the descent into the water, surfacing is more dangerous for the diver who fails to equalize pressure in the ears (Mano *et al.*, 1987). Imperfect adjustment of pressure while surfacing may result, not only in a rupture of the eardrum, but may also lead to rupture of the round window and the oval window (Bayliss, 1968; Lamkin and Axelsson, 1975; King, 1976; King, 1979). When inner ear damage occurs, the lymph is washed away, the outer hair cells are lost and clinically, it leaves after-effects such as dizziness, difficulty in hearing, ringing in the ears, and partial loss of hearing (Takahashi, 1985). In the case of novice divers who suffer from a stenosis of the Eustachian tube and dive regardless, even if they manage to descend, it is highly likely that they will suffer barotrauma when they surface, and this requires extreme caution.

V. COMPARISON OF DIVING PROFILES BETWEEN NOVICE AND EXPERT DIVERS

A. Characteristics of the Diving Recorder

The Adonis Diving Recording Computer System consists of four components: diving recorder (ADR), digital display (ADD), information adapter (AIA) and color printer plotter (APP) (Fig. 4). Of these, only the ADR and the ADD, which show water depth and time, are taken into the ocean. The ADR reads the initiation of dive at the point where the diver has dived to a depth of 60 cm below the water surface, and judges a dive finished when the diver rises to less than 40 cm below the surface of the water. ADR samples the water temperature and water depth in real time at 1 sec intervals, but depth measurement is registered only when there is a depth change of more than 50 cm. These data are stored in the ADR's memory (Shibayama *et al.*, 1987).

B. Subjects

We designated 35 trainees (13 men, 22 women) who were hoping to take a course in diving and divers who had been diving for less than 6 months as the novice diver group. The expert group consisted of 9 divers (6 men, 3 women) who were instructors or who had more than 3 years of diving experience.

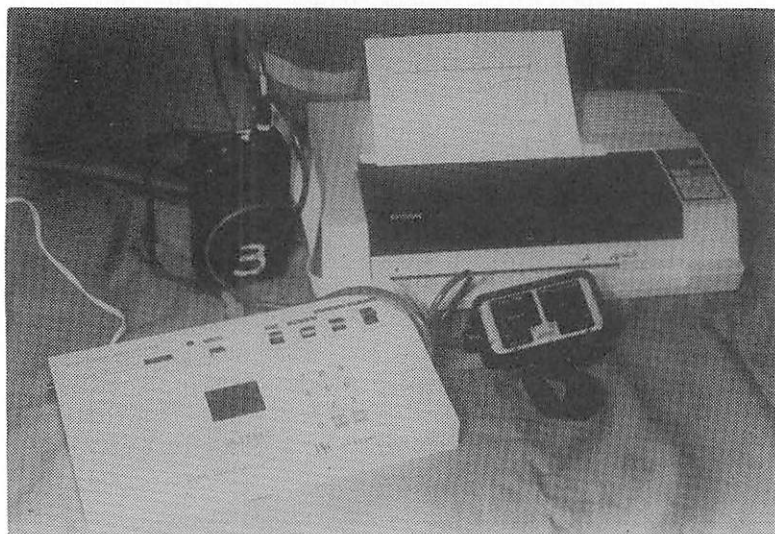


FIG. 4. Adonis Diving Recording (ADR) System for research work.

C. Procedures

We attached an ADR to each diver's waist and first had them dive vertically to a depth of 10 m with their head down, then had them repeat the procedure with their feet down in a standing position. We then brought the two groups (novice and expert) together and had them simultaneously descend to a depth of 10 m 3 times in a row. Each person's dive was recorded and comparisons made.

We studied the opening and closing function of the Eustachian tube by having the divers adopt the positions of head up, head down and lateral recumbent position. The objective is, of course, to discover the effect of varied body position on pressure adjustment in the middle ear.

D. Results

The majority of novice divers use Valsalva to open their Eustachian tube at around a depth of 3 m. The dive is halted at that depth if they are unable to do so properly (Fig. 5). In other words, as we showed before in the experiment with the monkey, the middle eardrum can withstand pressure up to a water depth of 2.8 m during descent. Novice divers try

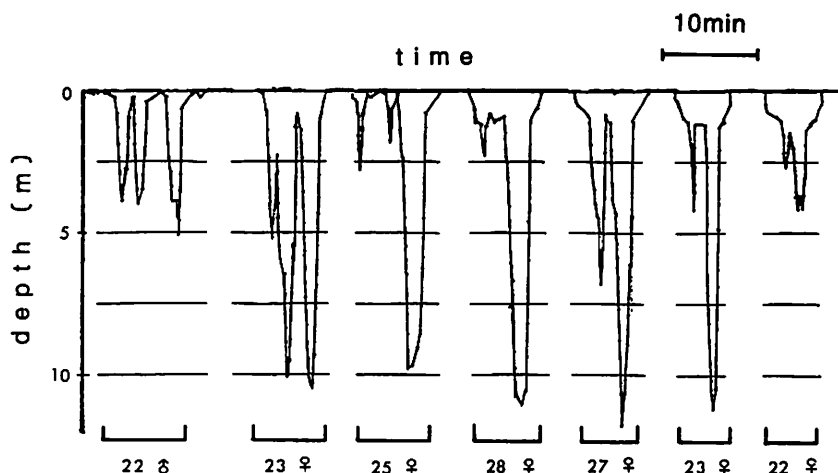


FIG. 5. Diving profiles of novice divers using two descent postures: first, head down; second, head up. Subject: I.D. number and gender are shown.

many times to open their Eustachian tubes with Valsalva at this depth. Once they successfully adjusted their ear pressure, they can proceed to a greater depth. However, as judged from their diving profiles, they find difficulty in reaching the target depth of 10 m.

Novice divers were first made to dive vertically downwards in a hand-stand position and then made to dive vertically with their feet down in a standing position. As shown in Fig. 6, it was more difficult for these divers to equalize pressure with Valsalva in the head down position as compared to the feet down position as indicated by a higher opening pressure required. Where the head was in the sideways position, we discovered that it was harder to open the Eustachian tube in the lower ear, in cases where the head was in the sideways position. The same phenomena were clearly shown in expert divers also.

When novice divers were sent down to dive with expert divers, the instructor can cleanly execute a box-type diving profile as recorded by ADR (Fig. 7), but the novice divers had difficulty descending and they repeated up and down movement 6 times from 4 m to 9 m to adjust the pressure in the middle ear. In the second dive they came closer to their target depth after repeating this process 3 times from 5 m to 9 m, whereas the expert diver reached the target smoothly and vertically. In the third dive to a depth of 5 m, after the expert diver's slow dive down, the novice divers were able to follow without failing to adjust their ear pressure.

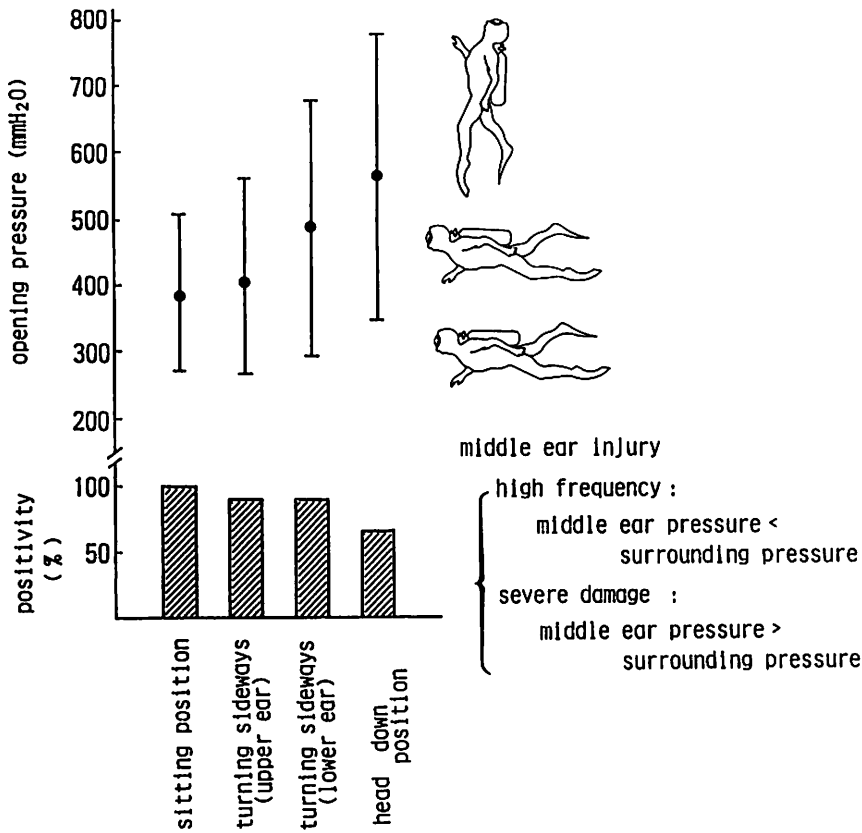


FIG. 6. Values of intranasal pressure at the auditory tube opening using Valsalva at different postures in novice divers.

Middle ear injury:

- high frequency: middle ear pressure < surrounding pressure
- severe damage: middle ear pressure > surrounding pressure

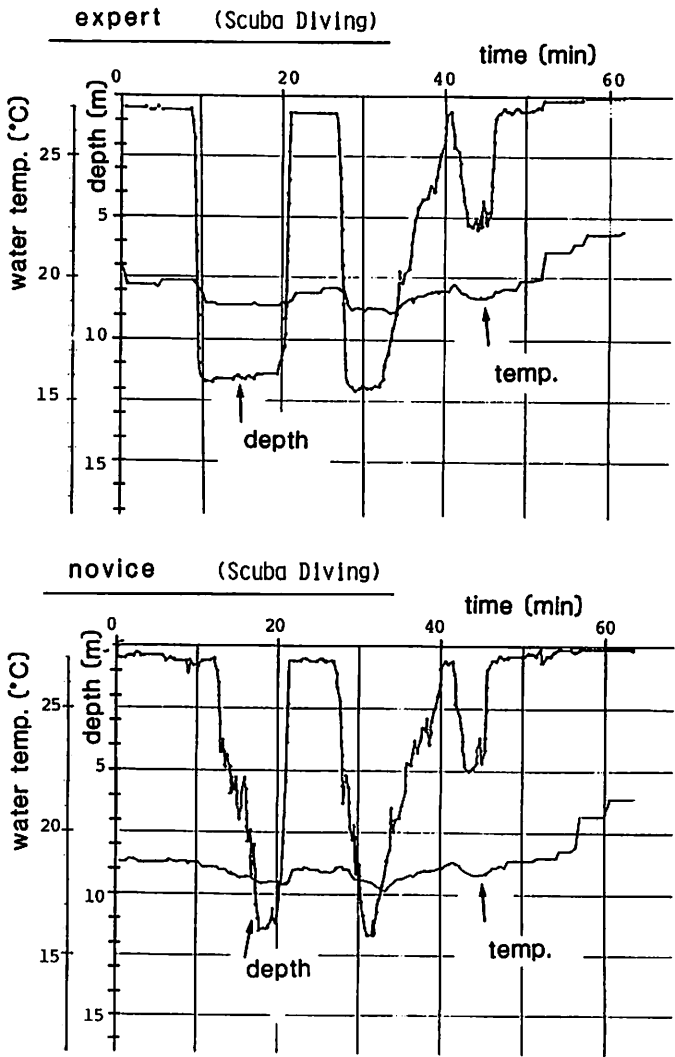


FIG. 7. Comparison of diving profiles between expert and novice divers.

VI. DISCUSSION

Recording diving data on the Adonis Diving Recorder clearly shows that often, novice divers cannot easily open the Eustachian tube at shallow diving depth (Fig. 5). This points to a probably frequent source of panic in the water. In other words, the novice fails to control the pressure of the middle ear with Valsalva maneuver loses his or her sense of ease in the water and feels dizzy or panics, leading to a diving accident. Almost half (45.5%) of diving accidents occur within a depth of 5 m and 65.8% occur within a depth of 10 m. This finding leads us to believe that many of these events are associated with failure to open the Eustachian tube to the middle ear (Table 2). Moreover, when we examined the occurrence of accidents in accordance with number of years of diving, divers who had a diving accident when practicing in the ocean for the first time accounted for 30.8% of the total (Table 1) indicating how important it is to prevent accidents in novice divers (Mano and Shibayama, 1987).

The hearing test results on ear-damaged novice divers showed various types of hearing loss such as sound propagation, sensorineural and compound hearing disorders. In comparison, the pattern of hearing loss with expert divers was all high tone loss type. The high level of damage to the ear drum and sudden cathodal depression of the middle eardrum that is experienced by some occupational divers and experts are mostly sensorineural hearing loss of high tone. These problems are also related to past ear barotrauma experience. The rupture of the inner ear though healed, is still highly susceptible to damage by the pressure difference (Ohkubo *et al.*, 1987, Takahashi, 1985). This is caused by damage to the inner ear due to relatively high pressure changes in the middle ear. This is particularly true for experienced and occupational divers who have more potential for experiencing excessive sudden pressure build-up in terms of the greater depths to which they dive.

Novices, on the other hand, are not skilled at adjusting the pressure of the middle ear and this results in various complex types of hearing loss. Among novice divers apart from simple differences in the environmental pressure on the middle ear, excessive holding of the breath may be a contributory factor, causing unexpected effects on the middle ear and inner ear resulting in various types of hearing loss.

Damage to the ear from sports diving is mostly caused by the incomplete function of the Eustachian tube. In the case of adults, ventilation of the middle ear cavity can be carried out only by active Eustachian tube opening and closing through swallowing and the Valsalva method. Closed Eustachian tube, like middle ear otitis in children, makes divers susceptible to functional damage even with small pressure differences

(Ohkubo, 1984). The middle ear cavity automatically expels the air by pushing the Eustachian tube open when the pressure in the middle ear cavity is at a high pressure environment. On the other hand, when this does not occur, one tries to ventilate the Eustachian tube by swallowing, jaw movement, and Valsalva maneuver. As shown in the experiment with the animal, if there is a relative difference in pressure of more than 2,800 mmH₂O, the eardrum ruptures, or results in various sensorineural losses through window ruptures (Farmer, 1977; Cantekin, 1977). It often results in rupture of the ear cavity when the pressure in the middle ear cavity is less than surrounding environmental pressure. The opposite squeeze results when the middle ear cavity pressure exceeds the surrounding environmental pressure. In terms of the level of damage, the latter situation is far worse and this is particularly relevant during surfacing rather than when descending.

With the aim of quantifying the rate of general public who may suffer damage to their ears through changes in pressure, we randomly selected 25 adults, whose ears are otherwise normal, and examined the condition of the Eustachian tube of the 50 ears by exposing them to a depth of 10 m (2.0 ATA) in a hyperbaric chamber. Among the normal adults who noted no irregularities during the examination, under the high pressure environment nevertheless there were 3 persons (12%) who could not ventilate their middle ear cavity. In clinical ear examination, it is normally difficult to detect people who will not be able to adjust the ear pressure well under a pressure environment. Moreover, it must be considered that the approximately 12% of the people who wish to start diving either have no experience at adjusting ear pressure (Valsalva method) or do not know how to do it at all. The overwhelming proportion of novice divers need to be taught about Valsalva maneuver. There are many injuries among those who have no experience at adjusting ear pressure, while experts tended to fall victim to barotrauma only if they dived with a cold or ignored the incomplete Eustachian tube condition.

As a means to avoid ear damage in novices, not only instructors but also doctors should ask "Can trainees perform the Valsalva?" and know how to teach students to perform it correctly. It is also necessary to conduct sufficient training of ear pressure adjustment and exert effort to avoid accident. For this purpose, it is necessary to find out if trainees can do the Valsalva properly during usual ear examination. This should first be accomplished using tympanometry in a quiet environment. The tympanometry should be recorded at a quiet time, the Valsalva conducted and immediately afterwards another tympanometry made. If Valsalva is completed, the peak should be moved to the positive pressure side. If this phenomenon is observed, it is proof that Eustachian tube ventilation through Valsalva is successful.

If divers, even novice divers, can successfully equalize ear pressure, the profile of diving recorded by the ADR will become smoother, and approach the ideal profile of expert divers, as shown in Fig. 7. Divers will be able to smoothly reach their target depth, which also gives them confidence and prevents panic. In other words, the improved diving skill should contribute to reducing diving accidents.

Being able to smoothly perform the correct ear pressure adjustment enables the diver to relax in water and is the best method of preventing panic. As can be seen from the ADR recordings, if both novice and expert divers can smoothly dive to their target depth without being bothered by adjustment of air pressure, a considerable portion of the accidents that occur within 10 m may be avoided, and thus help ensure the safety of sports diving. Many of the novice divers who encountered dangerous situations, though not to the point of death, supported this assumption during the interviews.

Sports divers have a log book but there are many cases when recording on the diving depth and time passage are vague and unreliable (DEMA, 1988; NAUI Japan, 1985; Graver, 1985). The ADR completely supplants that potential shortfall and can document improvements in the diving skill of novice divers. For instructors and expert divers, it tells them the accurate diving depth and to restrict diving time, thereby helping to avoid major diving load and contributing to the prevention of decompression sickness. It is no exaggeration to say that the basis of safe diving is determined solely by diving depth and time. ADR can be used continuously for up to 8 hr and can therefore realistically be used for all diving activities.

VII. CONCLUSION

It is clear that ADR shows distinct differences in diving profiles between novice and expert divers. The diving accidents of novice divers are caused mostly by imperfect pressure adjustments of the middle ear cavity, since this very often results in panic. The depth at which this occurs is usually around 3 m, which is consistent with diving accident statistics.

Preventing ear damage involves preventing unbalanced pressure inside and outside the middle ear cavity at shallow depth. If the diver can descend smoothly, even novice divers will be assured of safe sports diving. This in turn improves diving skill. These factors clearly show the advantages in using an ADR, whereby a record of diving activity can provide feedback, and facilitate diver training.

Two thirds of diving accidents occur at a depth of less than 10 m and half of those accidents involve novices. It is important to adjust pressure

on the middle ear cavity to prevent panic in the water. ADR can be used to evaluate how skillfully the diver ventilates the Eustachian tube. The ADR is not only beneficial for the novice diver but also guarantees safe diving for the expert diver, by providing reliable depth-time documentation. We wish this report will encourage a wider use of the ADR in the field conditions.

REFERENCES

- Bayliss, G.J.A. Aural barotrauma in naval divers. *Arch. Otolaryngol.* 88:141-147, 1968.
- Cantekin, E.L. Normal and abnormal middle ear ventilation. *Ann. Otolaryngol. Suppl.* 41:1-15, 1977.
- DEMA (Diving Equipment Manufacturer's Association). *Revised Instructional Standards: Minimum Course Content for Entry-Level SCUBA Certification* (in Japanese). Tokyo: NASDA Japan, p. 1-13, 1988.
- Farmer, J.C. Diving injuries to the inner ear. *Ann. Otolaryngol.* 30:1-20, 1977.
- Graver, D. *PADI Diving Manual* (Japanese edition). Tokyo: PADI International Japan, p. 1-166, 1985.
- Japan Association of Underwater Exploitation. *Annual Report of 1988 on Leisure Diving* (in Japanese). Tokyo: Nikkan Kaiji Tsushin Co., p. 1-34, 48-55, 1988.
- Japan Maritime Safety Board. *Safety of Diving* (in Japanese). Tokyo: Japan Assoc. for Disaster Relief in Water, p. 1-23, 1988.
- King, P.F. Otic barotrauma. *Audiol.* 15:279-286, 1976.
- King, P.F. The Eustachian tube and significance in flight. *J. Laryngol.* 93:659-687, 1979.
- Kitajima, T., Y. Mano, and M. Shibayama. Sports diving accidents and safety (in Japanese). In: *Proceedings of Second Symposium on Diving Technology*. Tokyo: Japan Association of Underwater Exploitation, p. 161-168, 1980.
- Lamkin, R. and A. Axelsson. Experimental aural barotrauma, electrophysiological and morphological finding. *Acta Otolaryngol. Suppl.* 335:1-24, 1975.
- Mano, Y. and M. Shibayama. *Recommendations for Diving Safety* (in Japanese). Tokyo: Japan Underwater Diving Federation, P. 1-22, 1987.
- Mano, Y., T. Shibayama, and T. Mizuno. Analysis of SCUBA diving accidents in Japan. In: *Proceedings of the Ninth Symposium of UJNR Diving Physiology and Technology Panel*. Yokosuka, JAMSTEC, p. 1-9, 1988.
- Mano, Y., M. Shibayama, T. Mizuno, and J. Ohkubo. Research on sonotubal function in SCUBA diving (in Japanese). *J. Japn. Hyperbar. Environ. Med.* 22:123, 1987.
- NAUI. *SCUBA Manual* (Japanese edition). Tokyo: NAUI Japan, Tokyo, p. 1-152, 1985.
- Ohkubo, J., Y. Mano, and M. Shibayama. Diving (SCUBA) accidents and Eustachian tube function, otic barotrauma (in Japanese). *Japn. J. Otolaryngol.* 59:573-578, 1987.
- Ohkubo, J. Middle ear otitis of children (in Japanese). *Japn. J. Otolaryngol.* 56:857-860, 1984.
- Ohkubo, J. and I. Watanabe. Sonotubometry (in Japanese). *Japn. J. Clin. Otolaryngol.* 77:1747-1754, 1984.
- Ohkubo, J. and I. Watanabe. Function of Eustachian tube by Valsalva and intranasal pressure (in Japanese). *Japn. J. Clin. Otolaryngol.* 78:339-345, 1985.
- Shibayama, M., Y. Mano, and J. Ohkubo. A study of Eustachian tube function in fishermen divers (in Japanese). *Japn. J. Indus. Health* 29:265-270, 1987.
- Takahashi, S. Inner ear barotrauma. *Bull. Tokyo Med. Dent. Univ.* 32:19-30, 1985.

the first of these is the fact that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The second fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike.

The third fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The fourth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The fifth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike.

The sixth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The seventh fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The eighth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike.

The ninth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The tenth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The eleventh fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike.

The twelfth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The thirteenth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike. The fourteenth fact is that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the government and the people alike.

6

Hyperbaric Medicine During Spaceflight

J.B. Boyce and J.R. Davis

| | |
|--|-----|
| I. Introduction | 105 |
| II. Spaceflight Physiology | 107 |
| A. Space Motion Sickness | 107 |
| B. Body Fluid Shift | 107 |
| III. Medical Kit | 107 |
| IV. EVA System | 108 |
| V. Decompression Sickness | 108 |
| A. Relative Risks | 110 |
| B. Altitude DCS | 110 |
| C. Treatment | 112 |
| VI. Escape System | 112 |
| VII. Manned Maneuvering Unit | 112 |
| VIII. Space Station Freedom | 114 |
| A. Health Maintenance Facility | 114 |
| B. Hyperbaric Airlock | 115 |
| IX. The Future | 115 |

I. INTRODUCTION

Since the beginning of man's endeavor to escape the bounds of Earth, he has provided for himself the basic requirements for life support that are normally given so easily by nature. This paper will deal mainly with our efforts at NASA to provide one such requirement, an adequate pressure envelope and a breathable atmosphere during space flight extravehicular activity (EVA). Man's ability to work during EVA has become an essential component of our space capabilities, and will continue to receive much attention in the future for Space Station Freedom construction and

beyond. Although the Soviets were the first to actually perform EVA when Cosmonaut Alexei Leonov went outside his Voshkod 2 capsule for 24 min on March 18, 1965, they had only one other EVA up to 1977. Since then, they have performed EVA infrequently or when essential to Salyut or Mir repair. We are far behind the Soviet Union in total manned space flight time (12 to 4.7 man-yr), but the United States is still the leader in terms of EVA, including total EVA time (300 US hours to 53 hours for the Soviets) and in terms of capabilities during EVA.

The United States had its first EVA in June, 1965, when Ed White went outside during Gemini 4 in a 3.7 psi suit after a 3 hr oxygen pre-breathe. Gemini 9 demonstrated the necessity of adequate thermal control when fogging of Gene Cernan's visor prevented completion of EVA objectives during a strenuous EVA. To reduce the workload and avoid this problem, foot restraints were first used on Gemini 12 by Buzz Aldrin in a 2 hr, 8 min EVA.

The Apollo project proved the value of EVA, when 12 men spent 160 man-hr on the Moon, covering 60 miles on foot and in the lunar rover, while collecting 2,196 soil and rock samples. The EVA suits used were pressurized to 4.3 psia with 100% oxygen, and cabin pressure was 5 psia with 100% oxygen also. The cabin was maintained at 14.7 psia with a normal air (21% oxygen) composition until just before liftoff, and then depressurized to 5 psia. A 3 hr 100% oxygen prebreathe was used to try to prevent decompression sickness (DCS) after launch, but in his books, Michael Collins reported a suspicious shoulder pain that probably represents the bends.

The potential benefits of EVA were nowhere more evident than in the first Skylab mission. After initial failure of a solar panel and the loss of power and cooling capability, Dr. Joe Kerwin and Pete Conrad saved the entire project by rigging a solar shade and freeing a solar panel during two separate EVAs. When the crew first entered the Skylab, the internal temperature was up to 160 degrees Fahrenheit and was almost uninhabitable.

The Soviets also have claim to the first female EVA, which was done July 25, 1984 by Svetlana Savitskaya in an EVA which demonstrated an electron beam welding tool which is still undeveloped in the West. The Soviet EVA capability consists of a variable pressure suit which is normally kept at 5 psia. During periods when increased hand/finger dexterity is required, the suit pressure may be lowered to a minimum 3.8 psia. Although this would increase the risk of DCS, there have been no reports of this from the USSR. The training, procedures, and detailed capabilities of Soviet EVA systems are not publicly known in the West.

II. SPACEFLIGHT PHYSIOLOGY

A. Space Motion Sickness

Prior to discussion of the Space Transportation System (STS) EVA suit, an understanding of the basic phenomenon of spaceflight physiology is needed. There are several effects of space flight that may have an effect on an EVA crewmember's health, including fluid shifts, Space Motion Sickness (SMS), and for long term flight, cardiovascular deconditioning and bone calcium loss. The most troublesome effect of space flight for short term flights is the malady known as space motion sickness (SMS), which affects about two-thirds of the astronauts on their first flight. Nausea, headache, malaise, and vomiting are a few of the symptoms of this self-limiting illness which typically lasts no more than 4 days on orbit. While no definite cause is known at present, the consensus of opinion among NASA researchers indicates that SMS is probably due to an alteration in the way acceleration is perceived by the vestibular organs. Whatever the cause, SMS may lead to serious limitations on crewmember performance over the first two days of a mission, and must be considered in any decision for early mission crew activities, including EVA.

B. Body Fluid Shift

A second well demonstrated space flight phenomenon is the shift of body fluids from the lower extremities to the head and trunk after the initial exposure to microgravity. This shift leads to an initial diuresis and eventual loss of about 10-15% of the blood plasma volume which can result in orthostasis on return to Earth. These two short term space flight effects, SMS and fluid shifts, set the stage for the baseline health of the crewmembers for missions of 1-2 weeks. For longer missions, the deconditioning effects from lack of gravitational stress on the muscles and bones must also be included. A rigorous exercise program may be needed to counteract these effects, but the exact "prescription" is still unknown at present due to our lack of expertise in long duration flights.

III. MEDICAL KIT

To treat any routine medical problems that may occur during STS flights, a small shuttle medical kit containing routine and emergency medications is carried on each flight, along with a contaminant cleanup kit for any toxic spills that may occur. The medical kit contains basic oral and IV medications, bandages, examination equipment, emergency cardiac

medicines, and a 100% oxygen manually triggered resuscitator, but does not contain a defibrillator at present. Two crewmembers from each flight are trained in the use of the various medications and the procedures that may be done on orbit, with flight surgeon consultation from over the air-to-ground communications as necessary.

IV. EVA SYSTEM

The space shuttle EVA system consists of a 4.3 psia, 100% oxygen suit with a liquid cooled garment for thermal control. The EVA crew leaves a cabin that may be either 14.7 psia and 21% oxygen or 10.2 psia, 27% oxygen. When the EVA crew leaves a 14.7 psia cabin, they must prebreathe with 100% oxygen for a minimum of 4 hr for denitrogenation according to the protocols now established. A preferred method utilizes a cabin depressurization to 10.2 psia for 12 to 24 hr prior to the EVA, with a 40-75 min oxygen prebreathe prior to depressurization to the suit's 4.3 psia (Fig. 1). For any break in prebreathe with exposure to less than 95% oxygen, a two-for-one payback time (e.g., 10 min for a 5 min break) is required prior to final depressurization. EVAs are normally planned for up to 6 hr duration, but this time does not include 15-30 min for suit donning and doffing. An additional contingency oxygen supply is also provided by a secondary oxygen pack that provides a minimum of 30 min additional time at a reduced pressure of 3.9 psi.

The EVA suit is a combination of several components shown in Fig. 2. Thermal control is provided by a liquid cooled garment, as mentioned. This system will handle up to 2000 BTU for up to 15 min (500 kcal/hr) at peak loads, 1600 BTU for up to an hour (400 kcal/hr), or 1000 BTU/hr for 7 hr. Carbon dioxide and other suit parameters are monitored through telemetry that is provided every 2 min when in radio range. CO₂ is kept below 7.6 mmHg by lithium hydroxide canisters that may be changed between EVAs. A urine collection device is provided with a capacity of 32 oz, along with a food bar and up to 21 oz of water that may be consumed during EVA.

V. DECOMPRESSION SICKNESS

The requirements for denitrogenation profiles were established by NASA with outside consultants over many years. A summary of the experimental exposures of over 600 volunteer subjects is shown in Fig. 3. Although higher EVA suit pressures are desirable for decompression sickness (DCS) prevention, they carry penalties in terms of suit mobility (especially hand function). Lower pressure suits, on the other hand, may

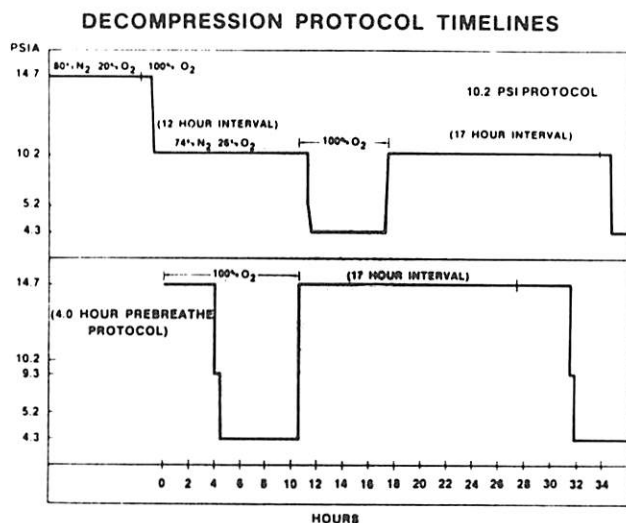


Fig. 1. Timelines and breathing gas composition when depressurizing from space shuttle cabin environment, either 14.7 psia or 10.2 psia, to extravehicular activity (EVA) suit pressure of 4.3 psia.

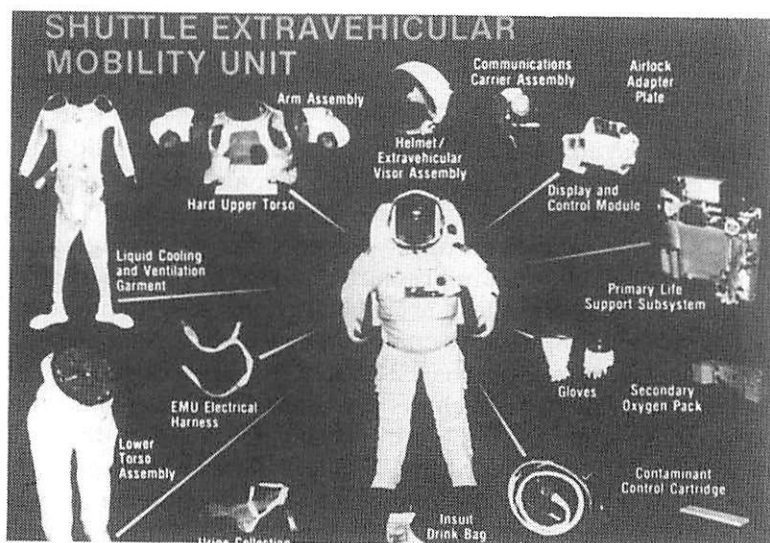


Fig. 2 Components of space shuttle EVA suit.

allow more ease of hand/finger function, but have more demanding and time consuming denitrogenation requirements, along with a higher risk of DCS.

A. Relative Risks

For calculation of relative risks for the various profiles that are considered, an R factor calculation (Fig. 4) is done. The R factor, a variation of Haldane's rule, is a ratio based on the initial tissue nitrogen stores compared to final ambient pressure based on "slow" 360-min half time tissue denitrogenation. The factor does not take into account the exposure time at the various pressures, however. This calculation allows comparison of the various profiles and risks involved based on the previous experience as shown in Fig. 3. As can be seen, the incidence of bubbles is much greater than the incidence of DCS, which is in turn much higher than the incidence of performance limiting DCS at all R factors. The present requirement for either EVA denitrogenation profile (14.7 or 10.2 psia) has been a maximum R factor of 1.65. The actual average R factor for the shuttle EVAs to date has been a safer 1.60, however. This may account for the fact that there have been no reports of any DCS symptoms from any shuttle EVA crewmember. For emergency EVA, our recommendation has been to provide a minimum of 2.5 hr of 100% oxygen prebreathe for a six hr EVA from a 14.7 psia cabin in order to lessen the risk of DCS to approximately 50%. For any EVA, an aspirin protocol is used to try and prevent platelet interactions with bubble surfaces that may form.

B. Altitude DCS

Although the symptoms of altitude DCS are typically mild and easily treated in comparison to diving DCS, they can also be life threatening if untreated. Altitude DCS can be seen as the equivalent of saturation diving DCS in some ways, since the crewmember is leaving a saturated gas equilibrium state at cabin pressure and venturing into a lower pressure environment. The gas load is obviously much less than in the diving setting, however. Symptoms normally occur within 2 hr of depressurization, and onset is rare later than 24 hr after repressurization. Interestingly, diving neurologic DCS shows a predominance of spinal symptoms, with brain symptoms noted more in altitude DCS. As in diving DCS, a predominant symptom for altitude DCS is still large joint pain, and fortunately, air embolism is rare in aviation. Treatment with the same 2.8 to 6 ATA tables used for diving DCS is the present standard, and a good prognosis may be expected.

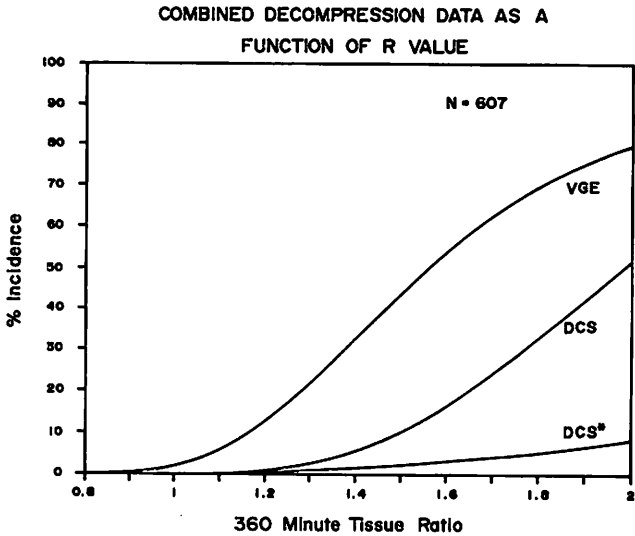


Fig. 3. Percent incidence of venous gas emboli (VGE) or decompression sickness (DCS) symptoms as a function of the calculated ratio of tissue N₂ before decompression to final N pressure. Tissue half time was assumed to be 360 min. VGE = venous gas emboli, DCS = decompression sickness, DCS* = symptoms resulting in test termination, or delayed symptoms.

EVA PREBREATHE PROTOCOL

THE R VALUE IS DEFINED AS THE ANALYTICAL RATIO OF TISSUE N₂ PRESSURE PRIOR TO DECOMPRESSION DIVIDED BY THE FINAL PRESSURE.

THE INITIAL N₂ PRESSURE IS ASSUMED TO BE EQUAL TO THE AMBIENT N₂ PRESSURE (EQUATION 1).

TISSUE N₂ IS ASSUMED TO CHANGE DURING EXPOSURE TO A BREATHING GAS WITH AN ALTERED N₂ PRESSURE AS DEFINED BY A NON-LINEAR HALF TIME EQUATION (EQUATION 2) WHERE THE CRITICAL HALF TIME IS 360 MINUTES.

EQUATION 1

$$R = \frac{P_{N_{2TIF}}}{P_F}$$

WHERE R = R VALUE
 $P_{N_{2TIF}}$ = FINAL TISSUE N₂ PRESSURE
 P_F = FINAL PRESSURE

EQUATION 2

$$P_{N_{2TIF}} = P_{N_{2TIO}} + [(P_{N_{2A}} - P_{N_{2TIO}}) > 1 - e^{-KT}]$$

WHERE $P_{N_{2TIF}}$ = FINAL TISSUE N₂ PRESSURE
 $P_{N_{2TIO}}$ = ORIGINAL TISSUE N₂ PRESSURE
 $P_{N_{2A}}$ = THE N₂ PRESSURE IN THE BREATHING GAS
E = BASE OF THE NATURAL LOG
T = PREBREATHE TIME IN MINUTES
K = 0.693/T 1/2

WHERE T 1/2 = TISSUE IN HALF SATURATION TIME IN MINUTES

Fig. 4. EVA prebreathe protocol.

C. Treatment

In the event that the conservative measures taken for prevention of DCS in an EVA crewmember are not enough and the bends develop, capability for limited treatment on orbit has been developed. After a crewmember returns to the cabin and the cabin is pressurized to 14.7 psia, the crewmember will hopefully have a resolution of symptoms. If not, the crewmember will be kept on 100% oxygen while a Bends Treatment Apparatus (BTA, Fig. 5) is placed on the oxygen delivery system of the EVA backpack. The BTA is essentially a higher pressure "pop-off" valve that allows cycling of the pressurization system and the suit in a stepwise fashion up to a maximum of 8 psi above ambient pressure. In combination with the maximum allowed cabin pressure (16 psia), the 1.6 ATA treatment will hopefully be adequate for resolution of DCS symptoms. If unsuccessful, however, return to Earth may be required. Landing sites will be chosen based on availability of and location of hyperbaric treatment centers, acceptability as a landing site, and other factors. Further transportation may be needed since a hyperbaric capability does not exist at any of the three primary landing sites; Edwards AFB, California, White Sands Space Harbor, New Mexico, or Kennedy Space Center, Florida.

VI. ESCAPE SYSTEM

Since the Challenger accident, an escape system has been added to the shuttle fleet permitting escape from the Shuttle during stable gliding flight by sliding down a telescoping pole from the left side crew hatch. As part of this capability, a new Launch and Entry Suit (LES), was developed to protect the crewmember from hypoxia and cold water exposure. The suit provides a G suit, a basic partial pressure suit capability which will provide a 2.8 psia differential pressure to the crewmember, and a thermal barrier to protect them from 45°F water for up to 6 hr, or up to 24 hr if used with a raft. The DCS protection of this LES in the event of rapid cabin depressurization lasting more than 30 minutes, without any oxygen prebreathe, is minimal.

VII. MANNED MANEUVERING UNIT

The EVA suit has worked well during numerous EVAs, and has enabled satellite capture and repair, and other strenuous procedures that would not have been possible otherwise. Several firsts have been accomplished, including the first female EVA done by Kathy Sullivan during mission 41-G in October 1984. Another first was the EVA by

BENDS TREATMENT APPARATUS

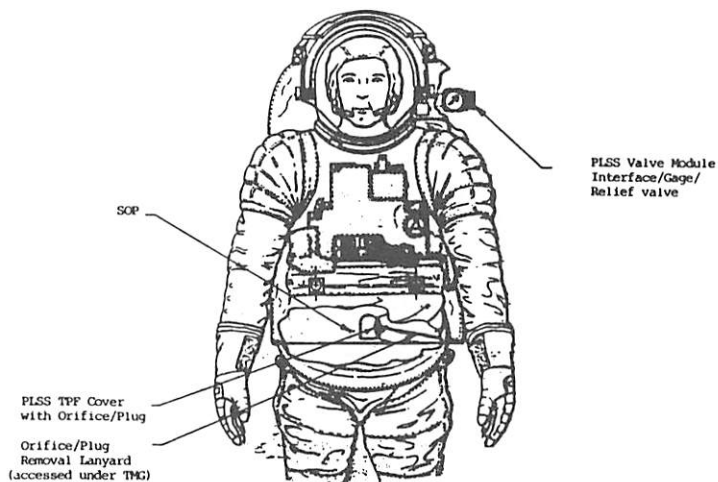


Fig. 5. The bends treatment apparatus allows treatment at a pressure of 8 psi above a maximum cabin pressure of 16 psia.

HMF COMPONENT HARDWARE

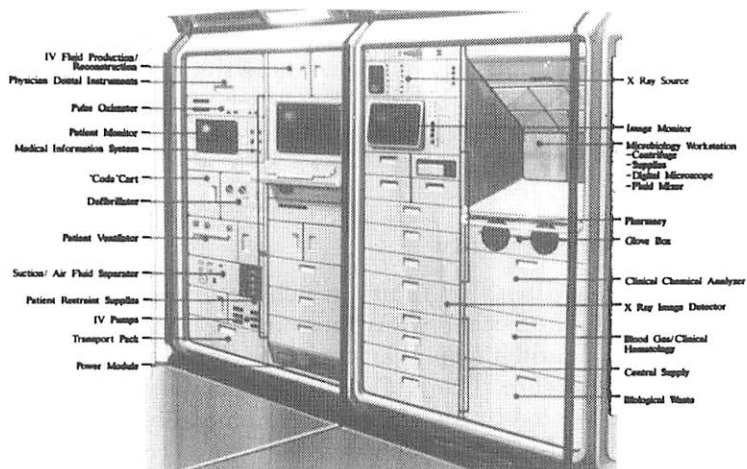


Fig. 6. Health Maintenance Facility on board Space Station Freedom.

Bruce McCandless in February, 1984, where the Manned Maneuvering Unit (MMU) was first used. This completely separate spacecraft allows maneuvering away from the Orbiter, and has been used for several spectacular missions. Three types of EVA are currently designated by NASA; scheduled, unscheduled, and contingency. A scheduled EVA will occur no earlier than flight day 3 unscheduled EVA used prior to this time only if the crew is healthy and fit for EVA (i.e. not suffering from Space Motion Sickness). A contingency EVA will be used for safe return of the crew or Orbiter as needed. The EVA crewmembers will always be tethered to the orbiter and will use foot and hand holds as needed to accomplish their objectives unless using the MMU.

VIII. SPACE STATION FREEDOM

For the Space Station Freedom era, a new level of capability in EVA systems will be needed. Freedom will be constructed over several years, requiring 17 flights of the Shuttle as now planned. EVA will play a major role in the construction and maintenance of the station, and may be used up to 2-3 times a week. To meet the challenge of providing frequent EVA support while lessening the risk of DCS even further, a "zero prebreathe" EVA suit is being developed in two separate designs at NASA's Ames Research Center and Johnson Space Center. For Space Station Freedom, the requirements state that the R factor can be no greater than 1.4, a substantial change from the shuttle era. Since a 100% oxygen prebreathe would take too long and the cabin for Freedom will always be kept at 14.7 psia, the suit must have a pressure of no less than about 8 to 8.3 psia, and may utilize a gas mixture rather than 100% oxygen. At this pressure level, the incidence of bubbles should be no more than 20%, and the incidence of pain from DCS should be very low. Much attention is presently being given to providing a functional hand/glove fit to operate at these higher pressures.

A. Health Maintenance Facility

In the event of an illness or injury which occurs on Space Station Freedom despite these and other safety measures, a Health Maintenance Facility (HMF, Fig. 6) and a Hyperbaric Airlock (HAL) will also be used to provide crew health care. The HMF will be a compact diagnostic and therapeutic facility located in the US habitation module, with capability to treat the majority of medical problems that may occur on Freedom. With the life support equipment from the HMF, including a ventilator and defibrillator, ACLS and ATLS level procedures may be provided for a

crewmember. State-of-the-art computer diagnostic and telemedical capability is also planned, and will aid the HMF medical officer in treatment. There are no current plans for a surgical capability or requirements for a physician as part of the crew at present.

B. Hyperbaric Airlock

The Hyperbaric Airlock (HAL) will be one of the two station airlocks which has been modified to accept pressures up to 6 ATA for periods up to 11 hr. Although there is much controversy concerning the necessity of 6 ATA versus 3 ATA treatment for air embolism, the consensus of our consultants forced use of the higher pressure. Using USAF hyperbaric treatment protocols as a starting point, this multiplace chamber will offer room for one patient and an attendant using masks or hoods for provision of breathing gas mixtures. The chamber atmosphere may have a decreased percentage of oxygen at higher pressures (i.e. 6-15%) to provide a fire safety margin to compensate for the increased difficulty of providing fire suppression in microgravity. This method may provide an improved safety margin for fire, but it will worsen the risk of increased absorption of nitrogen for patients or attendants who are unable to wear masks or hoods. The resultant increased DCS risk and nitrogen narcosis must therefore also be considered as a potential hazard to be weighed against the benefits of a decreased oxygen chamber atmosphere. Halon and nitrogen purges are being investigated as substitutes for water, which may not quench a microgravity fire. Studies to investigate these areas will be needed before final decisions on chamber atmospheres and procedures.

IX. THE FUTURE

Several questions remain concerning the effects of spaceflight and microgravity on decompression sickness risk and EVA. These questions include:

1. What is the effect of microgravity on lung blood flow and denitrogenation?
2. How will suit hand function and operational demands be balanced with long term risks of DCS and physiologic requirements?
3. How will EVA work and exercise alter the risk of DCS?
4. How will pharmaceuticals work in microgravity? How will absorption, distribution, and elimination be affected?

5. What will be the effective countermeasure to cardiovascular and muscular deconditioning?
6. What treatment profiles are really necessary to treat EVA DCS and/or air embolism? How will fire prevention modifications to chamber atmospheres affect nitrogen narcosis and attendant DCS risks?

These and other questions will continue to be investigated for the future development of EVA capabilities. Although the risks of EVA have, to date, been managed successfully, continued development of equipment and procedures will be necessary to make EVA a routine part of space flight. Space remains a hostile environment that will not suffer ignorance, lassitude, or indifference. EVA and a manned presence in space offer rewards far beyond the initial investment. The U.S. Space Program will continue to pursue these rewards in the safest possible manner to ensure our future in exploring our universe.

7

Hyperbaric Oxygen in a Modern Hospital Practice

*Mahito Kawashima, Hiroaki Tamura,
Katsuhiko Takao and Teruhiko Tsunosue*

| | |
|---|-----|
| I. Introduction | 117 |
| II. Method of HBO Administration | 118 |
| A. The Chamber | 118 |
| B. Patients | 119 |
| III. Results | 122 |
| A. General | 122 |
| B. Osteomyelitis | 123 |
| C. Chronic Obliterative Vascular Diseases | 126 |
| D. Necrotizing Fasciitis | 128 |
| IV. Discussion and Conclusion | 128 |
| References | 129 |

I. INTRODUCTION

Hyperbaric oxygen (HBO) therapy was begun by Saito in 1958 in Japan, who treated patients with apoplexia. In 1965, Wada treated burn and carbon monoxide toxication with HBO. Ever since the Japanese Society of Hyperbaric Medicine was established in 1966, HBO therapy has been common all over Japan. In 1985, there were 213 active chambers. The number of monoplace chambers was 172, and the number of multiplace chambers was 41. The current total number of chambers seems to be more than 300. My first work in the field of hyperbaric medicine started at Kyusyu Rosai Hospital in 1972. The first chamber in Kyusyu Rosai Hospital was constructed in 1960. The main use of this chamber was for the treatment of decompression sickness and carbon monoxide intoxication. We gradually increased the number of cases, by treating

patients with sudden deafness, osteomyelitis, refractory ulcer, gas gangrene and thermal burns. In 1981, the new facility in our private hospital was established in Nakatsu City, and a monoplace chamber was introduced. In 1984, a monoplace chamber was replaced with a multiplace chamber made by Nakamura Iron Works of Tokyo. This report pertains to treatment of 742 patients and 30,599 patient-treatments with HBO over a period of 8 years, 1981-1988.

II. METHOD OF HBO ADMINISTRATION

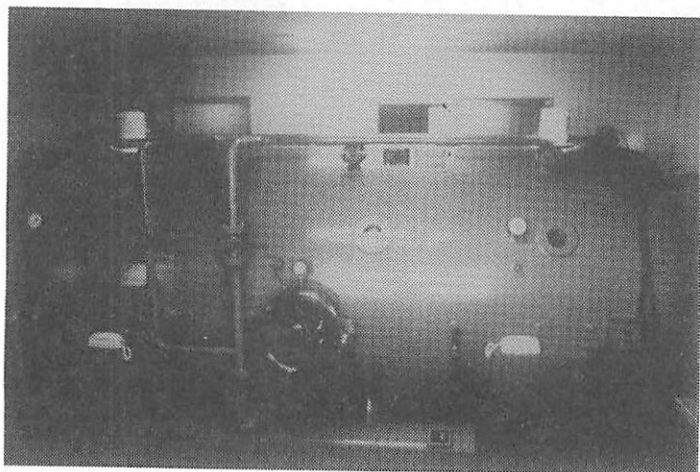


Fig. 1. A multiplace chamber.

A. The Chamber

The new multiplace chamber (1984) measures 2.5 m in diameter and 5 m in length, and is partitioned into two compartments (Fig. 1). The treatment compartment seats 6 patients comfortably, each equipped with an oxygen breathing mask. The entry compartment accommodates one attending physician and one nurse simultaneously and can be locked in and out, independent of the treatment section. Other equipment available are: video camera and recorder, wired and wireless telephone, transcutaneous oxygen meter, and ECG. Environmental control including pressure profiles, has been automated via computer control since 1986. Several kinds of treatment tables were used, such as Table A, B, and C. Maximum pressure in these Tables is 2.8 ATA, 2.5 ATA, and 2.0 ATA, for Table A, B, and C, respectively.

B. Patients

We treated 742 patients in the hyperbaric chamber in an eight year period between 1981 and 1988, and have accumulated 30,599 patient-treatments. Of the 742 patients, 344 were emergency cases, and 398 were non-emergency cases (Table 1). One hundred and twenty six dropped out and 616 were followed for 6 months or longer after HBO therapy.

TABLE 1.
Cases and Year

| Year | Cases | | |
|-------|-----------|---------------|-------|
| | Emergency | Non emergency | Total |
| 1981 | 0 | 6 | 6 |
| 1982 | 1 | 15 | 16 |
| 1983 | 0 | 36 | 36 |
| 1984 | 9 | 49 | 58 |
| 1985 | 86 | 103 | 189 |
| 1986 | 108 | 50 | 158 |
| 1987 | 96 | 101 | 197 |
| 1988 | 44 | 38 | 82 |
| Total | 344 | 398 | 742 |

For the cumulative 30,599 treatments, 872 were emergency and 29,727 were non-emergency (Table 2).

TABLE 2.
Number of HBO Treatments Between 1981 and 1988

| Year | No. of HBO Treatments | | |
|-------|-----------------------|---------------|--------|
| | Emergency | Non emergency | Total |
| 1981 | 0 | 114 | 114 |
| 1982 | 1 | 582 | 583 |
| 1983 | 0 | 1,379 | 1,379 |
| 1984 | 48 | 1,912 | 1,960 |
| 1985 | 312 | 7,665 | 7,977 |
| 1986 | 292 | 7,339 | 7,631 |
| 1987 | 161 | 6,958 | 7,119 |
| 1988 | 58 | 3,778 | 3,836 |
| Total | 872 | 29,727 | 30,599 |

The age distribution of patients between 7 and 81 years old, with an average age of 56.2 years old, is shown in Table 3.

TABLE 3.
Age Distribution

| Age | Male | Female | Total |
|-------|------|--------|-------|
| 0- 9 | 1 | 0 | 1 |
| 10-19 | 16 | 4 | 20 |
| 20-29 | 50 | 8 | 58 |
| 30-39 | 69 | 13 | 82 |
| 40-49 | 62 | 20 | 82 |
| 50-59 | 84 | 37 | 121 |
| 60-69 | 100 | 57 | 157 |
| 70-79 | 118 | 70 | 188 |
| 80- | 17 | 16 | 33 |
| Total | 517 | 225 | 742 |

The numbers of cases of treated disorders and percent of total cases were as follows: Acute cerebral thrombosis 210 (34.1%), osteomyelitis 107 (17.4%), decompression sickness 77 (12.5%), arteriosclerosis obliterans 56 (9.1%), crush injury 26 (4.2%), sudden deafness 24 (3.9%), refractory ulcer 19 (3.1%), acute arterial thrombosis 14 (2.3%), thromboangiitis obliterans 14 (2.3%), spinal nerve disorders 12 (1.9%), old cerebral circulation disorders 10 (1.6%), carbon monoxide poisoning 7 (1.1%), compromised skin grafts and flaps 5 (0.8%), burns 3 (0.5%), and others (Table 4).

Average number of HBO treatments for various disorders were as follows: 185.8 in thromboangiitis obliterans, 107.9 in arteriosclerosis obliterans, 81.4 in osteomyelitis, 63.4 in acute arterial thrombosis, 56.1 in spinal nerve disorders, 53.4 in refractory ulcer, 51.3 in old cerebral circulation disorders, 41.6 in vein thrombosis, 32.0 in burns, and 30.2 in sudden deafness. Raynaud's disease and SMON were treated 30 times each on the average (Table 5).

TABLE 4.
Case Distribution

| Disease | Case | (%) |
|---------------------------------------|------------|----------------|
| acute cerebral thrombosis | 210 | (34.1) |
| osteomyelitis | 107 | (17.4) |
| decompression sickness | 77 | (12.5) |
| arteriosclerosis obliterans | 56 | (9.1) |
| crush injury | 26 | (4.2) |
| sudden deafness | 24 | (3.9) |
| refractory ulcer | 19 | (3.1) |
| acute arterial thrombosis | 14 | (2.3) |
| thromboangiitis obliterans | 14 | (2.3) |
| spinal nerve disorders | 12 | (1.9) |
| old cerebral circulation disorders | 10 | (1.6) |
| vein thrombosis | 9 | (1.5) |
| gas gangrene, necrotizing fasciitis | 8 | (1.3) |
| carbon monoxide poisoning | 7 | (1.1) |
| compromised skin grafts and flaps | 5 | (0.8) |
| burns | 3 | (0.5) |
| head injury | 1 | (0.2) |
| cerebral edema | 1 | (0.2) |
| Raynaud's disease | 1 | (0.2) |
| radiation necrosis | 1 | (0.2) |
| decubitus | 1 | (0.2) |
| subacute myel-optic neuropathy (SMON) | 1 | (0.2) |
| other circulation disorders | 2 | (0.3) |
| others | 7 | (1.1) |
| Total | 616 | (100.0) |

TABLE 5.
Average Number of HBO Treatments

| Disease | No. of HBO Treatments |
|---------------------------------------|-----------------------|
| Thromboangiitis obliterans | 185.8 |
| Arteriosclerosis obliterans | 107.9 |
| Osteomyelitis | 81.4 |
| Acute arterial thrombosis | 63.4 |
| Spinal nerve disorders | 56.1 |
| Refractory ulcer | 53.4 |
| Old cerebral circulation disorders | 51.3 |
| Vein thrombosis | 41.6 |
| Burns | 32.0 |
| Sudden deafness | 30.2 |
| Raynauds disease | 30.0 |
| Subacute myel-optic neuropathy (SMON) | 30.0 |

III. RESULTS

A. General

Table 6 shows a numerical analysis of our clinical results. Crush injury, acute arterial thrombosis, thromboangiitis obliterans, spinal nerve disorders, vein thrombosis, gas gangrene and necrotizing fasciitis, compromised skin grafts and flaps, and burns all showed excellent results (100% effectiveness). Good results were observed in the following: 98.7% in decompression sickness, 98.6% in acute cerebral thrombosis, 98.2% in arteriosclerosis, 94.7% in refractory ulcer, 94.4% in osteomyelitis, 85.7% in carbon monoxide poisoning, 80.0% in old cerebral circulation disorders, and 71.4% in sudden deafness. The following categories (sections B, C, and D) deserve special mention.

TABLE 6.
Effectiveness of HBO Treatments

| Disease | Good | Failure | % Effectiveness |
|-------------------------------------|------------|-----------|-----------------|
| Crush injury | 26 | 0 | 100.0 |
| Acute arterial thrombosis | 14 | 0 | 100.0 |
| Thromboangiitis obliterans | 14 | 0 | 100.0 |
| Spinal nerve disorders | 12 | 0 | 100.0 |
| Vein thrombosis | 9 | 0 | 100.0 |
| Gas gangrene, necrotizing fasciitis | 8 | 0 | 100.0 |
| Compromised skin grafts and flaps | 5 | 0 | 100.0 |
| Burns | 3 | 0 | 100.0 |
| Decompression sickness | 76 | 1 | 98.7 |
| Acute cerebral thrombosis | 207 | 3 | 98.6 |
| Arteriosclerosis obliterans | 55 | 1 | 98.2 |
| Refractory ulcer | 18 | 1 | 94.7 |
| Osteomyelitis | 101 | 6 | 94.4 |
| Carbon monoxide poisoning | 6 | 1 | 85.7 |
| Old cerebral circulation disorders | 8 | 2 | 80.0 |
| Sudden deafness | 13 | 11 | 54.2 |
| Others | 5 | 2 | 71.4 |
| Total | 580 | 28 | 95.4 |

B. Osteomyelitis

Osteomyelitis is common, and despite modern advances in antibiotic therapy, may still cause disastrous disability. Systemic application of chemotherapy is not so effective if septic emboli, thrombi and necroses have formed in the infected area. As we have reported since 1969, closed irrigation-suction methods are very effective in the healing of osteomyelitis. We worked on improving the irrigation system and at last completed the new instrument, with a channel changer consisting of multiple valves (Kawashima and Tamura, 1983). From 1980 to 1982, a total of 232 patients were treated for bone and joint infection by closed irrigation. Of these cases, 226 (88.3%) were successful, 7 (2.7%) showed some improvement, and 3 (9.0%) were failures. For the purpose of more effective therapy (Hunt and Pai, 1972), HBO therapy was introduced in 1981. From 1981 to 1987, 109 cases of osteomyelitis were treated by HBO. Among them, 55 cases were treated by closed irrigation associated with HBO therapy, and 54 cases were treated by HBO only. Infection was localized in the sites noted in Table 7.

TABLE 7.
Sites of Infection of HBO Cases

| Sites | Patients |
|-----------------|----------|
| Mandible | 8 |
| Clavicle | 2 |
| Sternum | 2 |
| Humerus | 3 |
| Radius and ulna | 4 |
| Hand carpal | 1 |
| Finger | 1 |
| Pelvis | 4 |
| Femur | 26 |
| Tibia | 43 |
| Fibula | 2 |
| Foot carpal | 9 |
| Toe | 4 |
| Jaw | 1 |
| Knee joint | 1 |
| Ankle joint | 3 |
| Total | 114 |

A bacteriologic analysis was made on materials obtained from 73 cases. *Staphylococcus aureus* and *Pseudomonas aeruginosa* were the predominant organisms found (Table 8). Of the 109 patients treated, good results were noted in 95 (87.1%). Ten patients (9.2%) improved with either objective or subjective healing with later breakdown, or with decreased drainage and lessening of pain. Four patients (3.7%) showed recurrence (Table 9).

TABLE 8.
Microorganisms Found in 73 Patients with Osteomyelitis

| Microorganisms | Cases |
|-----------------------------------|-------|
| <i>Pseudomonas aeruginosa</i> | 14 |
| <i>Staphylococcus aureus</i> | 18 |
| <i>Staphylococcus epidermidis</i> | 5 |
| <i>Serratia marcescens</i> | 2 |
| <i>Streptococcus</i> | 2 |
| <i>Bacteroides</i> | 2 |
| <i>Klebsiella</i> | 1 |
| Tuberculosis | 5 |
| Negative | 24 |
| Uncertain | 37 |
| Total | 110 |

TABLE 9.
Effectiveness of HBO Treatment of Osteomyelitis

| Result | Case (%) |
|---------|-------------|
| Good | 95 (87.1) |
| Fair | 10 (9.2) |
| Failure | 4 (3.7) |
| Total | 109 (100.0) |

The transcutaneous oxygen pressures ($TcPo_2$) were measured during HBO treatment to estimate the effect of oxygenation. Figure 2 shows $TcPo_2$ at first HBO for 10 cases. The $TcPo_2$ at 2 ATA was significantly higher in normal tissue. Fig. 3 shows $TcPo_2$ after 10 HBO treatments. Here, the $TcPo_2$ at 2 ATA is more nearly equal when comparing between normal and infection sites.

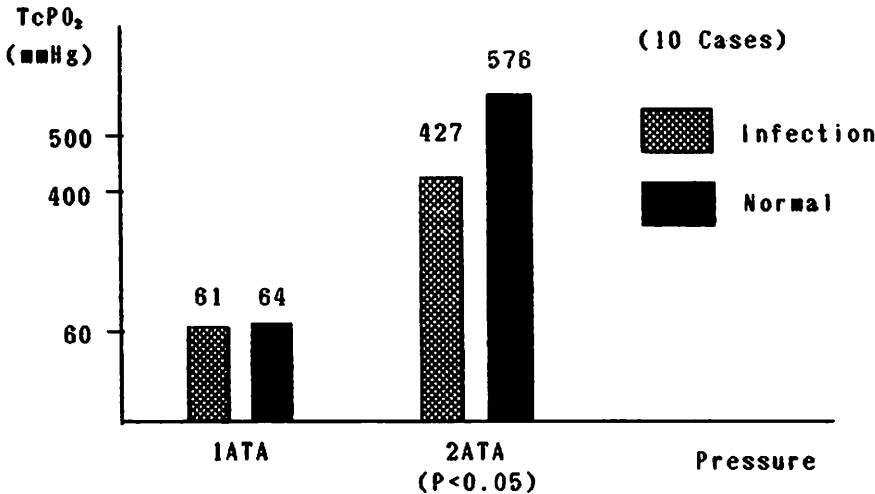


Fig. 2. Transcutaneous oxygen pressure ($TcPo_2$) at first HBO.

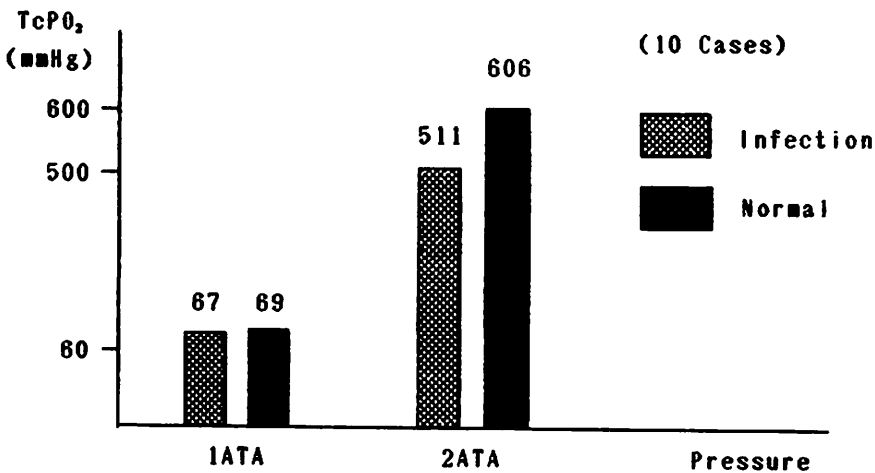


Fig. 3. Transcutaneous oxygen pressure ($TcPo_2$) after tenth HBO.

C. Chronic Obliterative Vascular Diseases

The use of HBO for healing indolent ulcer due to peripheral vascular disease has been the subject of much controversy. Most of the discussion has taken place in the absence of basic facts. Sakakibara (1983) reported that 161 patients obtained no relief despite surgical procedures. Among them, 106 cases were thromboangiitis obliterans (TAO), and 43 cases were arteriosclerosis obliterans (ASO). After HBO therapy, of the 106 cases of TAO, 74 (70%) reported disappearance of pain. Eighty-four cases (79%) had complete healing or substantial improvement of ulcers. As for the 43 cases of ASO, disappearance of pain was seen in 30 (70%), and 35 cases (81%) showed complete scar formation over the ulcers or a remarkable reduction in the size of the ulcers. In our patients with TAO, all showed good results, and 55 of 56 (98.2%) with ASO also showed substantial improvement.

Transcutaneous oxygen pressures ($TcPO_2$) were again measured during HBO to estimate the effect of oxygenation. Prostaglandin E_1 (PGE_1) was administered before HBO. Ascending rate of $TcPO_2$ was significantly higher in cases after the administration of PGE_1 . HBO combined with administration of PGE_1 seemed to be effective in increasing tissue oxygenation for the treatment of chronic obliterative diseases (Fig. 4).

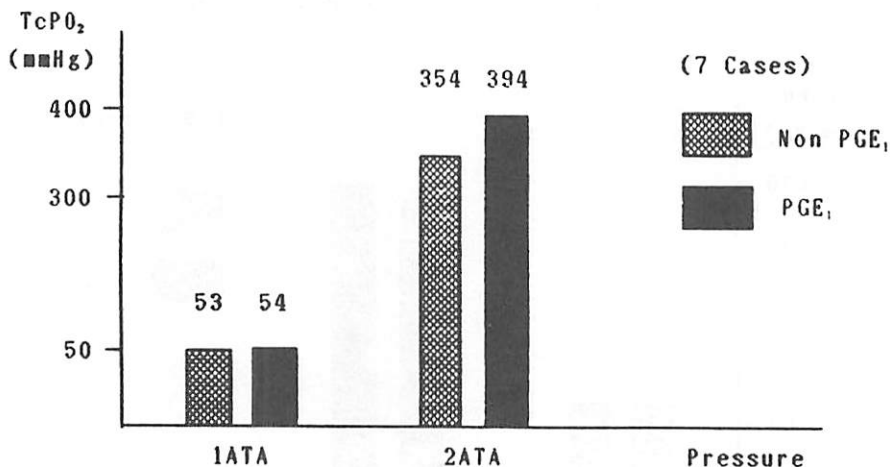


Fig. 4. Transcutaneous oxygen pressure ($TcPO_2$) in patients with circulation disorders. HBO treatment was done either with or without prior prostaglandin administration.

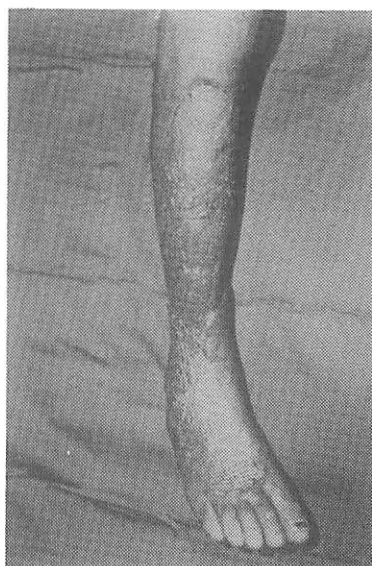
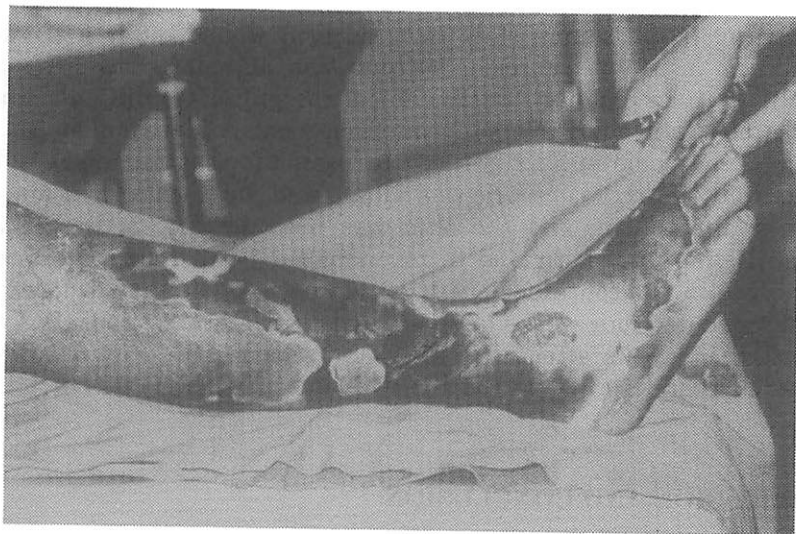


Fig. 5. (Top) A case of necrotizing fasciitis on admission. (Lower left) Debridement during operation. (Lower right) Wound healing after HBO.

D. Necrotizing Fasciitis

Necrotizing fasciitis may start in a surgical wound, after a trivial injury like an insect bite, abrasion, or contusion, and may show up spontaneously, even in children. Early clinical recognition is difficult, and there is often a fatal delay in appropriate treatment. A 57-year old farmer had a small contusion in his right foot. He complained of pain and swelling. Three days after the onset he was admitted to our hospital. The lower leg was swollen, firm, and the skin was necrotic (Fig. 5, top). The administration of antibiotic and intravenous fluids was begun at once. HBO was begun and continued every day. Two weeks later, debridement was performed and four weeks later, skin grafts were carried out (Fig. 5, lower left). Six weeks after, the skin was in good state (Fig. 5, lower right). As Bakker described in 1984, HBO is a useful therapy for necrotizing fasciitis.

IV. DISCUSSION AND CONCLUSION

A total of 742 cases were treated in the West Japan Medical Research Institute and Kawashima Orthopedic Hospital from 1981 to 1988. HBO was a very useful therapy in the orthopedic department. However, the significance of HBO is not yet well appreciated in Japan. Cost of HBO is cheap in Japan and the maintaining of a multiplace chamber is not difficult in private hospitals. We think more cases should be accumulated to demonstrate the clinical applicability of HBO.

REFERENCES

- Bakker, D.J. Pure and mixed aerobic and anaerobic soft tissue infections: The role of surgery, hyperbaric oxygen and antibiotics. In: J.H. Jacobson, G.B. Hart, M.B. Strauss, and E.P. Kindwall, eds. *Proceedings of the Eighth International Congress on Hyperbaric Medicine*, Memorial Medical Center of Long Beach, p. 112-119, 1984.
- Brummelkamp, W. Hyperbaric oxygen drenching of tissues in gynecological and obstetric infections. In: B.C. Ten Berge, ed. *Pregnancy, Chemistry and Management*. Springfield, IL: C.C. Thomas, p. 244-262, 1964.
- Davis, J.C. *Hyperbaric Oxygen Therapy: A Committee Report*. Bethesda, MD: Undersea Medical Society, Publ. No. 30, 1983.
- Hunt, T.K. and M.P. Pai. The effect of varying ambient oxygen tensions on wound metabolism and collagen synthesis. *Sur. Gynecol. Obstet.* 135:561-567, 1972.
- Kawashima, M. and H. Tamura. The treatment of pyrogenic bone and joint infections by closed irrigation-suction. *Clin. Orthop.* 148:240-244, 1980.
- Kawashima, M. and H. Tamura. A new instrument for closed irrigation-suction treatment. *J. Japn Orthop. Assoc.* 57:643-650, 1983.
- Kawashima, M. and H. Tamura. Topical therapy in orthopedic infection. *Orthopedics* 7:1592-1598, 1984.
- Mader, J.T. Phagocytic killing and hyperbaric oxygen: antibacterial mechanisms. *HBO Rev.* 2:37-49, 1981.
- Sakibahara, K. and H. Takahashi. Clinical experience of hyperbaric oxygen therapy for chronic peripheral vascular disorders. In: K. Shiraki and S. Matsuoka, eds. *Hyperbaric Medicine and Underwater Physiology*, Kitakyushu, Japan: Univ. of Occup. and Environ. Health, p. 337-344, 1983.
- Saito, Hyperbaric oxygen therapy for apoplexia (in Japanese), *Nihon Iji Shimpō*, 1808:3-36, 1958.
- Wada, J., T. Ikeda, K. Kamada, et al. Oxygen hyperbaric treatment for severe CO poisoning and severe burns in coal mines (Hokutan-Yubari) gas explosion. *Igaku Japn.* 54:68-69, 1965.

1. The first part of the document is a letter from the President of the United States to the Congress, dated January 3, 1862. It is a long and detailed letter, covering many topics, including the state of the Union, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the President's views and policies at that time.

2. The second part of the document is a report from the Secretary of the Treasury, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Treasury, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

3. The third part of the document is a report from the Secretary of the Interior, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Interior, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

4. The fourth part of the document is a report from the Secretary of the War, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the War, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

5. The fifth part of the document is a report from the Secretary of the Navy, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Navy, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

6. The sixth part of the document is a report from the Secretary of the Army, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Army, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

7. The seventh part of the document is a report from the Secretary of the Marine Corps, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Marine Corps, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

8. The eighth part of the document is a report from the Secretary of the Coast and Geodetic Survey, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Coast and Geodetic Survey, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

9. The ninth part of the document is a report from the Secretary of the Smithsonian Institution, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Smithsonian Institution, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

10. The tenth part of the document is a report from the Secretary of the Department of Agriculture, dated January 3, 1862. It is a long and detailed report, covering many topics, including the state of the Department of Agriculture, the progress of the war, and the administration of the government. It is a very important document, as it provides a clear and concise summary of the Secretary's views and policies at that time.

8

Medical Advances in Compressed Air Construction Work

Eric P. Kindwall

| | |
|--|-----|
| I. Introduction | 131 |
| II. Decompression Procedures | 132 |
| III. Comparison of Tables | 137 |
| IV. Clinical Evaluation | 144 |
| V. Summary | 147 |
| Acknowledgements | 147 |
| References | 147 |

I. INTRODUCTION

Caisson and compressed air tunnel exposures are different from those seen in diving in several important ways. Caisson work is invariably carried out at very modest pressures never exceeding the equivalent of 34 meters (112 feet) of sea water or 50 pounds per square inch. Bottom times or shift lengths, however, almost always range between 4 and 8 hr on a daily basis with 6 to 8 hr exposures being the most common. Furthermore every caisson exposure completely fills the envelope covered by the decompression schedule in use. A new schedule is provided for every additional 1 to 1.5 meters equivalent seawater depth. This is done for economic reasons as a pound or two difference would mean a new schedule and contractors with large gangs of men need to minimize decompression time. Thus, approximately 4 out of 5 caisson exposures will test the table to its limit whereas only about 3 in 10 water dives push the table to a similar extent. Diving tables are invariably set up in 3 meter increments.

Caisson workers also work in a dry environment and thus must support the weight of their own bodies throughout the exposure as well as the

tools and the burdens which they carry. The atmosphere in the tunnel frequently has more contaminants than are present in the divers air supply. Tunnelers are rarely chilled during the exposure but may be exposed to excessive heat during concrete pours. Chilling takes place during decompression when they are seated in a dry chamber. Balldin has demonstrated that there is 30% less nitrogen elimination in those decompressing in the dry as opposed to those decompressing while immersed (Balldin and Lundgren, 1969). Finally, caisson exposures are much more numerous than diving exposures. At the present time some 236,000 man-decompressions are being analyzed which were carried out using the Blackpool caisson schedules (Evans, 1987). Despite the great amount of material available for analysis, there has been relatively little study or scholarly work invested in caisson tables as opposed to those for deep sea divers. Physiologists used to dealing with the diving community often assume that caisson decompression sickness data can be treated in the same manner as diving statistics. This is not true as the decompression sickness figures from caisson work are never correct as reported. Official rates deal only with treated cases of decompression sickness. This is because the men fear for their jobs if they complain of symptoms. Because caisson tables have been notoriously bad for so many years, those engaged in this kind of work accept symptoms of decompression sickness as a fact of life (Nashimoto, Personal Communication, 1987). They rarely report for treatment unless symptoms are unbearable or incapacitating. Too frequent recompression treatment would mean loss of a job. Using an anonymous system of reporting on a tunnel project in Milwaukee in 1971-72, we found that up to 26% (Kindwall, 1975) of a shift might be bent on any given day and that we had decompression sickness present on the job on 42.5% of the working days. This was despite the fact that our "official" decompression sickness incidence was 1.44%.

II. DECOMPRESSION PROCEDURES

Traditional caisson decompression was accomplished by simply opening the valve to the manlock at the end of each shift and bleeding off pressure in a continuous manner. Stage decompression was unknown. There was a 25% mortality among the workers building the Hudson tubes which dropped to 4% with the introduction of a recompression treatment chamber. Knowing that shorter exposures seemed to decrease the incidence of decompression sickness, the concept of the split shift arose. This meant that instead of one continuous long working period during the day, the workmen would work a relatively short shift in the morning, followed by a rest period on the surface which in turn would be followed by a

second shift in the afternoon. It was thought that by working two shorter shifts, the decompression sickness rate would be lower. It was not understood that the time period on the surface between the two shifts was pitifully inadequate to allow release of meaningful amounts of nitrogen. The second failing point of the split shift was that it exposed the worker to the trauma of two decompressions per day instead of just one.

In addition to devising the first set of decompression tables in 1908, Haldane also tested different decompression schemes. He found that continuous decompression, such as caisson workers used, is inferior to stage decompression causing serious symptoms or death 25 times more often than stage decompression (Boycott *et al.*, 1908). Nevertheless, in 1922 the State of New York produced a decompression code embodying not only the split shift but continuous or uniform decompression. Unfortunately the split-shift is still in use in Japan. Fig. 1 shows how the Japanese caisson schedule compares to a U.S. Navy repetitive dive for the same exposures.

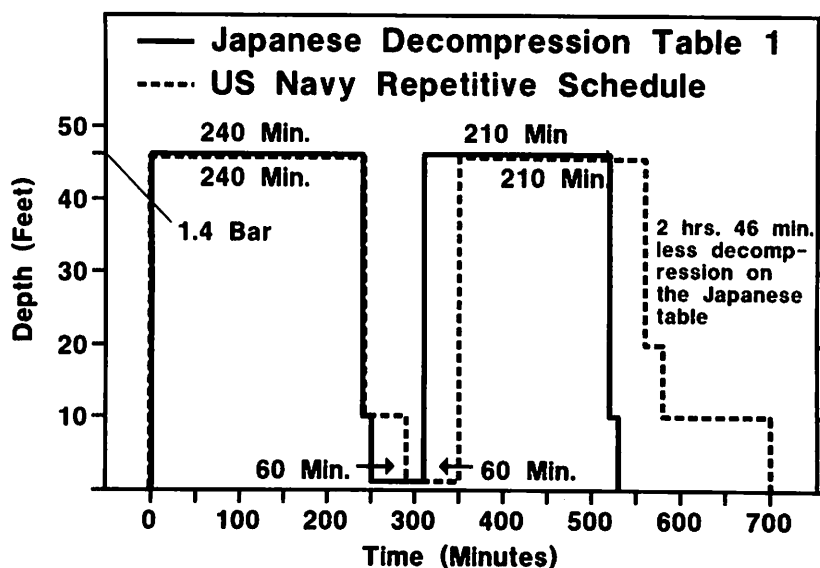


Fig. 1. Japanese caisson schedule compared to U.S. Navy dive schedule.

Aseptic necrosis was first recognized in tunnel workers in 1912 (Bornstein and Plate, 1911-12). Bone disease appears to be caused by improper decompression. Very costly human experimentation has shown that in the production of bone necrosis, decompression time is the only independent variable. Short tables produce necrosis — long tables produce less or none.

The 1922 New York table remained the gold standard of decompression in the United States until the 1960's. Beginning in 1958, the British Medical Research Council Panel on Decompression Sickness began to investigate the incidence of aseptic necrosis in tunnel workers and found a 19% incidence of bone disease in the workers building the Clyde tunnels (Jones and Behnke, 1978). The Blackpool tables were adopted in 1966 in an effort to avoid aseptic necrosis. Nevertheless, 5 of 59 workers on the Dungeness SB power station contract developed bone disease on the Blackpool tables (Trowbridge, 1977). Yau has reported that 83% of the men working on the Hong Kong Subway project reported decompression symptoms in association with the Blackpool schedules (Lam and Yau, 1988; Yau Personal Communication, 1987). The "official" bends rate however was low.

In 1963, Duffner devised the Washington State tables which later were adopted as the OSHA tables in 1971. These tables abolished split-shift, but retained continuous or uniform decompression in stages after an initial pressure drop. This was in deference to the contractors and workers who did not wish to break with tradition. When asked why he did this, Duffner said, "There are certain battles that you just can't win" (Duffner, Personal Communication, 1987). The Washington State tables used only 3 tissue half times, (the 30, 60, and 120 minute) but were basically Haldanian (Sealey, 1969). Duffner's new tables produced no incidence of aseptic necrosis when used in Seattle and in the construction of the San Francisco subway, but on this latter job only 135 feet of tunnel was dug at greater than 16 pounds. Ten years later, Sealey (1975) surveyed 83 workers who had worked on the Seattle project and found only 4 men with tibial shaft lesions, one bilateral. There was no juxta-articular involvement, which requires a greater decompression insult. These tables were adopted in Milwaukee in 1970 by emergency order because of a 35% incidence of aseptic necrosis which had been experienced using a modification of the 1922 New York code which had been part of Wisconsin law (Kindwall *et al.*, 1982, Nellen and Kindwall, 1972). Nevertheless, at pressures greater than 36 pounds, the Washington State tables (which by then had become the federally enforced OSHA tables) produced a 33% incidence of aseptic necrosis (Kindwall *et al.*, 1982). Fig. 2 compares the OSHA and Blackpool tables for the 2, 4, 6 and 8 hour exposures. Note that

on the Blackpool tables, the decompression time at any given pressure is the same for any time period over 4 hr up to 8 hr. The OSHA schedule is more conservative for most long exposures between 14 and 44 psig. The lines (dashed or solid) become thicker with increasing exposure.

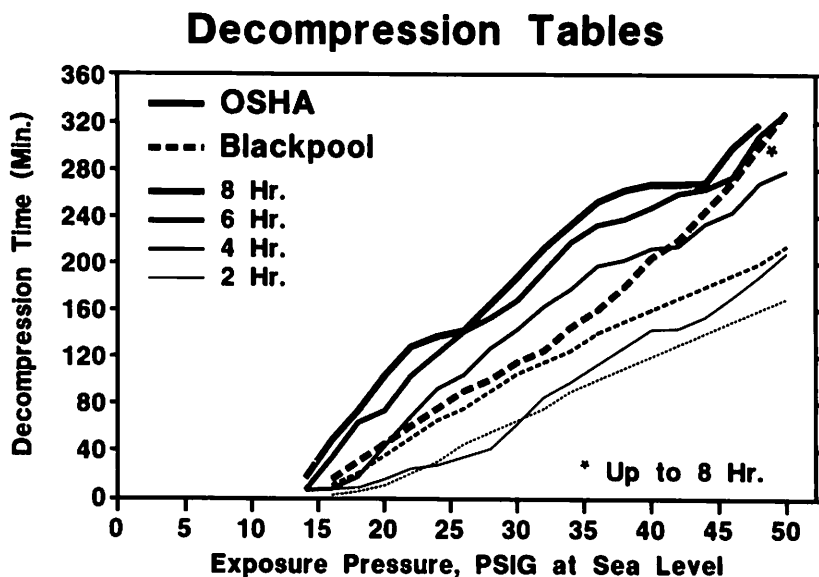


Fig. 2. OSHA and Blackpool tables compared.

In retrospect, there was a high bends rate using the OSHA table both in Seattle and California which was confirmed by the Milwaukee experience. Because of this problem, we obtained a grant from the National Institute of Occupational Safety and Health to devise new decompression tables for tunnel workers. Using Peter Edel's computer memory bank which contained data on 15 years of successful and unsuccessful dives in the Gulf of Mexico and elsewhere, the computer was directed to construct a line between safe and unsafe decompressions. The final result was that the Milwaukee tables which were prohibitively long when air was breathed for decompression (Kindwall *et al.*, 1983). Thus, truly "safe" tables seemed incompatible with the use of compressed air in underground construction or caisson work. However, when an oxygen decompression variant of the table was developed and tested, the decompression times compared favorably with the present OSHA schedules.

Decompression Tables

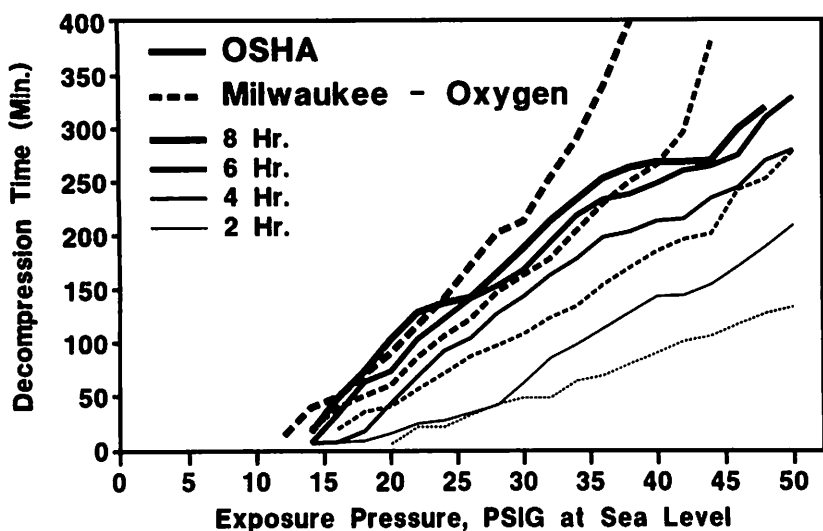


Fig. 3. Milwaukee and OSHA tables compared.

The reason Edel's decompressions were so long was that he included commercial diving data, altitude work and data from his own experiments. Previously, most investigators had relied almost exclusively on naval data for extrapolating to tunnel exposures. However, navies have little or no experience in the extremely long exposures used by tunnel workers, the stressfulness of which is compounded by an unbroken string of daily decompressions which may go on for months or years. Decompressions from 7 and 8 hr exposures are getting close to limits we would consider for saturation. The new oxygen tables were tested in increments of 0.14 kg/cm² (2 psig) from 14 to 46 psig (.95 bar to 3.13 bar). Spot checking of these tables using the longest shifts which could be fitted into an 8 hr working day failed to produce any decompression sickness or aseptic necrosis in our test subjects. These tests, of course, could not predict an incidence of decompression sickness but could only rule out a catastrophic error.

Tunnelling has taught us that daily decompression from extreme exposures even at modest depth require inordinate lengths of time if air is breathed during decompression. However, I am beginning to doubt that even long air decompression can accomplish this reliably. In Milwaukee

we have had bends symptoms following 7 hr exposure to 15.5 psig followed by 54 min decompression. Behnke has remarked that the same incidence of decompression sickness may be seen after the same exposure with widely divergent air decompression times (Behnke, 1969). For all the above reasons I believe that air decompression has now shown its limits.

III. COMPARISON OF TABLES

Fig. 4 shows the OSHA table compared to the U.S. Navy decompression table. Note that at pressures from 15 to 25 psig, the OSHA table is equal to or slightly more conservative than the Navy table but at higher pressures for longer times, the Navy table is much more conservative. These 6 and 8 hr exposures on the Navy table, however, were taken from the exceptional exposure air tables which have shown a bends incidence between 17-33% on test. Therefore, one would predict that the OSHA tables would be inadequate for the longer time periods. The irregularities seen in the OSHA table curves are due to the fact that these tables were put in final form by placing a ruler across a nomogram. A very slight movement of the ruler can produce the aberrancies noted.

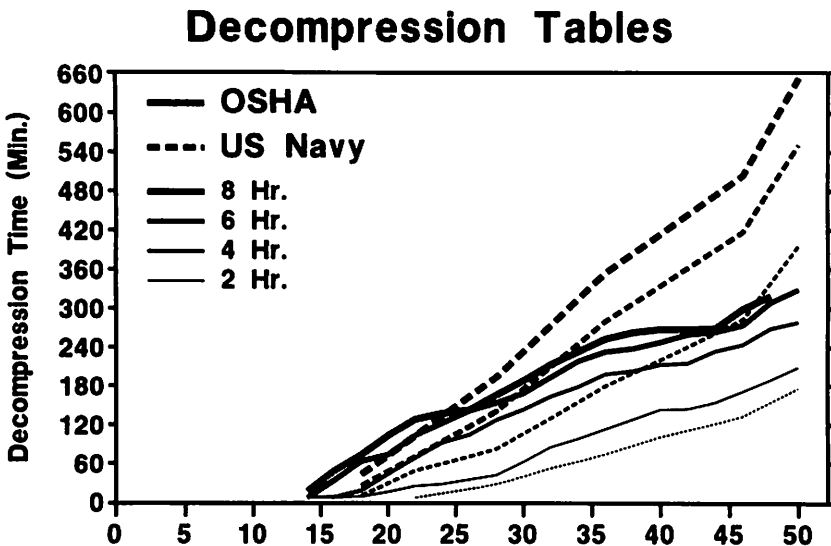


Fig. 4. OSHA table and U.S. Navy schedules compared.

Fig. 5 compares the French air table to the OSHA table. Note that it is much more limited and that work at above approximately one bar is not

permitted for more than a duration of 4 hr. The French tables appear to be inadequate by OSHA or Navy standards but obviate many problems by not permitting long duration work at high pressure.

Decompression Tables

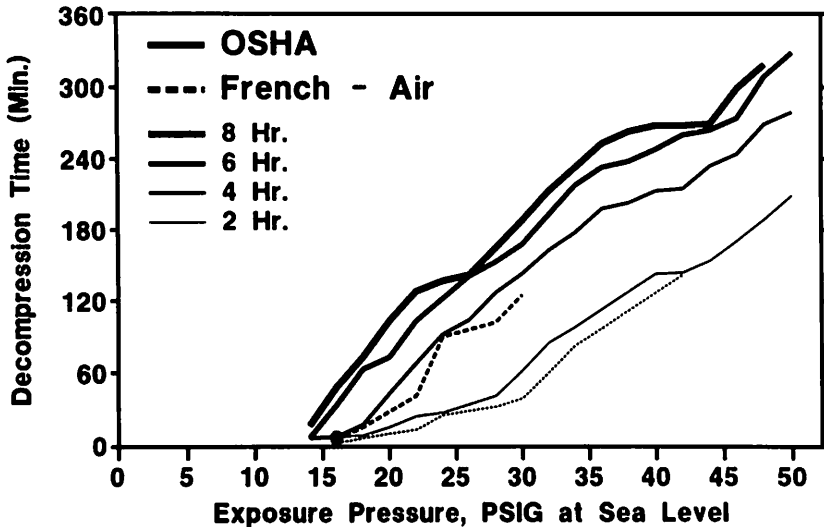


Fig. 5. French air table and OSHA table compared.

Fig. 6 compares the German air tables to OSHA schedules. Again, the OSHA schedules appear to be more conservative despite their known predilection to produce aseptic necrosis at high pressure.

Fig. 7 compares the exposure limits of the various tables. The French are most conservative, cutting back their time sharply after 1 bar is reached. The Germans are next in line. Blackpool tables limit all exposures to a maximum of 8 hr at 3.4 bar but note that the diagonal lines depict the area where decompression is the same at a given pressure for any work period between 4 and 8 hr. The U.S. Navy exceptional exposure air tables go to 12 hr but have been arbitrarily cut off for exposures deeper than would be of interest in tunnel work. They, like the Blackpool tables, increase decompression in 4 hr increments of exposure. The OSHA tables are shown to extend infinitely as a schedule is given for "greater than 8 hr" for all of the working pressures up to 46 psig (3.13 bar).

Figs. 8, 9 and 10 show a comparison of the decompression times for 4, 6 and 8 hr work at 1.5, 2.0 and 3.0 bar. The "X" above the vertical bar

Decompression Tables

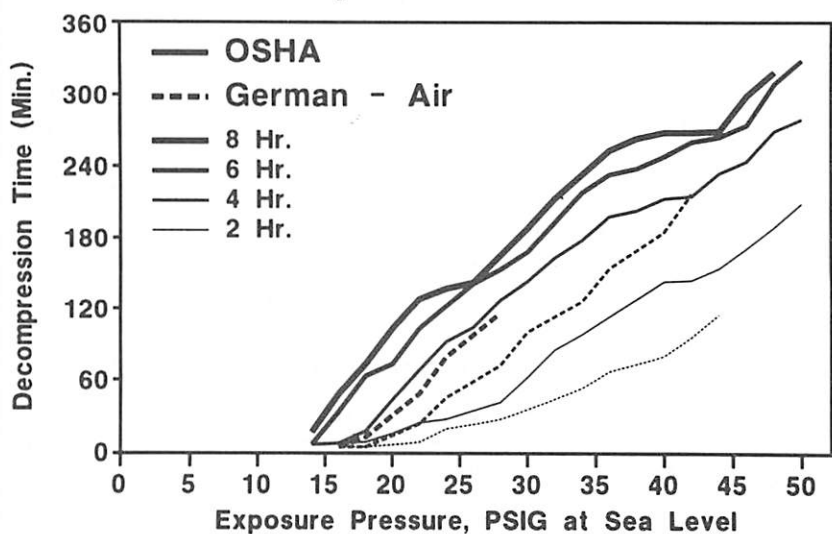


Fig. 6. German air table and OSHA table compared.

Comparison of Exposure Limits

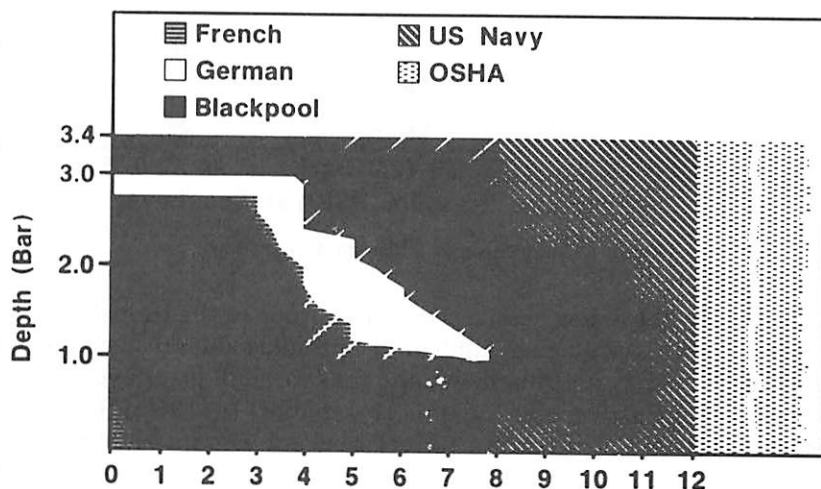


Fig. 7. Exposure limits of various tables compared.

indicates an oxygen variant of the table. At the pressures noted, the German air tables are the least conservative with the Blackpool and OSHA tables not far behind. The OSHA schedule is slightly better or equal to the Blackpool table at most exposure pressures. Note, however, the length of the new Milwaukee air table which is orders of magnitude greater than any of the existing air tables. This is what renders it unusable commercially. However, the oxygen variant of the new Milwaukee table brings it into the range of commercial utility which compares favorably with any of the existing schedules for the usual working times. The Canadian DCIEM tunnel tables are better than any of the tables on the graphs which are currently in use, but they are still less conservative than the new Milwaukee tables, they sharply limit exposure and there is no oxygen variant.

Comparison of Decompression Times for 4 Hours of Compressed Air Work

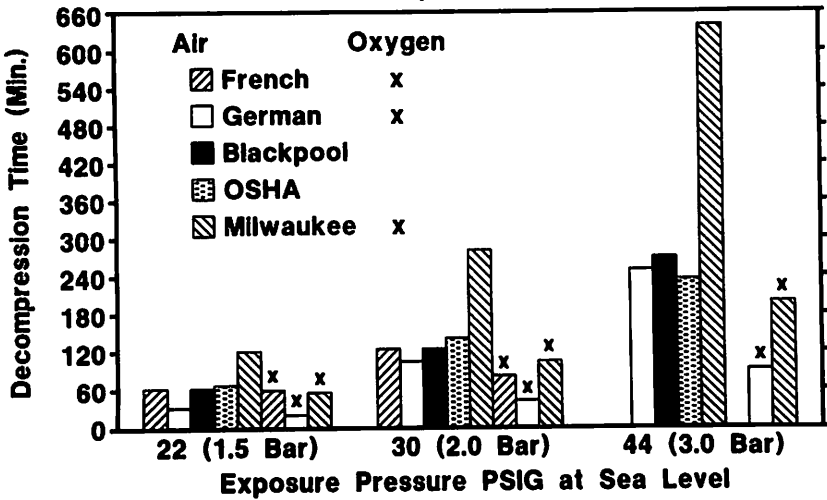


Fig. 8. Comparison of tables for 4 hours work.

Traditionally, there has been a cavalier attitude in the tunnel industry and also among physiologists that mild musculo-skeletal bends are an annoyance, but really nothing more, and that we shall just have to put up with them. This attitude is no longer tenable. In 1965, Rozsahegyi reported that 42% of Hungarian tunnel workers who had never experienced neurologic decompression sickness had abnormal electroencephalograms (Rozsahegyi, 1967). Gorman *et al.* (1986) have demonstrated abnormal EEG's, abnormal psychometric testing and abnormal CAT scans

**Comparison of Decompression Times for
6 Hours of Compressed Air Work**

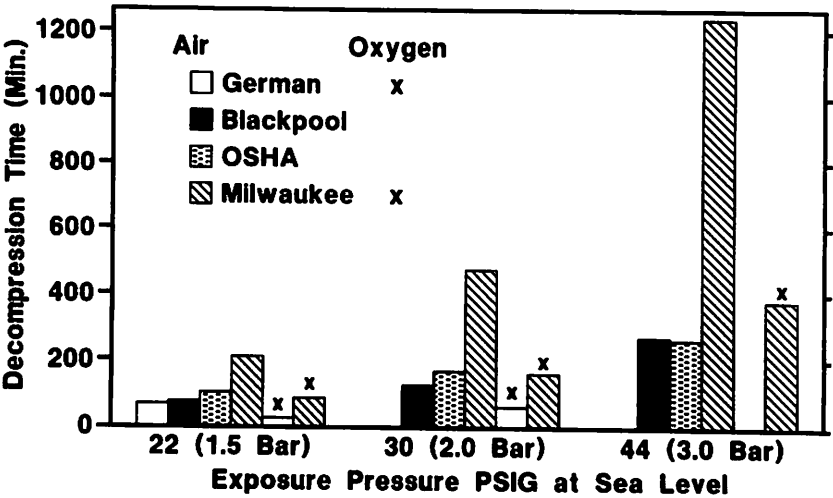


Fig. 9. Comparison of tables for 6 hours work.

**Comparison of Decompression Times for
8 Hours of Compressed Air Work**

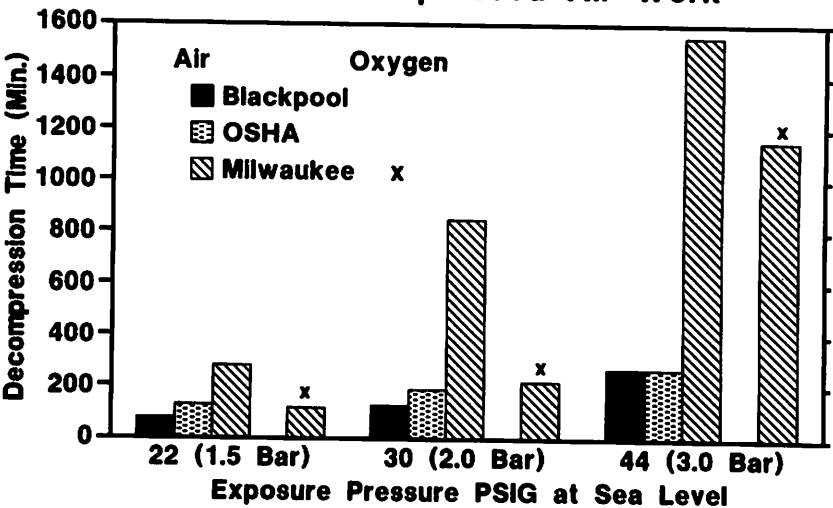


Fig. 10. Comparison of tables for 8 hours work.

showing brain atrophy in divers who have been treated for pain only bends. There must be no letup in our effort to improve caisson schedules until the bends incidence is *truly* minimized.

Tunnel tables are necessarily different than dive tables in what they provide. Ideal tables should be computed for all pressures above 0.90 Kg/cm² in 0.1 Kg/cm² or 1 pound increments. Exposure times should not only be listed in regular half-hourly increments, but should also be listed as the maximum working time at any given pressure which, when combined with the decompression time, produces an 8 hr working day. This will make them "user friendly". The Germans are the only ones who have attempted this thus far. I favor this approach, even for countries where a greater than 8 hr workday is acceptable, as I feel that at least a 16 hr surface interval should be afforded the workers before the next shift. Additionally, exposures of exactly 4 hr and 6 hr should be listed, as these divide evenly into a 24 hr day and make either 4 or 6 work shifts possible per day. On some jobs, the working face cannot be left unguarded between shifts, so one shift must immediately relieve the other. On this type of job, the only other alternative is to erect breasting boards between shifts, a costly and time consuming procedure. A listing of "no-decompression" exposures for each pressure should also be supplied for brief visits of one-time visitors such as company officials and the physician. Finally, repetitive exposure schedules must be provided to accommodate non-shift workers such as inspectors, electricians, engineers and others who need to be in the tunnel for short periods more than once a day. Failure to do this forces the people involved to "add up their times" and take all their decompression at the end of the day. This is non-physiologic and dangerous, and may have produced some of the aseptic necrosis seen in Hong Kong (Personal Communications, Walder, 1987; Yau, 1987). These multiple schedules should not be too difficult to produce with the aid of a computer.

It is clear from past experience that physiologists must no longer permit themselves to compromise on the length of decompression times, the decompression profile or the gas breathed during decompression out of deference to tradition or contractors' and unions' wishes. These compromises have always produced tables ranging from bad to catastrophic. Economics *do* play the principal role in the construction business, but those who derive tables must supply useable schedules which will not cause injury. Our job is not to compromise based on tradition, but to protect the worker through new technology.

Of utmost importance is that *any* new table enacted into an officially enforced regulation be labeled as an *interim schedule*. A method for quick modification of the new table as experience dictates must also be included

in the regulation. This will avoid the present dismal situation where a given table is known to be bad but it must be grudgingly enforced because there is no mechanism to abolish it. In Russia all tables are brought up for automatic change every five years should any need have arisen.

Oxygen decompression which was first used in tunnelling on a regular basis by the Germans in 1972 and then adopted by the French in 1974 seems to be the only viable daily decompression technique which is acceptable for tunnelling. Based on comparisons in our computer data bank, the French and German tables seem to be too short. Indeed the Germans report that they plan on revising them although they have reported a "remarkable drop in decompression sickness despite longer shifts" (Personal Communication, Altner, 1986). The French have reported no aseptic necrosis since the adoption of their tables in 1974, but as yet these new oxygen tables remain to be tested on a large project (Personal Communication, LePechon, 1987). Brazil used oxygen decompression successfully in 1975 in the construction of the Sao Paulo subway with a nearly 80% drop in decompression sickness (Personal Communication, Ribeiro, 1985).

Because of a tunnel fire which occurred during early experimentation with oxygen decompression in Japan in the early 1960's there has been much resistance on the part of the British and American regulating bodies to adopt oxygen decompression. This is despite the fact that some 14,000 experimental oxygen decompressions were carried out without mishap in 1938 and 1939 in the City of New York during the construction of the Queens Midtown tunnel (Jones *et al.*, 1940). The oxygen delivery system was poor on that project, providing the workers with too much breathing resistance. Nevertheless, there were no serious cases of decompression sickness in those workers who breathed oxygen. The main advantages of oxygen are vast savings in time, which of course makes economic sense for the contractors.

There are other advantages of oxygen decompression which are frequently not considered. End, in 1939, described a marked reduction in blood sludging in bent animals concomitant with simply raising the arterial PO_2 (End, 1939). Recently Mathieu *et al.* (1984) have reported that red blood cell filterability is doubled after 15 hyperbaric oxygen treatments. The need to improve the filterability of the blood during and after decompression is underscored by the work of Pimlott, Ormsby and Cross who found that white cells show an 81% decrease in deformability and filterability after exposure to air at 1.5 bar for 4 hr (Pimlott *et al.*, 1987). The toxicity of oxygen is well understood and its early signs, should they appear in tunnel workers, are easily reversible. This is not true for brain damage and bone disease.

The regulatory bodies in Great Britain are nevertheless adamant in refusing to accept any change in the present tunnelling decompression schedules until it can be assured that "no new risk is added." The United States is slightly more advanced in that the Office of Variance Determination of the Occupational Safety and Health Administration has now indicated that "oxygen decompression may be feasible" and they will permit contractors to apply to use this method under an "interim order" (Personal Communication, Concannon, 1987). Based on the known record of the present OSHA tables to produce bends and bone necrosis, we petitioned the Secretary of Labor to rescind these tables immediately as an imminent hazard, but we were told that it may be up to nine years from the time the first report of aseptic necrosis appeared in the open literature until these tables are rescinded. Meanwhile, U.S. contractors may be permitted to work under an interim order when the Office of Variance Determination is able to supply its "standard" for the workplace.

On August 2, 1988, Dr. Ralph Yodaiken, Director of the Office of Occupational Medicine of OSHA, circulated a memorandum to his staff regarding the present U.S. tunnel decompression tables. The memorandum indicated government acceptance of the inadequacy of the present tables stating, "(they). . . are flawed by modern standards" and that "The OSHA tables have failed any reasonable test of adequate performance over the past 16 years." He went on to say, "there is no dispute within the scientific community that the (Autodec III) oxygen tables would be a great improvement." He also stated that, "The Undersea and Hyperbaric Medical Society in Bethesda can be instrumental in developing training requirements. With these and other requirements cited by Dr. Kindwall, oxygen decompression can be very safe as proven by the German, French and Brazilian experience. In summary, oxygen decompression is long overdue in caisson work."

In line with this recommendation, I am developing a training package and intend to submit it to the Hyperbaric Chamber Safety Committee of the UHMS for review and approval before submission to OSHA.

Nevertheless, I am told by an OSHA staff member that even under the most *optimal* circumstances, it will be at least two years before the present tables are rescinded.

IV. CLINICAL EVALUATION

We recently have gathered data, however, which may conceivably speed this process. We used Magnetic Resonance Imaging (MRI) to look for brain damage in compressed air workers. At this time I would like to acknowledge the work of co-investigators Dr. David Czarnecki and Dr. George Fuerdi of the Department of Radiology, Randall Daut, Ph.D.,

psychologist, St. Luke's Medical Center and also Paul Grebe, a fourth year medical student without whose assistance this work could not have been completed. As of two weeks ago, we finished MRI studies of the brain in 28 tunnel workers, 20 of whom had worked in compressed air and 8 who had worked in tunnels but had never been exposed to compressed air. The MRI's were carried out using a new Phillips Gyroscan® Model S-15, a 1.5 Tesla machine. It was capable of 10 milli-Tesla gradients and had 5.5 level software.

With the exception of two tunnel inspectors, one in the compressed air group and one in the control group, all were from the Laborers Union. The mean age difference between the two groups was only 1.9 years, the compressed air group averaging 54.5 years (range 34-63) and the control group being 52.6 years (range 38-65). The mean education of both groups was 11 years. Four men in the compressed air group were illiterate and one was illiterate in the control group. In those individuals, the Shipley test of verbal IQ could not be utilized. In 12 of the 20 compressed air workers, excessive alcohol consumption was noted in the history. Of the 8 control subjects, 5 out of the 8 had a history of excessive alcohol consumption. Three of the 8 controls had a history of head trauma with unconsciousness versus 9 of the 20 in the compressed air group so those parameters were similar. To our knowledge, there were no insulin dependent diabetics in either group. We asked about current medications, but not specifically about insulin.

The Shipley verbal IQ scores were 30.6 in the compressed air group and 30.1 in the control group. Among literate subjects, the Shipley score is thought to correlate best with innate intelligence and is supposed to suffer least in dementia and later impairment from any cause. Our subject groups were incredibly well-matched with regard to innate intellectual ability. Subjects were also given neurophysical evaluation with tests of hand to eye coordination in both the left and right hand (pegboard), a digit symbol test, trailmaking of two kinds, going from point to point with a pencil and a letter cancellation test. Trailmaking and letter cancellation were scored both for time and number of errors. In all there were 10 separate scores of mental performance.

The MRI scans were analyzed for unidentified bright objects (UBO's), ventricular size and an estimate of cortical atrophy. Only the number of UBO's and ventricular size could be quantified precisely. The assessment of cortical atrophy remained the radiologist's impression. Two independent radiologists reviewed the MRI's blinded as to whether they were compressed air workers or controls. It was the radiologist's impression that cortical atrophy was greater in the compressed air group, but this remained only an impression. Four the the compressed air group had

enlarged ventricles versus none for the controls but this was not statistically significant. However, the compressed air group averaged 3.8 UBO's (range 0 to 17.5) versus 0.4 for the control group (range 0 to 1.5). This was significant with a p value of 0.02. Nine of the 20 compressed air workers had UBO's whereas three of the 8 controls had UBO's. Half point units on the UBO scores indicate an average between the two radiologists' independent recognition of UBO's.

Unidentified bright objects are associated firstly with lacunar infarcts secondary to vascular disease, second most commonly with demyelinating disease, third most commonly with gliosis and as a fourth category they sometimes appear without apparent lesions which can be confirmed at autopsy. None of our subjects was suffering from known demyelinating disease. The lesions in multiple sclerosis and other demyelinating diseases tend to be tightly periventricular. Most of our lesions fell into the general periventricular and central white mass areas.

Of the 10 psychometric tests given, the compressed air group scored better on only one (the time taken to complete Trails B) but they made twice as many errors. On one test there was no difference (neither group made any errors on the digit symbol test). However, the compressed air group scored slightly lower than the control group with regard to all of the remaining 8 scores. Although no single test showed a significant difference between the two groups, the difference when all the scores were totaled, showing the 8 out of 10 skewing, was significant at the 0.05 level. This might be expected in diffuse disease.

We tried to correlate an index of previous exposure to compressed air [months of work, times the maximum pressures and an arbitrary aseptic necrosis score (13 of our 20 compressed air workers showed some manifestation of bone disease)], but these could not be significantly related to the presence or absence of UBO's. There were trends, but they did not reach statistical significance, perhaps due to the crudeness of the exposure index and the small sample size.

We were limited by lack of funds in doing great numbers of MRI's and this is admittedly a small sample. However, I think preliminary results warrant further investigation.

On large jobs at high pressure, saturation exposures may be indicated. Here the workers would remain at pressure for a week or two at a time in a pressurized habitat which has some of the amenities of a small submarine. They then would go to work via personnel transfer capsule (PTC) or connecting lock to the heading. The advantage here is that daily 8 hr shifts are possible with no decompression required, and workers are exposed to decompression trauma only once every week or two weeks. Saturation for compressed air workers was originally suggested by

Behnke in 1969. It is only now that an economic demand for tunnel saturation is materializing.

V. SUMMARY

In summary, modern caisson and tunnel construction is a high technology industry. Rapid advances have been made in automated tunnelling machinery, but where personnel must be exposed to these pressures, government regulations have failed to keep pace with requirements. Modern advances in decompression physiology have made possible oxygen decompression which is not only more economical but vastly safer than traditional air decompression. The diving industry has led the way in this respect. However, even though these techniques are now available to the tunnelling industry, bureaucratic inertia must first be dealt with so that further damage to the brains and bodies of tunnel workers can be avoided.

ACKNOWLEDGEMENTS

The authors wish to express their sincere appreciation to the individuals and organizations listed below for financial and other support which made this research possible:

Phillips Medical Systems Incorporated

The Eastman Kodak Company

Tomaro Contractors of Milwaukee

Milwaukee Metropolitan Sewerage Commission

Joseph J. Adamkiewicz, M.D. and Thomas E. Knechtges, M.D.
Department of Radiology, St. Luke's Medical Center

Laborers International Union, Local 113 whose members willingly and unselfishly agreed to become subjects for this study.

REFERENCES

- Ballin, U.I. and C.E.G. Lundgren. The effect of immersion on inert gas elimination. *Acta Physiol. Scand.* (Suppl. 330): 83, 1969.
- Behnke, A.R., Jr. New approaches to medical aspects of work in compressed air, *J. Soc. Occup. Med.* 5:266, 1969.
- Bornstein, A. and E. Plate. Über chronische Gelenkveränderungen, entstanden durch Presslufterkrankung. *Fortschr. Geb. Röntgenstrahlen*, 18:197, 1911-12.

- Boycott, A.E., G.C.C. Damant, and J.S. Haldane. The prevention of compressed air illness. *J. of Hygiene* (Cambridge) 8:342-423, 1908.
- End. E. The physiologic effects of increased pressure. In: *Proceedings of the Sixth Pacific Science Congress* (Univ. of CA), 6:91-97, 1939.
- Evans, A. Plan Announced at Meeting of the Compressed Air Working Group; *British Medical Research Council*, Sept. 22, 1987.
- Gorman, D.F., C.W. Edmonds and R.G. Beran. The neurological sequelae of decompression sickness: A clinical report. In: A.A. Bove, A.J. Bachrach and L.J. Greenbaum, eds. *Underwater and Hyperbaric Physiology IX*. Bethesda, MD: Undersea and Hyperbaric Medical Society, p. 993-998, 1987.
- Jones, J.P. and A.R. Behnke, Jr. Prevention of dysbaric osteonecrosis in compressed air workers. *Clin. Orthopaed. Rel. Res.* 130:118-128, 1978.
- Jones, R.R., J.W. Crosson, F.E. Griffith, R.R. Sayers, H.H. Schrenk and E. Levy. Administration of pure oxygen to compressed air workers during decompression: Prevention of the occurrence of severe compressed air illness. *J. Ind. Hyg. and Tox.* 22:427-444, 1940.
- Kindwall, E.P. Medical aspects of commercial diving and compressed air work, In: C. Zenz, ed. *Occupational Medicine, Principles and Practical Applications*. Chicago, IL: Year Book Medical Publishers, p. 361-421, 1975.
- Kindwall, E.P., J.R. Nellen, and D.R. Spiegelhoff. Aseptic necrosis in compressed air tunnel workers using current OSHA decompression schedules, *J. Soc. Occup. Med.* 24:741-745, 1982.
- Kindwall, E.P., P.O. Edel, and H.E. Melton. *Safe Decompression Schedules for Caisson Workers, Final Report*. National Institute of Occupational Safety and Health Research Grant No. 5R 01 OH 00947-03, Washington, DC, 1983.
- Lam, T.H. and K.P. Yau. Manifestations and treatment of 793 cases of decompression sickness in a compressed air tunnelling project in Hong Kong, *Undersea Biomed. Res.* 15:377-388, 1988.
- Mathieu, D., J. Coget, L. Vinkier, F. Saulnier, A. Durocher, and F. Wattel. Erythrocyte filterability and hyperbaric oxygen therapy (French). *Medsubhyp*, 3:100-104, 1984.
- Nellen, J.R. and E.P. Kindwall. Aseptic necrosis of bone secondary to occupational exposure to compressed air: Roentgenologic findings in 59 cases, *Amer. J. Roent. Radium Ther. Nuc. Med.* 65:512-523, 1972.
- Pimlott, J., P.L. Ormsby, and M.R. Cross. The effect of white cells on blood filterability at pressure. *Proc. of the European Undersea Biomed. Soc. Ann. Sci. Mtg.* Palermo, 1987.
- Rozsahegyi, I. Neurological damage following decompression: In: R.I. McCallum, ed. *Decompression of Compressed Air Workers in Civil Engineering*. Oriel Press Ltd, p. 131, 1967.
- Sealey, J.L. Safe exit from the hyperbaric environment, *J. Soc. Occup. Med.* 11:273-275, 1969.
- Sealey, J.L. Aseptic bone necrosis survey in compressed air workers. *J. Soc. Occup. Med.* 17: 666-667, 1975.
- Trowbridge, W.P. Bone necrosis in British compressed air workers. In: *Aseptic Bone Necrosis, Proceedings of the Symposium of the European Undersea Biomedical Society*, Newcastle-Upon Tyne, 1977.

9

Diving Accident Management in Remote Areas: Current Concepts and Controversies

Carl Edmonds

| | |
|---|-----|
| I. Introduction | 149 |
| II. Arterial Gas Embolism (AGE) | 150 |
| III. Pulmonary Barotrauma — Other Than Air Embolism | 151 |
| IV. Decompression Sickness (DCS) | 152 |
| V. Drowning | 153 |
| VI. Hypothermia | 153 |
| VII. Inner Ear Barotrauma | 153 |
| VIII. Infections | 155 |
| IX. Marine Animal Injuries | 156 |
| A. Neuromuscular Venom | 156 |
| B. Fish Spine Injuries | 156 |
| C. Jellyfish | 156 |
| X. General | 157 |
| Appendix | 157 |
| A. Oxygen Administration Equipment | 157 |
| B. Underwater Oxygen Recompression | 158 |
| C. Positioning, Moving and Transport of the Patient | 159 |
| D. Aviation Medivac | 160 |

I. INTRODUCTION

A great deal of compressed air diving is carried out by recreational, scientific and commercial divers, in areas remote from major medical centers.

Because of the high incidence of morbidity, and even mortality, it is reasonable to expect that some attention would be given by the diving supervisors to appropriate first aid management and medivac facilities.

This presentation presumes the presence of knowledgeable divers and/or paramedics adequately equipped for such treatments, but with limited communication, consultation and medivac capability.

In selecting the disorders to address priority has been given to those likely to cause death or permanent disability. In each case the basic pathology is described.

II. ARTERIAL GAS EMBOLISM (AGE)

This term implies that gas, predominately nitrogen, has entered the arterial system. The gas may enter from pulmonary barotrauma (through the site of lung damage) or decompression sickness — where venous gas emboli pass through the pulmonary system or via an atrial septal defect. These may only become patent after some of the pulmonary embolism, increasing pulmonary artery and right heart pressures.

Initially, in the larger arteries, the emboli will be distributed according to buoyancy. Divers, during ascent and on reaching the surface, are usually in the upright position and therefore the AGE tends to move to the cerebral circulation. When the bubble reaches a small vessel (e.g. arterioles around 50-200 microns diameter) it may coalesce with other bubbles and physically fill the whole vessel. The bubble is then no longer a sphere, but a cylinder which may bridge over the junctions between this and other vessels. Most of the AGE pass through the arterial system and split up as they reach the capillary bed, which has a much larger cross sectional area. It is then likely to pass into the venous system, subsequently becoming filtered by the pulmonary circuit or elsewhere.

In the cerebro-vasculature system the bubble may block, because of the frictional resistance to flow as it spans three or more generations of vessels. Its travel is then more dependent on the hydrodynamic force (blood pressure) than gravity. If the AGE does become blocked in the cerebral arterioles, a reflex rise in arterial pressure, lasting less than 15 minutes, may well force the embolus through this vascular system.

The diver may have some transitory neurological symptoms, such as loss of consciousness, from temporary blockage of local cerebral blood flow. He may then regain consciousness (presumably as the embolus passes through the arterial system) and may or may not subsequently develop further symptoms. These relapses can be fatal or very difficult to treat.

Secondary pathology of AGE may be due to more emboli, either from the original cause, from a redistribution of the existing AGE, or from blood/bubble or blood/tissue interaction — both taking some time to develop. Target organ disease may also supervene. See DCS.

Treatment of AGE involves immediate recompression with the patient breathing maximum oxygen pressures, and in a head-down position.

Rapid recompression reduces the length of the obstructing bubbles, thereby reducing the resistance to the bubble flow and expediting the passage of the bubble into the venous system.

The head down posture can increase the hydrostatic force driving bubbles through the arterial bed and can possibly direct the gravity affected AGE into areas less damaging than the cerebral circulation.

The use of 100% oxygen, reduces the production of more AGE and decreases those still within the circulation.

In the absence of an appropriate recompression facility, and while this and suitable transport is being arranged, one is limited to the ancillary forms of first aid. Oxygen should continue to be administered in the head-down left-lateral position, until recompression facilities are employed, or the bubbles have been dissipated. 100% O₂ should continue during any transport or changing of position. Some clinicians use steroids in the hope of reducing CSF pressures.

Factors which may aggravate the condition include assuming a vertical, head up position, movement, either active (muscular) or passive (transport), altitude exposure, certain drugs, e.g. aminophylline, heparin, aspirin, etc.

III. PULMONARY BAROTRAUMA — OTHER THAN AIR EMBOLISM

Pulmonary barotrauma has 4 major types of pathology:

Air Embolus, which has been dealt with under AGE.

Pulmonary Tissue Damage.

Pneumothorax.

Mediastinal Emphysema.

The last two of these pathologies can be considered together, as both are simply the presence of unnatural air pockets — in the interpleural space or the mediastinum, sometimes extending into the cervical subcutaneous tissues.

The management of diving induced pneumothorax and mediastinal emphysema is no different than that produced by other causes, in remote areas. They both respond to the effects of denitrogenation, as produced by 100% oxygen breathing.

Usually the resting subjects are considerably improved within a couple of hours and the asymptomatic in a few more. They can then be gradually weaned from the oxygen by including progressively longer air breaks.

There is no genuine need for concern regarding oxygen toxicity, as the treatment will be completed long before 24 hours.

The disorder can be aggravated by coughing, altitude exposure, further diving, overzealous treatment (trying to remove the air surgically by someone untrained in these techniques).

IV. DECOMPRESSION SICKNESS (DCS)

The primary pathology is that of a bubble, developing in either blood or tissues. The intravascular bubbles act pathologically in the same way as other gas emboli.

Treatment of decompression sickness due to intravascular bubbles is along the same lines as that of the treatment of AGE, and this is dealt with previously.

Bubbles within tissue may act by causing foreign body reactions, pressure necrosis or perfusion abnormalities.

Secondary pathologies include:

- Blood/bubble interaction,
- Tissue/bubble interaction,
- Primary and secondary ischemias,
- Hemorrhages, dehydration and hemoconcentration,
- Target organ disease (brain and spinal cord, heart, lungs, etc.).

The conventional treatment of bubbles in tissues is: immediate recompression to reduce the size of the offending bubble, 100% oxygen (off-gassing nitrogen from the bubble), treatment of target organ disease e.g. CPR, epilepsy, urinary retention, general measures, such as rehydration — oral (not alcohol) or i.v. fluids, drug therapy if applicable, hyperbaric oxygen therapy.

Hyperbaric oxygenation has some other values, apart from its affect on the size of bubbles and promoting denitrogenation. These include a reduction in raised intracranial pressure of cerebral DCS and a lessening of the blood/bubble interactions. It also ensures a higher oxygenation of ischemic tissues.

Problems are produced by: delaying treatment and thus increasing morbidity, altitude exposure, muscular activity and rough transport, some drugs, more hyperbaric air exposure.

Whether the academics like it or not, in-water treatment of decompression sickness has been used by many traditional diving groups, such as the pearl and sponge divers. It has also been used in much colder climates by abalone and salvage divers. If an underwater recompression capability is available, or can be improvised, then it is possible to improve dramatically the effects of oxygen breathing by administering this underwater.

V. DROWNING

The primary pathology is hypoxia from impaired ventilation, acidosis and decreased compliance of the lungs due to the fluid aspirated or interference with surfactant activity. Secondary pathology may include the complications of hypoxia, acidosis, pulmonary edema, hemodynamic and biochemical anomalies. Vomiting and aspiration is frequent and may be hazardous. Delayed lung damage may result from foreign bodies, chemicals, infections, etc.

First aid treatment involves: maintenance of airway and respiration, CPR, 100% oxygenation, possibly with positive pressure, positioning of the patient to avoid aspiration of vomitus, avoid unnecessary movement, e.g. physio, monitoring and observation for 24 asymptomatic hours, antibiotics.

VI. HYPOTHERMIA

Mild. In these cases there is a clear level of consciousness, and ability to swallow and there is not uncontrollable shivering. The core temperature is above 34 degrees centigrade. For mild cases all that is needed is to remove the patient from the environment producing the problem, and supply added warmth in the form of hot water bottles in the groin under the arms and around the head, chest and neck, and the use of warm drinks, not coffee or alcohol.

Severe. With core temperatures below 34 degrees, there is often a clouding of consciousness, cardiac instability and muscular rigidity or paralysis. In these serious cases, handle the patient gently, or fatal arrhythmia may eventuate. It is not necessary to remove the clothing, but the patient should be protected from the cold environment. CPR may be required. Active rewarming by immersing the diver in a warm bath or tub, is more effective than the passive rewarming described above. This may not be appropriate for the slowly produced hypothermic patients, such as those exposed to cold or alpine conditions. Death should not be presumed in a hypothermic patient.

VII. INNER EAR BAROTRAUMA

This is one of the commonest serious problems encountered by divers. The basic pathology may be due to either rupture of the labyrinthine windows, usually the round window, or hemorrhage into the inner ear. An alternative pathology recently noted by Japanese workers, is the passage of air across the damaged round window, into the inner ear.

There is a problem in differential diagnosis from DCS, but this is much less common with compressed air diving. If the patient is in a remote locality, the treatment need not vary a great deal between the two disorders. It is very likely that DCS also produces an inner ear hemorrhage.

Inner ear involvement can be indicated by symptoms such as: disorientation and ataxia, persisting after the dive, tinnitus, or a high pitched ringing in the ear, hearing loss affecting the higher frequencies.

Audiometers are not commonly available in remote areas, but wrist watches are. The frequency of a small watch ticking is usually in the 4-8 kHz range. The alarm noises that are able to be produced with digital watches may have even higher frequencies. Thus there is available a wrist audiometer with which to detect high frequency hearing loss in one ear compared to the other, or compared to other peoples' hearing.

It is very common for divers with middle ear barotrauma to complain of a dullness to hearing, a conductive deafness affecting mainly the lower (speech) tones, and which need not imply inner ear damage. This is usually less ominous and self correcting, and may not be detected with the wrist watch. In such a case the chances of inner ear damage are less.

If a presumptive diagnosis of inner ear barotrauma has been made then the patient must be given every possible opportunity to allow the labyrinthine window to heal over and to avoid further hemorrhage.

The first aid is designed to ensuring that there is no physiological increase in CSF pressure produced in the subject, because this can be transmitted through the cochlear aqueduct to the inner ear and is likely to prolong both the window rupture and the hemorrhage. Absolute rest to the inner ear is indicated and this can be achieved by total rest of the subject, in a sitting-upright position. This will ensure there is minimal CSF pressure.

First aid management is as follows:

1. Chair-rest position.
2. No strenuous effort or unnecessary movements.
3. Avoidance of breathholding or ear equalizing maneuvers. Divers employ Valsalva maneuvers when they have ear problems.
4. Avoidance of other activities which could increase the CSF pressures, such as sneezing, nose blowing, straining, sexual activity, etc.
5. Repeated monitoring of the hearing, using whatever means available (e.g. wrist watch).
6. Avoid any drugs which could increase either hemorrhage or vasodilation in the inner ear, e.g. aspirin, nicotinic acid.

7. It may be reasonable to administer oxygen, not only because of the possibility of incorrect diagnosis (inner ear decompression sickness, cerebral decompression sickness) but also because of one of the alternative pathologies proposed for inner ear barotrauma, i.e. the passage of air through the damaged round window into the perilymph system. Under these conditions a trial of perhaps six hr oxygen, with occasional air breaks should be adequate.
8. Very gentle transport (sea level, not aviation) to an area where more sophisticated neuro-otological investigations can be performed.

The first aid treatment is more likely to be effective and appropriate, than the surgical techniques available from more sophisticated neuro-otological centers.

VIII. INFECTIONS

Making allowance for individual hypersensitivities, divers in remote areas should be supplied with antibiotics, both local and oral. These can be used for the various relatively common infections associated with water and diving exposure. These include such conditions as:

1. Coral cuts.
2. Otitis externa.
3. Sinusitis.

It is axiomatic that the divers should be trained in recognizing the symptoms of these disorders, so that a relatively accurate diagnosis can be made, and the use of these antibiotics. To delay such effective treatments until the patient can reach a medical facility can result in very severe infections or permanent damage. Both the diving operation and the individual's health are unnecessarily compromised by withholding these simple measures.

Antibiotics in common use are:

1. Topical Neomycin or Cicatrin, powder and ointment.
2. Kenacomb ear drops.
3. Doxycycline or Erythromycin tablets.

IX. MARINE ANIMAL INJURIES

A. Neuromuscular Venom

Envenomation with respiratory paralysis may result from:

1. Sea snake bite.
2. Blue ringed octopus bite.
3. Some cone shell stings.

These produce a neuromuscular venom without much local inflammation (pain). The treatment is:

1. Cardiopulmonary resuscitation if and when required.
2. Pressure bandage/immobilization techniques.
3. Observation charts with monitoring.

Only after the patient is at a medical facility that has provisions for coping with evenomation, should the pressure bandage be removed. The main problem with management is that the patient is not kept "immobile." If the patient, or specifically if the affected limb, is subjected to movement then the venom will be absorbed despite the application of the pressure bandage. The pressure bandage only prevents lymphatic absorption of the venom if the area is immobilized.

B. Fish Spine Injuries

This evenomation, commonly from rays, cat fish and scorpion fish, produces an intense pain and significant inflammation around the wound. To reduce this pain, the wound (together with some unaffected skin) is immersed in water of approximately 50°C. This is about as hot as most people can stand with the normal skin, and this can be used if a thermometer is not available. If the pain recurs when the wound is removed from the water, it should be reimmersed.

Analgesia may be required. One of the most effective methods is to inject a local anesthetic such as 10 cc. of Xylocaine, without epinephrine or adrenaline, infiltrated directly into the wound puncture. This may have to be repeated a couple of times. The wound should be surgically cleaned as soon as possible and antibiotics used (topical and systemic). Antivenom may be used in the case of stone fish stings.

C. Jellyfish

In cases of box jellyfish (Chironex, sea wasp) injuries large quantities of vinegar irrigated over the area may reduce further damage from the

tentacles. This does not necessarily work with other jellyfish, and for most minor injuries from these animals, a local anesthetic may be of more value in both prevention of further envenomation and in treatment. Xylocaine has been used in the past, but this could be superseded by the new skin penetrating anesthetics, such as EMLA (Astra). Most remedies for burns will produce some degree of relief.

X. GENERAL

A detailed chronological description of both the incident and the post incident activities should accompany the patient, together with full documentation of the first aid treatment. If possible, one conscientious diver should be allocated the task of ensuring that this is performed. The description of the incident can be obtained from the diver, if possible, and the companion diver and other observers. The chronological details of the therapy, together with the patient's response should also be carefully noted. A separate chart should be kept on the patient's vital signs (pulse, respiration, conscious state, temperature, fluid intake and output — measured by very clear standard e.g. use a cup or mug to determine the amounts consumed and passed in the urine. This information should accompany the patient to the designated medical facility.

APPENDIX

A. Oxygen Administration Equipment

1. Mini Cylinders, positive pressure and flow capability, often with a limit of less than 30 min of 100% O₂, sometimes with suction facilities.
2. Large Medical (or Industrial Oxygen Cylinder) (220 cu ft, 7000 liters), with dedicated 1st stage regulator (550 kPa, 80 psi) and dedicated 2nd stage regulator or flow meter (for bag). Adaptors: AirDive, Manly, Bendeeze, etc.
3. Rebreathing set with a small O₂ cylinder and CO₂ absorbent, e.g. Draeger SR45, Navy/Fire-fighting/Mines O₂ sets.
4. Superoxide Rebreather. Chemical supply of oxygen, absorb CO₂. Suitable for air transport e.g. AUER, for longer than 4 hours.

Training is required for both surface and underwater use, including fire and explosion risks and prevention. It is in common use by respiratory patients, divers and paramedics.

B. Underwater Oxygen Recompression

The underwater oxygen regime is specifically of value in cases where there is likely to be a delay in the transfer to a chamber, or where the area is so remote that many hours will be lost during the transfer. It is also of more value in the recent cases, which are the very ones otherwise most likely to deteriorate.

A surface supply oxygen system is very easily developed and is dedicated entirely to this eventuality. It comprises:

- A G-size oxygen cylinder (220 cu. ft. or 7,000 litres),
- An oxygen regulator set at about 550 kPa (80 psi),
- A safety valve,
- About 12 meters (about 40 feet) of supply hose,
- A non-return valve at the diver's end,
- A demand valve,
- A full face-mask.

Technique

The affected diver is taken to a maximum of 9 m (often off to a wharf) breathing oxygen. The companion diver is on air and can communicate with the surface. After 30-60 min (depending on symptoms and severity) the ascent is commenced at 12 min per meter. On the surface, the O₂ is continued with progressively increasing air intervals. Recurrence of symptoms, which are sometimes produced by more rapid ascents, require a halting of this ascent.

There has been some suggestion by Hawaiian groups that a deeper air exposure, followed by oxygen in the shallower depths, may facilitate the treatment. A few minutes at greater depth, in the case of intravascular bubbles, could be of benefit. It is very hard to understand how this could be of value for the bubbles within tissues.

The advantages of underwater regime are:

- Immediate reduction of symptoms, by Boyle's Law,
- Maximal outgassing of bubble N₂ by combined pressure and O₂,
- Maximal outgassing of tissue N₂ by combined pressure and O₂, and
- Hyperbaric oxygenation of tissues.

Early treatment is able to be implemented because of the shallow depth (9 m or off wharf), avoids hazardous sea conditions, wet suits are still effective, attendants not exposed to DCS, narcosis, high seas.

Termination of treatment is possible without N₂ load penalty e.g. for reassessment, transport, loss of gas supply, etc.

With this set-up even semi-conscious divers have been adequately treated by companion divers, and the full-face mask allows for a variety of activities, such as communication and vomiting, without prejudicing the diver's air supply. Currently the underwater oxygen system is used in many of the island countries around Australia, especially by the locals, who may not be able to afford the cost of transportation to hyperbaric facilities in Australia. It also tends to be used by the small commercial diving operations in these areas, and abalone divers in the much colder waters in the south of our continent. In areas such as the Australian bases in the Antarctic, where no recompression facilities are available, and diving can be carried out throughout the year, it could also be used — but with significant modifications in protocol and equipment.

C. Positioning, Moving, and Transport of the Patient

In some recent American publications, a diagram of the head-down position with the patient lying supine was proposed and was advised for AGE, DCS, or near drowning. In fact such a position would certainly aggravate many near drowning cases, as aspiration is almost inevitable, unless there was a cuffed endotracheal tube inserted. Similar reservations must be held for the dysbaric disorders especially if there is any compromise of respiratory function.

Because of recurrent or redistributed emboli, the head down left lateral position should be maintained in cases of AGE. The concept of recurrent embolization has been used to describe the cases that suddenly deteriorate, after initial symptoms may have cleared, especially when the patient resumes an erect posture.

Body movement, both active (muscular) and passive (transport) may aggravate a number of diving illnesses. These include:

1. *AGE* — the upright position may aggravate redistribution of emboli in large arteries, towards the cerebral circulation, and reduce the hydrostatic forces that drive obstructed bubbles through the cerebral circulation.
2. *DCS* — muscular activity or body movement can precipitate VGE.
3. *Near-Drowning* — movement (esp. physiotherapy) redistributes loculated intrapulmonary fluids, and decreases compliance.
4. *Hypothermia* — movement precipitates cardiac arrhythmias, possibly by returning a bolus of acidotic blood from hypothermic tissues to the heart.

5. *Inner Ear Barotrauma* by perilymph pressure or hemorrhage.
6. *Marine Envenomation* — negates the pressure bandage effect.
7. *Shark Attack* — increases blood loss.

D. Aviation Medivac

The benefit of moving a seriously ill patient by aircraft to more sophisticated medical facilities, must be weighed against the hazards of this procedure. Disembarking a patient from an aircraft in a much worse clinical condition than when he went in is a humbling experience.

Air transport is of most value in AGE and DCS if it is carried out with continuous medical support, in the following order:

1. Under hyperbaric conditions (portable RCCs) — recommended.
2. With aircraft pressurized to ground level — possible.
3. At altitude pressures — needs careful consideration.

Because of the value of hyperbaric oxygenation in the acute treatment of decompression sickness, prompt contact should be made with a recompression facility, to initiate retrieval under hyperbaric conditions. Depending on the area this might involve a transfer of a portable RCC to the site, and treatment within that facility, while the patient is being transferred to a major RCC center, with appropriate transfer under pressure facilities.

There are certain problems that develop during aviation medivac activities. Often the physical disruption seems to make the patient worse.

1. It is possible that the vibration in many helicopters, and to a less degree in fixed wing aircraft may aggravate the clinical condition.
2. Any exposure to altitude will expand bubbles and thereby increase damage. This is even observed by patients with decompression sickness driving motor vehicles over hills and mountains. There is no "safe" altitude at which one can fly, once a bubble is present. Whether there is such a safe altitude for asymptomatic divers, could be argued, but those with symptoms do not have such a luxury.
3. Often the aircraft can only take one-man chambers. Under these conditions there is an impaired capability of patient care throughout the transfer.

4. Unless there is a formal aviation medivac facility, such as in the armed forces, there tends to be more attention and decision-making related to the transport logistics than patient care.
5. There is often a loss of continuity of treatment and communications during the transport.

on the other hand, it is not possible to find a single set of conditions which will hold for all cases. The only way to avoid this is to restrict the scope of the theory to a specific set of conditions.

It is also possible to restrict the scope of the theory to a specific set of conditions. This is done by specifying the conditions under which the theory is to be applied.

10

Changes Found in the Central Nervous System from Autopsies on Divers

Ian M. Calder

| | |
|--|-----|
| I. Introduction | 163 |
| II. Neuropathology of Decompression Sickness (DCS) . . . | 164 |
| III. Morphological Observations | 165 |
| A. Acute Changes | 167 |
| B. Sub-Acute Changes | 167 |
| C. Chronic Changes | 168 |
| IV. The Brain and DCS | 173 |
| V. DCS and the Inner Ear | 174 |
| VI. Discussion | 175 |
| VII. Conclusion | 177 |
| Acknowledgements | 178 |
| References | 178 |

I. INTRODUCTION

With the exploitation of gas and oil resources in the northern sector of the North Sea of Europe up to 3,000 divers were employed in the 1970s. Inexperience, new technology and hostile weather conditions resulted in numerous fatal accidents. It was fortunate that an accident investigation philosophy developed based on that used by the United Kingdom Royal Air Force (Calder, 1985). By this method it was possible to have a limited number of observers and build up a data bank on the tissues obtained from the autopsies. Such material has been rare and valuable, as the opportunity had not previously presented itself to study a series of workers exposed for long periods to a hyperbaric environment with numerous compressions and decompressions.

It has also been fortunate to have the cooperation of Diving Contractors on a world-wide basis who have accepted the rationale for a thorough autopsy and detailed investigation of fatal accidents, especially in countries where there is no formal or statutory method of investigating fatal accidents.

This has enabled a more scientific appraisal of tissue changes to be made, although as long ago as 1908 (Boycott *et al.*, 1908) observations were reported on sixty fatal pearl diving accidents. Unfortunately these were only investigated at the macroscopic level as no microscopic techniques were available.

Analysis of data of such accident situations may also prove a useful indication of prevention of accidents by regulation (Calder, 1979).

From the point of view of pathology induced by the hyperbaric environment evidence is accumulating that the effects are on multiple organs, other than bones and spinal cord — lung (Calder *et al.*, 1987), ear (Money *et al.*, 1985), eye (Bird *et al.*, in press), teeth (Calder and Ramsey, 1983) and liver (Dornan, 1988).

The purpose of this presentation is to discuss the spectrum of changes in the central nervous system which is essentially confined to the spinal cord.

II. NEUROPATHOLOGY OF DECOMPRESSION SICKNESS (DCS)

A detailed review of the literature in 1957 (Haymaker, 1957) showed that soon after decompression spinal hemorrhage commonly occurred, affecting both white and grey matter, especially in the upper thoracic cord. Survivors for longer periods showed softening of white matter in the dorsal and lateral columns (Van Rensselaer, 1891). A caisson worker examined histologically twenty-five years after partially recovering from spinal paraplegia showed similar tract degeneration with preservation of subpial myelin (Lichenstein and Zeitlin, 1936). It was suggested that there was a primary lesion in the thoracic region, and that there was secondary upward and downward tract degeneration to cervical and lumbar regions, and that the white matter infarcts were due to intra-arterial bubble formation. These early reports showing degenerative changes in grey matter of the cord and involvement of the brain were rarely described.

In a review of seventeen cases of DCS at altitude (Fryer, 1979) pathological changes observed were disappointing, possibly due to the short survival time after the incidents.

Since the 1957 review there have been several reports of clinical features of spinal DCS (Frankel, 1977). Reports on the pathology of such cases have been confined to two caisson workers.

The Japanese have published some pathological reports on fatal diving accidents (Hayashi, 1974). Bubble formation was described in liver, heart, brain and spinal cord. Independent observers found similar changes (Takagi and Mano, 1982) but, in addition, platelet aggregation in the vicinity of the bubbles. In addition more profound changes were found in the spinal cords of two divers who had suffered Type II DCS with five and fifteen day survivals. There was conspicuous necrosis of the dorsal and lateral columns in the case which survived for fifteen days.

Post-mortem findings in a diver who died eight hours after an acute episode of Type II DCS were described in 1981 (Kitano and Hayashi, 1981). Fat emboli were found in pulmonary arterial capillaries, epidural veins of the spinal cord, glomerular capillaries, but not in the brain. It was suggested these originated from necrotic bone marrow by way of the sinusoids. In a case of Type II DCS in a diver who survived for twenty-four hours bubbles were found in the superficial veins of the brain, spinal cord and bone marrow (Kitano *et al.*, 1977). Although no lesions were observed in the brain early degenerative changes were seen in the cervical thoracic cord.

The Russians have reported necrosis in fatal diving accidents (Akimor *et al.*, 1969) and in one spinal cord there was severe damage to the thoracic cord where architecture was replaced by hyperplastic vascular tissue, astiocytes and macrophages. Long-term damage affecting dorsal and lateral columns between cervical and lumbar segments with the primary focus of infarction between the lower cervical and mid-thoracic levels.

These reports on the observations of the neuropathology of Type II DCS in man supports the role of the intra-vascular bubble widely distributed throughout the body and resulting in infarction of the spinal cord.

III. MORPHOLOGICAL OBSERVATIONS

The histological examination of the human spinal cord tissue is of necessity limited due to autolytic changes occurring between death and time of autopsy with subsequent fixation. Although it is possible to discriminate, using the light microscope, to 5μ much cytological detail is lost. This can however be complemented by material from animals subjected to experimental Type II DCS. By this it is possible to build up a mosaic of the pattern of changes. The use of histochemical, together with immunoperoxidase techniques can more specifically identify areas of degeneration even to the level of a single axon which would escape notice by hematoxylin and eosin staining.

Some of the earliest, and very elegant, observations on experimental DCS were published in 1900 (Haymaker, 1957). These showed that sixty minutes after decompression petechial hemorrhages were present. At five days there was focal malacia in the white matter with, sometimes, necrosis of the grey matter. The lateral columns were most frequently affected with preservation of the sub-pial white matter. The dorsal and ventral columns were also frequently affected.

In 1908 Boycott (Boycott *et al.*, 1908) conducted original experiments on goats in the course of the design of safe decompression tables. Bubbles were observed intra-arterially and intravenously in many sites, including cerebrospinal fluid and urine, but were guarded in attributing too much significance to the presence of bubbles found at autopsy to their situation in life. The occurrence of bubbles outwith vessels were most frequently distributed in the spinal cord, which are the so-called 'autochthonous bubbles'. The development of these at microscopic level, although it may appear to have little significance, may leave epitaphs in the way of damage to individual axons. There is considerable reserve in the spinal cord and such damage as will be later described cannot be detected by conventional diagnostic techniques. However, the cumulative effects of multiple such insults will result in some long-term compromising of the spinal cord.

This view of the spinal cord is however somewhat controversial as these observations of Boycott mistakenly interpreted status spongiosus — a feature of an established infarct of white matter — as being directly related to bubble formation occurring immediately after decompression. Acute extravascular bubble formation can be expected to disrupt the white matter matrix, which is readily distinguished from status spongiosus. This error has led to the assumption that extravascular bubble formation is an important feature in initiating the lesions of Type II DCS. This does not however negate the earlier thesis of the single autochthonous bubble causing a minute lesion.

Nevertheless, despite the concept of extravascular bubble formation Boycott considered the pathogenesis of lesions in the white matter of the spinal cord was related to the distribution of small bubbles by the blood stream. The slow circulation in the white matter allowed bubbles to increase in size causing infarction due to the effects of bubble emboli.

Localization of intravascular bubbles in the spinal cord after decompression was investigated in dogs (Hallenbeck *et al.*, 1975) and found to cause widespread regional obstruction of the epidural vertebral venous system, and it was suggested this made a significant contribution to the pathogenesis of spinal cord lesions. These are the typical changes of acute venous infarction — hemorrhage in the white matter with spanning of the grey matter.

Elegant studies of the distribution of blood vessels in the spinal cord not unexpectedly closely relate to areas of damage due to DCS (Adamkiewicz, 1884, Hassler, 1966).

Morphological changes recognized by light microscopy form the standard pattern of reactions in the central nervous system which are chromatolysis and ischemic cell changes in neurons, axonal swelling, proliferation of microglia, lipid phagocytes and inflammatory cells as well as vascular hyperplasia. Such changes take between 24 and 48 hrs to develop. From the practical aspect in order to identify the genesis of DCS in man, it is within this time zone research has to be concentrated. Observations on the immediate changes following DCS in the main have relied on experimental work on goats (Palmer *et al.*, 1976) which complements that in man.

The pathological effects of DCS may be broadly divided into three time zones — acute, sub-acute and chronic.

A. Acute Changes

The acute lesions may range from small wedge-shaped infarcts, with the bases pointing towards the periphery of the cord, to massive infarcts affecting all the white matter, but always spanning a rim of sub-pial myelin. Especially affected are the dorsal and lateral columns. The histological changes in the time zone of 24 and 48 hrs is that of focal areas of Wallerian degeneration, swollen axons and distended sheaths sharply demarcated from the surrounding normal tissue. The foci of degeneration are often centered round a vessel with pyknotic endothelial nuclei with occasional evidence of slight hemorrhage in the infarcted area. Grey matter is generally spared the major effect of infarction, although again small hemorrhages may occur.

In experimental material from goats it has been observed that there are perivascular proteinaceous globules associated with small capillaries; this is now complemented by human material. Associated with the vascular lesions microthrombi were usually found in what were probably arterioles in the white matter. Ultra-structural studies on formalin fixed material have shown the microthrombi to be associated with endothelial loss and platelet aggregation (Palmer, 1986).

B. Sub-Acute Changes

Observations on the early organization and repair of infarcted areas depends on experimental material (Palmer and Blackmore, 1980). The first stages become apparent at about five days after decompression.

Lipid phagocytes accumulate with vascular hyperplasia and later gliosis. After two weeks no damage is detectable in the grey matter, and at three-six months the only remaining evidence may be a small gliotic scar, often difficult to detect. Such a scar has however to be regarded as an area of permanent damage and consequently with a loss of function, which individually may be of no clinical significance, and not detectable by routine clinical techniques. The cervical cord appears to be the area of predilection, but this certainly reflects the total amount of tissue at risk.

C. Chronic Changes

This may be considered to be the most important aspect of the effects of DCS. Over many decades many thousands of divers have suffered DCS incidents from which complete or partial recovery has been achieved — with or without treatment. However, it is to the group who have no residual signs that the detailed pathological/histological investigations have to be addressed. It is only by following up as many fatal diving accidents as possible that patterns of change emerge, and then only by careful review of diving and medical histories.

Until 1981 there was little evidence that there was residual damage to the spinal cord following treated Type II DCS. However, a case of 'recovered' spinal DCS was reported (Palmer *et al.*, 1981). This concerned an amateur diver who had suffered Type II DCS with significant clinical signs. These subsequently recovered following treatment, and although subjectively recovered, on examination four years later minimal signs were present. Some twelve days after this examination he met a violent death.

At post-mortem examination there was a long-standing tract degeneration which appeared to stem from focal damage in the spinal cord at C₇ to T₄. The fasciculus gracilis showed bilateral tract degeneration from C₁ to T₄, although parts of it had survived. The lateral corticospinal and spinocerebellar tracts showed degeneration from C₁ to L₄, and the vertical columns were damaged from T₃ to T₅. Sub-pial myelin was present.

Such findings pose three important questions:

1. What is recovery from spinal DCS?
2. How can cord damage be detected during life?
3. What advice shall be given with regard to future diving activities?

This case can be complemented by a further case in which paraplegia was induced following severe Type II DCS (Calder and Palmer, 1988). The histological findings were similar in distribution to the previous case but quantitatively greater. This poses the dilemma of the fine balance between 'clinical recovery' and resulting spinal cord disaster.

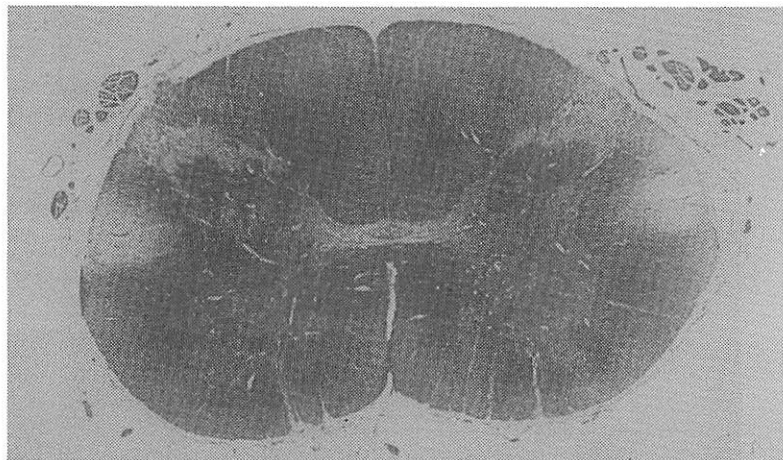


Fig. 1. Cervical cord at C₃ from 48-year old male who survived 4-years as a paraplegic following a Type II DCS incident. Degeneration of posterior and lateral columns, with conspicuous hyalinized vessels. Subpial myelin and grey matter preserved. MFB x 14.

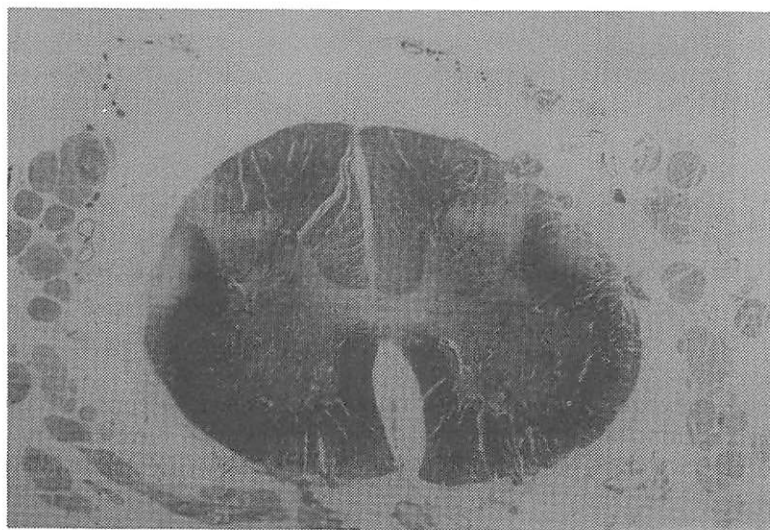


Fig. 2. Cervical cord at C₇ with degeneration of lateral corticospinal tracts. MFB x 14.



Fig. 3. Cervical cord at level of C₅ from 38-year old male who had 'recovered' from a DCS incident, with loss of myelin from dorsal and lateral tracts.

There is however a much more important aspect to consider in the routine examination of spinal cords from divers who have had no evidence of DCS. The relevant features are the identification of subtle changes which can only be identified by sensitive histo-chemical methods of which the Marchi method is the most significant for identifying degenerate myelin (Swank and Davenport, 1934).

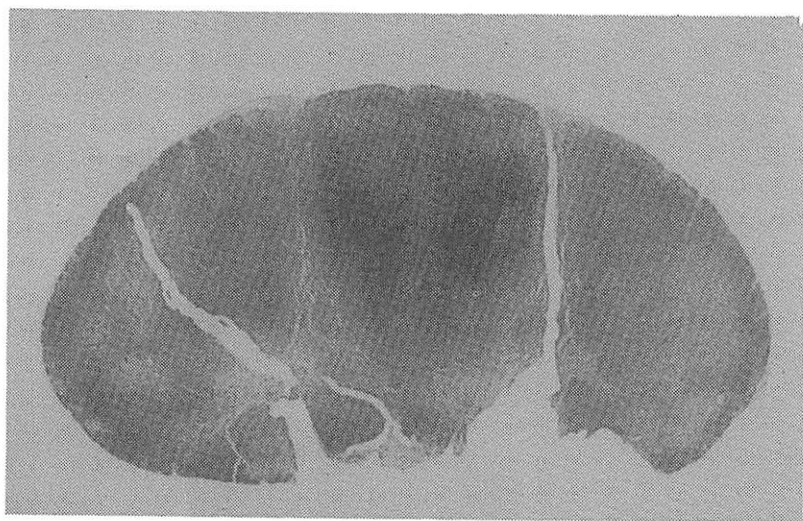


Fig. 4. Marchi positive degeneration of C₇ in posterior columns.

Marchi positive material does not occur in degenerating myelinated fibers until seven to ten days after the initiating lesion (Smith, 1951). Also it does not appear intracellularly until 10 weeks after the lesion has been initiated.

Unfortunately the Marchi method can only be applied for technical reasons to tissue fixed in formal saline, and much of the available material had been processed into paraffin wax. This limited the amount of material suitable for this specific stain. This was applied to eleven cases with ages ranging from twenty-two to thirty-seven (mean 28.4) years (Palmer *et al.*, 1987).

In tissues routinely stained by the hematoxylin and eosin method minor chronic changes were found which consisted of hyalinization of blood vessels in the white matter, corpora amylacea and occasional foci of gliosis in the gracile funiculi and some fibrosis in the posterior nerve root in one case. These features of chronic degenerative change were judged to be small but relevant in view of the age group studied.

The sensitive Marchi stain has, however, been found to be positive and to highlight areas of degeneration not found by the hematoxylin and eosin method. It was found that there was a wide distribution of positive staining in three of the cases. There was degeneration in some of the posterior column and anterior and lateral columns, with bilateral degeneration



Fig. 5. Degeneration in posterior, lateral and anterior columns. Marchi x 10.

of the tract of Lissauer in one case. In all the cases the Marchi-positive material was extracellular and took the appearance of true degeneration (Smith, 1956).

Although only a small number of cases have so far been examined and reported the results must be regarded as significant. The lesions could not have arisen from other causes, such as multiple sclerosis, subacute

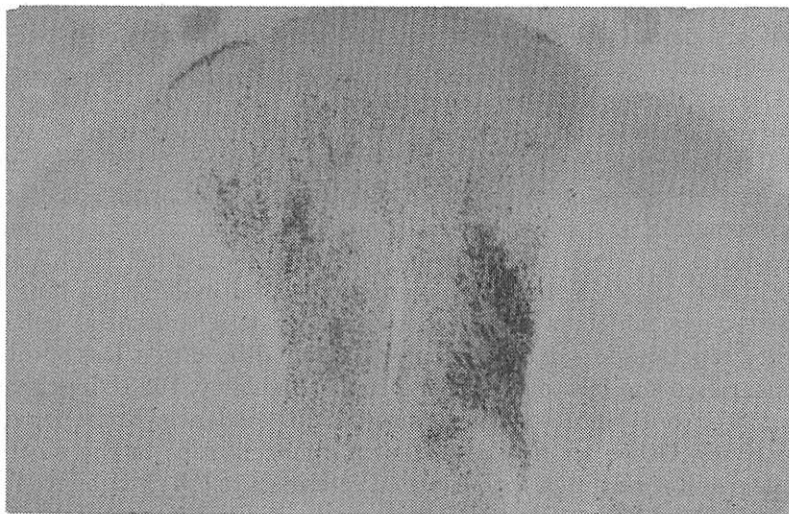


Fig. 6. Marchi positive degeneration in afferent fibres of posterior columns, Lissauer's tract and the anterior and lateral columns. Marchi x 10.

combined degeneration of the cord or alcohol toxicity, as these are readily recognizable in paraffin sections stained with hematoxylin and eosin. Thus it has to be concluded that the degeneration observed was induced by diving activities. From what has been previously described it is accepted that Marchi-positive material does not appear in degenerating myelinated fibers until 7-10 days after the initiating lesion. Moreover, it does not appear intracellularly until about ten weeks after the lesion. The Marchi-positive material described appears therefore to have occurred between one and ten weeks before death. There was, however, no record of any diving accident during this time zone and there had been no clinical evidence of spinal cord changes. These findings would suggest that some divers are unwittingly working with tract degeneration in the spinal cord.

IV. THE BRAIN AND DCS

It is surprising that lesions and changes in the brain resulting from DCS have not been documented. In view of the damage found in the relatively small and compact spinal cord weighing some 40 g it is also interesting that some of these changes have not been reflected in an

organ some thirty times greater (Boyd, 1962). This may of course be due to the paucity of material which has been examined histologically. Such examination may be fraught by technical difficulties, not the least of which is poor preservation of cytological detail due to poor fixation. Blood vessels are usually better preserved and may be a more definitive indicator in future research, especially in view of the *prima facie* evidence of small vessel damage in the spinal cord.

Palmer has reviewed a series of goat brains from cases used for experimental spinal DCS and no changes have been observed. This may be due to anatomical variation in the blood supply to the goat brain with the rete mirabilis acting as a filter. There is, however, accumulating evidence of behavioral effects in divers (Rozsahegyi, 1959; Elliott *et al.*, 1974; Vaernes and Eidsrik, 1982). This complements anecdotal evidence of long-term brain damage which is manifest as memory loss, antisocial behavior, retarded reaction, loss of concentration, visual difficulties, loss of interest in appearance and low tolerance of alcohol (Baddeley, 1983). In a group of caisson workers there was more substantial evidence (Rozsahegyi and Roth, 1966). Other studies have shown abnormalities in the electroencephalograph recordings with especial reference to the rhombencephalon and neuropsychological tests (Peters *et al.*, 1977; Nix and Hopf, 1980). The Diver Performance Research Unit at the University of Lancaster suggests that there is a deterioration in short-term memory and reasoning skills among deepsea divers in the British Isles, especially in those working for more than 8 years. More recently this evidence has been substantiated in a series of professional abalone divers (Williamson *et al.*, 1987). Long term behavioral changes have been reported following DCS at altitude (Mebane, 1955).

It has earlier been mentioned that there is so far little evidence detected of morphological damage to the brain. In acute cases of air/gas embolism, even in those cases who survive for 24 hr following recompression/resuscitation, it is too early for morphological and cellular changes to develop. It would, however, be expected in the long term for the changes to be ischemic, an observation which has been found fitting Type II DCS at altitude. Other such cases which have been reported show mainly perivascular edema in hemorrhage (Haymaker, 1957, Sillery, 1958).

V. DCS AND THE INNER EAR

The inner ear is considered to be part of the central nervous system and the causations have to be differentiated from auditory barotrauma and auditory deficiency resulting from DCS damage to the brain stem.

In acute cases of Type II DCS induced inner ear injury there is hemorrhage in the perilymph spaces of the cochlea. In a chronic case in which a diver had suffered from vestibular and auditory signs there was new bone growth in the semicircular canal of one ear 56 days after the initial incident (Money *et al.*, 1985).

Experimentally induced inner ear dysfunction has been produced in the squirrel monkey (Landolt *et al.*, 1977). Histological studies were carried out in a series of animals killed from one hour to twelve months after the decompression episode. In animals killed one month or less after the decompression incident hemorrhage and deeply staining basophilic material was found in the otic fluid spaces. Similar material was seen to a lesser extent in control animals. The animals killed at more than a month after decompression showed that new ectopic bone growth was present in the areas of the semicircular canals but not in the cochlea. It has been suggested that bubbles forming within the osteoclasts of the semicircular canals may result in fracture (Fraser *et al.*, 1983).

More recent work in minipigs subjected to multiple compression/decompression (Wilkes *et al.*, in press) has shown that there is a loss of hair cells throughout the cochlea. These effects were observed despite the fact that there was adherence to a standard decompression schedule.

VI. DISCUSSION

It is only within the last decade that attention has focused on the probable long term effects of DCS. In fact it does appear that it may be the very effects of an alien hyperbaric environment with no manifestation of DCS that can cause changes, and there may be subtle effects on multiple target organs. This is by way of contrast to the gross effects of dysbaric osteonecrosis. This, however, on the one hand may be due to the more readily available techniques for diagnosis. On the other hand many cases of spinal DCS must have recovered and left no overt clinical signs, possibly due to the lack of sensitive diagnostic methods. It is only by careful examination in the long term of tissues from a diving population that a realistic appraisal can be made.

The case of a 'recovered' spinal bend reported in 1980 identified the potential problems of apparent clinical recovery which may mask gross pathological changes (Palmer *et al.*, 1981). In this an amateur diver suffered a severe Type II bend, from which almost complete recovery was achieved, and kept under clinical review. Four years later he was killed and the spinal cord obtained at autopsy. Histology showed considerable damage, especially to the posterior columns, which was out of proportion to that which could have been expected from clinical examination. This

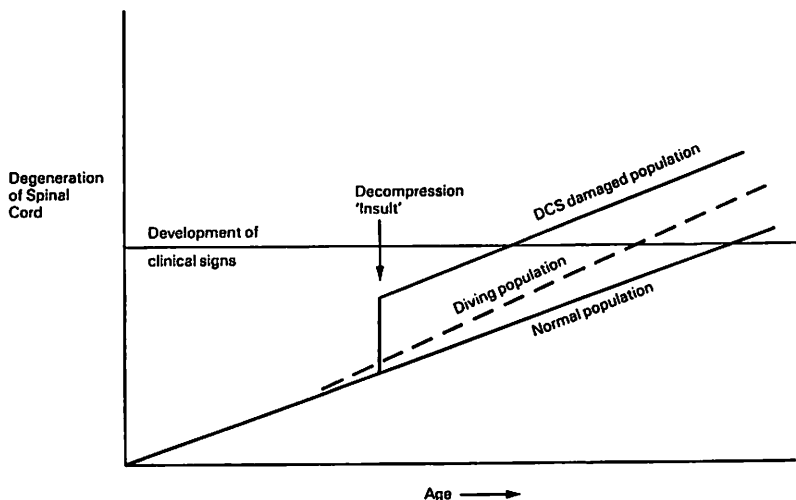


Fig. 7. Diagram showing relationship between normal degeneration of central nervous system with age, against probable changes due to diving and following a DCS insult. The age of onset of possible clinical signs is indicated.

case poses a dilemma as to whether complete recovery from a spinal bend occurs and furthermore whether such an incident should preclude further diving activities. This problem was addressed in 1983 at a symposium on the long term consequences of deep diving, the results of which were rather inconclusive (McCallum *et al.*, 1983).

It would seem however, taking the basic pathological premise that there is no regeneration following damage to the central nervous system, that any insult or insults compromise the reserve. On theoretical grounds this would therefore substantiate the argument that any case suffering a spinal bend should not return to diving, on the assumption that a subsequent incident could result in further damage giving clinical manifestations (Fig. 7). However, the problem of restricting further diving on the grounds of history is fraught with medico-legal ramifications.

The distribution of lesions induced by DCS in the cord bears an interesting anatomical relationship to the vascular supply. Despite the severity of the insult a small sub-pial rim of white matter is preserved as is also the case in the grey matter. In addition there is considerable variation of arterial arcades between various segments of the spinal cord. This is of relevance in that blockage of the anterior spinal artery or central arteries would cause a lesion of larger extent in cervical and

lumbo-sacral cord rather than thoracic. This accords with the histological findings following severe DCS.

From detailed histology it does appear that small arteries reflect damage by DCS and this has been found within the spinal cord and nerve roots. This is hyalinization and cannot be regarded as an age change. It does, however, have to be considered that such changes may not be the result of overt DCS and reflect the effects of 'silent' bubbles. It has been predicted on theoretical grounds that even following a recognized decompression schedule these bubbles may remain in the circulation for up to 4 days (Personal Comm. Daniels, 1988). The presence of a microbubble in the circulation has been investigated in detail (Ackles, 1973). Much of this has been as a result of acute and major DCS insults and has shown thrombotic changes within vessels.

It is necessary though to consider the role of multiple micro-bubbles within vessels over considerable periods of time which can cause changes. This can be regarded as the concept of Virchow's Triad (1860). In this there are three factors promoting thrombosis — the vessel wall, the flow of blood and the constituents of blood. All these can be affected by bubbles. The intima may be damaged by the change of electrical potential at the cell/gas interface; the flow of blood may develop eddy currents due to the change of rheology by the physical presence of bubbles; and finally the very presence of bubbles may initiate the cascade of blood clotting. The concept of this process may then be related to the increase of the vessel wall and the leakage of protein ultimately developing hyaline change.

It is finally necessary to consider the long term effects on the cord. It is known that there are well recognized degenerative changes in the spinal cord with age (Bailey, 1953). Such changes do not usually commence until about the age of fifty years. However, there are two important aspects of this with regard to diving. The one is those who 'recover' from a spinal DCS incident, and these undoubtedly will have clinical manifestations of cord degeneration at an earlier age. The other is applicable to those in that group who have had no clinical manifestations of DCS but develop occult damage and therefore have premature aging of the cord.

VII. CONCLUSION

The pathological findings in the central nervous system are obviously more privileged than those which can be obtained by clinical techniques. Nevertheless, damage is present and the whole problem of application of more sophisticated diagnostic methods has to be addressed. This is not only in the context of the problem of fitness to dive, but also in the long term consequences of diving.

ACKNOWLEDGEMENTS

Thanks are due to Mrs. Susan Crane for help with the typescript and to Mr. Ivor Northy for assistance with the illustration.

REFERENCES

- Ackles, K.N. Blood-bubble interaction in decompression sickness. *DCIEM Conference Proceedings*, No. 73-CP-960, 1973.
- Adamkiewicz, A. *Sitzungsberichte der Mathematisch-Naturwissenschaftlichen Classe der kaiserlichen Akademie der Wissenschaften*. 8vo. Wien. Vols. 69, 1874-88, 1884.
- Akimov, G.A., M.P. Elinskii, and A.M. Lovovskii. Changes in the nervous system with decompression sickness. *Zhurnal Nevropatologii i Psikiatrii imeni S.S. Korsakova* 69:979-984, 1969.
- Baddeley, A. Long term neurological consequences of deep diving. In: T.G. Shields, B. Minasaas, D.H. Elliott, and R.I. McCallum, eds. *Proceedings of Workshop Organized by the European Undersea Biomedical Society and the Norwegian Petroleum Directorate*. Stavanger, Norway, p. 165, 1983.
- Bailey, A.A. Changes with age in the spinal cord. *Arch. Neurol.* 70:299-309, 1953.
- Bird, A., D. Polkinhorn, and M. Cross. *Choroidal Vascular Change Following Decompression*. In press.
- Boycott, A.E., G.C.C. Damant, and J.S. Haldane. The prevention of compressed air illness. *J. Hyg.* 8:342-443, 1908.
- Boyd, E. *Growth, Including Reproduction and Morphological Development. Biological Handbooks*. Washington: Federation for Experimental Biology, p. 346-348, 1962.
- Calder, I.M. The prevention of the re-occurrence of fatal underwater accidents. V. Annual Meeting of European Underwater Bio-Med. Soc., Bergen. 1979.
- Calder, I.M. and J.D. Ramsey. Odontocrexia — The effects of rapid decompression on restored teeth. *J. Dent.* 11:318-323, 1983.
- Calder, I.M. A method for investigating specialized accidents with special reference to diving. *For. Sci. Int.* 27:119-127, 1985.
- Calder, I.M., C.K.K. Sweetenham, and M. Williams. Relationship of alveolar seizure to forced vital capacity in professional divers. *B.J. Ind. Med.* 44:467-469, 1987.
- Calder, I.M., A.C. Palmer. Spinal cord degeneration following Type II decompression sickness. *Paraplegia* 1988 (in press).
- Dornan, J. *Liver Enzyme Changes in Experimental Diving*. Royal Society of Medicine, London. November 1988.
- Elliott, D.H., J.M. Hallenbeck, A.A. Bove. Acute decompression sickness. *Lancet* II:1193-1199, 1974.
- Frankel, H.L. Paraplegia due to decompression sickness. *Paraplegia* 14:306-311, 1977.
- Fraser, W.D., S.P. Landholt, K.E. Money. Semicircular canal fractures in squirrel monkeys resulting from rapid decompression. Interpretation and significance. *Acta Otolaryngol.* 95:95, 1983.
- Fryer, D.I. Subatmospheric decompression sickness in man. *Technivision Services, Slough* p. 1-343, 1979.
- Hallenbeck, M.M., A.A. Bove, D.H. Elliott. Mechanisms underlying spinal cord damage in decompression sickness. *Neurology* 25:308-316, 1975.
- Hayashi, K. Clinical and experimental studies on decompression sickness. *Fukuoka Acta Med.* 63:889-908, 1974.

- Hassler, O. Blood supply to human spinal cord. *Arch. Neurol.* 15:303-307, 1966.
- Haymaker, W. Decompression sickness, In: O. Lubarsch, F. Henke, and R. Rossle, ed. *Handbuch der Speziellen Pathologischen Anatomie und Histologie. Nervensystem.* Springer-Verlag, Berlin, p. 1600-1612, 1957.
- Heller, J. Quoted by Haymaker, W. 1957.
- Kitano, M. Hayashi, and M. Kawashima. Three cases of acute decompression sickness. *Journal of West Japan, Orthopaedic Trauma* 26:402-408, 1977.
- Kitano, M. and K. Hayashi. Acute decompression sickness. *Acta Pathologica Japonica* 31:269-276, 1981.
- Landolt, J.P. K.E. Money, E.D.L. Topliff, K.D. Powers, and W.H. Johnson. Vestibule cochlea dysfunction in squirrel monkeys in simulated diving experiments. *Med. Aeronaut. Spat. Med. Subaquat. Hyperbar.* 16:377, 1977.
- Lichenstein, B.W. and H. Zeitlin. Caisson disease — A histologic study of late lesions. *Archives of Pathology* 22:86-98, 1936.
- McCallum, R.I., T.G. Shields, B. Minsaas, and D.M. Elliott. *Long Term Neurological Consequences of Deep Diving.* Stravangen Norway: As Verbum, 1983.
- Mebane, J.C. Clinical neuropsychiatry in aviation medicine. *J. Aviat. Med.* 26:471-478, 1955.
- Money, K.E., I.P. Buckingham, I.M. Calder, W.H. Johnson, J.D. King, J.P. Landolt, J. Laufer, and H. Ludman. Damage to the middle ear and the inner ear in underwater divers. *Undersea Biomed Res.* 12:77-84, 1985.
- Nix, W.A. and H.C. Hopf. Schädigungen des zentralen nervensystems nach dekompensionszwisehenfällen. *Deutsche Medizinische Wochenschrift* 105:302-306, 1980.
- Palmer, A.C., W.F. Blakemore and A.G. Greenwood. Neuropathology of experimental decompression (dysbarism) in the goat. *Neuropath. Appl. Neurobiol.* 2:145-156, 1976.
- Palmer, A.C. The neuropathology of decompression sickness. *Recent Advances in Neuropathology.* No. 3. Ed Cavanaugh, pub. Churchill 1 Livingstone, p. 149, 1986.
- Palmer, A.C. and W.F. Blakemore. Neuropathology of experimental decompression (goats). In A. Grimstad, ed. *Proceedings of the 5th Annual Scientific Meeting of the European Undersea Biomedical Society.* Bergen, p. 153-156, 1980.
- Palmer, A.C., I.M. Calder, R.I. McCallum, and F.L. Mastaglia. Spinal cord degeneration in a case of 'recovered' spinal decompression sickness. *British Medical Journal* 283-888, 1981.
- Palmer, A.C., I.M. Calder, and J.T. Hughes. Spinal cord degeneration in divers. *Lancet* 11:1365-1366, 1987.
- Peters, B.H., H.S. Levin, and P.J. Kelly. Neurologic and psychologic manifestations of decompression illness in divers. *Neurol.* 27:125-127, 1977.
- Rozsahegyi, I. Late consequences of the neurological forms of decompression sickness. *Br. J. Ind. Med.* 16:311-317, 1959.
- Rozsahegyi, I. and B. Roth. Participation of the central nervous system in decompression. *Ind. Med. Surg.* 35:101-110, 1966.
- Sillery, R.J. Decompression sickness. A review of the literature and previously unreported histological observations. *Arch. Pathol.* 66:241-246, 1958.
- Smith, M.C. The use of Marchi staining in the later stages of human tract degeneration. *J. Neurol. Neurosurg. Psychiatry* 14:222-225, 1951.
- Smith, M. Observation on the extended use of the Marchi methods. *J. Neurol. Neurosurg. Psychiatry* 19:67-73, 1956.
- Swank, R.I. and H.A. Davenport. Marchi's staining method. Studies of some of the underlying mechanisms involved. *Stain Tech.* 9:11-19, 1934.
- Takagi, M. and Y. Mano. Acute decompression sickness — report of an autopsy case. *Bull. Tokoyo Medical and Dental Univ.* 29:71-76, 1982.

- Vaernes, R.J. and G. Edisvik. Central nervous dysfunction after near-miss accidents in diving. *Aviat. Space Environ. Med.* 53:803-807, 1982.
- Van Rensslaer, H. The pathology of caisson disease. *Medical Record* 40:141-182, 1891.
- Wilkes, M.D., A.C. Palmer, P.C. Pearce, N.P. Luff, M.J. Halsey, and I.M. Calder. Cochlea degeneration — minipigs after repeated hyperbaric exposure. In press.
- Williamson, A.M., B. Clarke, and C. Edmonds. Neurobehavioral effects of professional abalone diving. *Br. J. Ind. Med.* 44:459-466, 1987.

11

The Alvin Program

Barrie B. Walden

| | |
|--|-----|
| I. Introduction | 181 |
| II. History | 182 |
| A. The Early Years | 182 |
| B. Support Ship R/V Lulu | 182 |
| III. Accomplishments | 183 |
| A. Recovery of a Hydrogen Bomb | 183 |
| B. Research Applications | 183 |
| C. International Cooperation: The Mid-Atlantic Ridge | 184 |
| D. Increasing Capabilities | 185 |
| IV. Flagship R/V Atlantis II | 186 |
| V. The Future | 189 |

I. INTRODUCTION

The Alvin submersible will be twenty five years old in June, 1989. Throughout its history, Alvin's principal activity has been to provide support for scientific investigations of the deep ocean. Both the submersible and management structure that maintains and operates it have been in a constant process of evolution.

I suspect the Alvin program is much like many others which exist to provide a service; the objective is to do as much as possible for an acceptable cost. This is different than minimum cost since the latter provides a well-defined goal while the meaning of "acceptable cost" is elusive and constantly varying. Throughout its twenty five years, the Alvin Program has remained close to the point at which its service to the scientific community is no longer worth the dollars required. Alvin's success is at least partially attributable to a conscious attempt to insure that the submersible's capabilities continued to advance in step with rising costs.

II. HISTORY

A. The Early Years

Alvin was designed in the early sixties and delivered to Woods Hole in June of 1964. Its construction was funded by the Office of Naval Research (ONR) in the belief that it would be a useful scientific tool and, more importantly, that its operation would provide valuable knowledge in the area of deep sea engineering and technology. It is hard to imagine how the latter would not be true; practically every aspect of the submersible's design had new, innovative and untested components. Even if much of it did not function as intended, lessons would be learned from the mistakes.

The usefulness to the scientific community was not a sure thing however. First, technological lessons learned from mistakes are not of real interest to an oceanographer, particularly when they result in cancellation of the dives necessary to his research. Also, the submersible's designers had little to go on when deciding what research capabilities should be included. Fortunately, the initial users were also inexperienced and many of the deficiencies that existed were overlooked. The fact is that the race to remain ahead of the scientific requirements began on the first cruise but luckily, the scientists got off to a slow start.

As you might expect, most of the dives made during the early years were intended to test the submersible's performance, safety and reliability. In the first year, the deepest of the 77 dives was to a depth of 65 feet. This was at least partially due to the fact that there was no support ship available for Alvin, and therefore all diving operations were conducted from the Wood Hole dock. It was obvious that this severely limited potential science programs, but a major ship procurement effort was not justified by the submersible's performance to date. Instead, a compromise was reached in which funding was provided to obtain a limited support capability allowing slightly better than "day trip" diving operations close to land.

B. Support Ship R/V Lulu

The support ship R/V Lulu was constructed by a highly respected local contractor based upon a quotation submitted on the back of a shingle torn from the side of the Alvin Operations building. Two surplus mine sweeping pontoons were used as the starting point for a one hundred and five foot long by forty-five foot wide catamaran having an elevator launch and recovery system in the center well. The intent was to utilize this support platform only long enough to convince the funding agencies that

Alvin had a future, and therefore, an improved support capability was justified.

In April of 1985, a Navy tug towed Lulu with Alvin aboard to Port Canaveral, Florida for deep water trials. Twenty nine dives were made, the deepest being a tethered, unmanned dive to depth of 7500 feet. The dedication of the Group's personnel was tested during this period as many problems arose and were overcome despite sub-normal living conditions aboard the support ship. The year ended with the submersible's return to Woods Hole for an inspection and repair period.

III. ACCOMPLISHMENTS

A. Recovery of a Hydrogen Bomb

Early in 1966 Alvin was called upon to assist in the recovery of a hydrogen bomb accidentally dropped as the result of a mid-air collision over the coast of Spain. This was the first useful application for the manned vehicle, and it proved to be a challenging operation which pushed the capabilities of the equipment and personnel to their limits. The search was successful; the bomb was recovered and Alvin justifiably earned the respect of the operational Navy, at least temporarily.

B. Research Applications

It was not until 1967 that an appreciable number of dives were made in support of scientific research. The initial science dives were uncomplicated consisting primarily of observations made through the viewports and the collection of rocks and animals with the single manipulator. As time progressed, simple tools were designed for use with the manipulator and experiments were conducted which involved the submersible deploying and manipulating increasingly complex apparatus. Throughout this period the Alvin engineering and operations teams worked at improving the submersible's reliability and assisted in the development of special science equipment. The Office of Naval Research was still paying a large percentage of the operating costs, and few, if any scientists were risking their careers on Alvin's performance. Nevertheless, useful work was being done and the price was right.

By the early seventies, the Office of Naval Research believed that they had learned enough from Alvin to warrant building two similar submersibles for Naval applications. These were to be the DSV's Seaciff and Turtle and their design was based upon that of Alvin but with the known problems corrected. Unfortunately, their construction and eventual operation

by the Navy led to an abrupt change in Alvin's funding picture. Suddenly, the service Alvin provided no longer included obtaining technical experience of value to the Navy since they believed it would be obtained from the operation of their own vehicles. The Office of Naval Research withdrew Alvin's block funding and requested that the science community pay all costs for the dives they received.

C. International Cooperation: The Mid-Atlantic Ridge

As always, the Alvin Group was close to the line in the areas of the cost to benefit ratio, remaining on the plus side only because of the value of the operational experience which was accumulating. The submersible had not proven itself to the scientific community to the extent required to allow 100 percent science support. As a result, the loss of Navy block funding caused the Alvin Program to come precariously close to ending. Salvation came in the form of a planned international co-operative scientific investigation of the Mid-Atlantic Ridge, a mountain range extending north and south down the middle of the Atlantic Ocean.

French and American scientists were extremely interested in making first hand observations of the Mid-Atlantic Ridge in the hope that they would provide answers to fundamental questions relating to the theory of plate tectonics. Both countries were to provide submersibles for the research expedition, and, except for one problem, Alvin was the obvious choice for the United States. The problem was that Alvin was rated to operate at a maximum depth of 6000 feet and the Mid-Atlantic Ridge dives were to be to a depth in excess of 9000 feet.

Fortunately, at about the same time, the Navy had become interested in obtaining some practical experience in the fabrication of large objects made from titanium. The construction of a new pressure hull for Alvin suited the purpose, and, if done successfully, would allow an operating depth increase to 12,000 feet, more than enough for the Mid-Atlantic ridge.

If the hull construction had not succeeded and the expedition to the Mid-Atlantic Ridge had not been an outstanding success, this paper would be considerably shorter or non-existent. Instead, Alvin's depth capability doubled and its potential value to the science community had been demonstrated. Ten years had passed since the delivery of the vehicle, and throughout this time, the submersible and its support ship had undergone a continuous process of development. Alvin's second ten years began with the understanding that the future lay in support of science.

D. Increasing Capabilities

The decade beginning in 1975 was tremendously exciting for researchers interested in the deep ocean. It included the discovery of warm water vents, their more robust black smoker relatives, and the unsuspected biological communities associated with each of them. Alvin was in the forefront of many of these discoveries and was frequently the tool of choice for follow-up investigations. The Alvin engineering staff and operators worked to increase the submersible's utility by improving its reliability and capability.

During this period, the design and performance of Alvin's hull was reviewed resulting in an increase in the certified operating depth from 12,000 feet to 13,124 feet, or 4000 meters (Fig. 1). The aluminum frame was replaced with one made from titanium providing greatly increased corrosion resistance. The submersible's length was increased by two and one half feet to provide room for a fourth battery compartment. Perhaps most importantly, Alvin was designated as a National Oceanographic Research Facility and the National Science Foundation, the Office of Naval Research and the National Oceanic and Atmospheric Administration developed and signed the Alvin Joint Founders Agreement. With this renewable agreement, the joint funders pledged financial support for the Alvin program for periods of three years thus providing a degree of stability missing since the removal of ONR block funding.

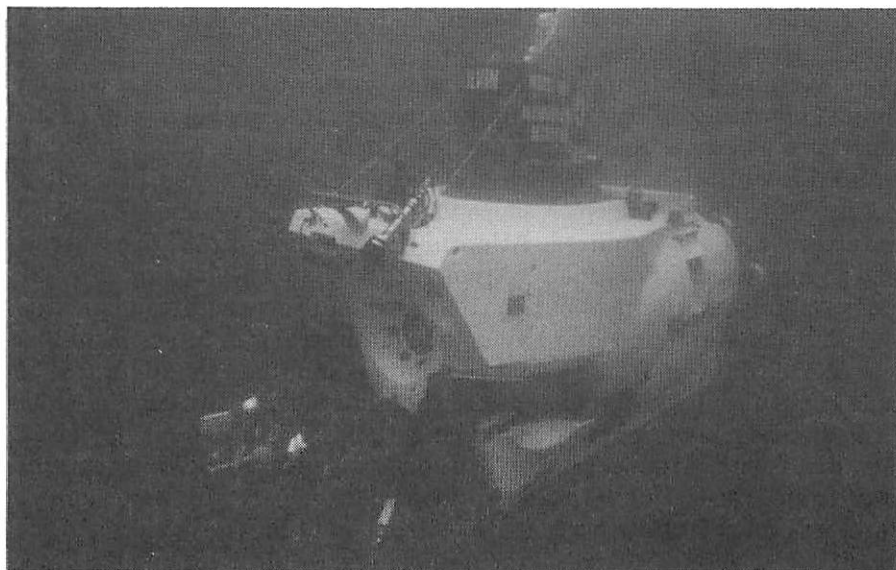


Fig. 1. DSV Alvin beginning 13,000 foot dive for scientific research.

As time progressed, the sophistication of the research equipment intended for use with Alvin increased greatly and this necessitated continuous development of the submersible. The progression was far from smooth; frequently the necessary equipment and capabilities could not be brought together for testing until the start of the cruise, leaving little time for problem solving. The proven development process of design, construct, test and modify was often short-circuited because thorough testing required the submersible which was not always available. Diving programs were conducted at greater distances from Woods Hole thus increasing the difficulty in providing engineering support for operations.

Many solutions to the problems existed; all would result in increased costs. Although Alvin was doing well, the signs indicated that a major increase in operational expenses would not be acceptable without an accompanying highly visible increase in capability.

IV. FLAGSHIP R/V ATLANTIS II

By the early eighties, the demand for Alvin dives greatly exceeded the time available. The slow speed and small size of the support ship R/V Lulu frequently limited cruise dive time, the number of investigators and their activities. In addition, research programs in the planning stages would require expeditions distant enough from the United States to make the use of Lulu questionable for safety and reliability reasons. The time was right for a new support vessel, and Woods Hole flag ship, R/V Atlantis II was available.

Considerable effort would be required to provide the Atlantis II with an Alvin support capability. The ship was not new, having been built at the same time as Alvin, and it was due for a mid-life refit. Additionally, it required installation of a launch and recovery system and all the other necessary facilities from work shops to tracking sonar systems. An Alvin conversion was also required; Atlantis II would use an "A" frame overhead single point lift launch system rather than an elevator (Fig. 2). Unfortunately, Alvin had not been designed for overhead lifting and had no points above the mid-line having adequate strength. Major modifications to the titanium frame would be required and these would need to be made without substantial changes to the remainder of the submersible.

The necessary work was done during 1983, and in January of 1984, Atlantis II with Alvin aboard sailed for two years of science programs away from Woods Hole. The operational pace aboard the new support vessel was considerably increased above that on Lulu. The doubling of the transit speed greatly increased the number of possible dives and the bunk space available for scientists jumped from eight to nineteen. Atlantis II



Fig. 2. Recovery of DSV Alvin aboard its support ship Atlantis II.

was equipped with a complete set of standard oceanographic tools which allowed research by more conventional means to be conducted in conjunction with Alvin dives. The learning curve for all involved was particularly steep, but the demands associated with the step increase in operational costs did not allow a leisurely climb.

Generally, the operations proceeded smoothly and the science programs were successful. Lessons were learned, however, which indicated that Alvin needed some major modifications before it would be ready for some of the work planned for the western Pacific. Reliability issues developed from the fact that the previously available maintenance time associated with Lulu's slow transits was now diving time. Failures related to the number of dives became more of a problem as their frequency increased in proportion to the extended diving schedule. The operational manpower was fully occupied with the effort of daily operations and began to lose sight of the fact that they were also the second half of a capability development team. This latter problem manifested itself in the inability of the Alvin Group as a whole to accomplish the final small percentage of the work required to make a project successful. The same complaints were received cruise after cruise while the Operations Group and the Engineering Staff pointed accusing fingers at each other. Fortunately, progress was made despite the problems, and a major submersible overhaul was scheduled prior to the western Pacific expedition.

The 1985-1986 overhaul resulted in numerous improvements and eliminated many of the reliability problems. Alvin received a new propulsion system, battery system, hydraulic system, pressure compensation system, emergency release systems, personnel sphere interior, manipulator, and variable ballast pump motor (Fig. 3). Smaller changes were made in other areas and in total, the reliability and capability were vastly improved. On the first of May, 1986, the Atlantis II sailed for the longest continuous operational period in Alvin's history. By December of 1988, Alvin will have made 485 dives since its last major overhaul,

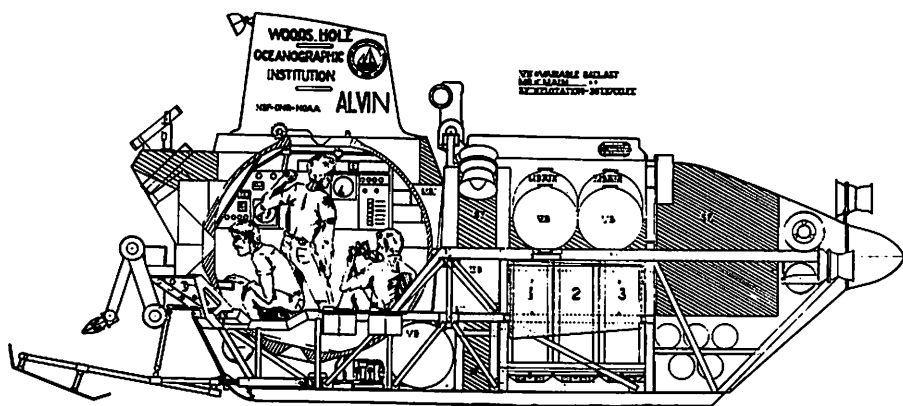


Fig. 3. Inboard profile of the DSV Alvin operated by the Woods Hole Oceanographic Institution.

doubling the previous maximum. It will have traveled from Woods Hole to Tokyo and back in support of science programs for 72 principal investigators. At the time of this writing, with five scheduled dives remaining, only ten dives have been lost due to any cause.

IV. THE FUTURE

This is a remarkable record and yet the future is not assured. Alvin funding does not depend upon past performance but upon present demand and, as always, the cost to performance ratio. It would be a grave error to believe that Alvin's present valued position as a principal tool for deep ocean research is enough to carry forward an overly expensive or operationally faulty program. The Alvin Group will enter its twenty fifth year with the knowledge that the scientific expectations will soon exceed its capabilities and that the race continues.

the first of these is the fact that the majority of the population of the United States is of European descent. This is a fact which has been recognized by the majority of the people of the United States for many years. It is a fact which has been recognized by the majority of the people of the United States for many years.

It is a fact which has been recognized by the majority of the people of the United States for many years. It is a fact which has been recognized by the majority of the people of the United States for many years. It is a fact which has been recognized by the majority of the people of the United States for many years. It is a fact which has been recognized by the majority of the people of the United States for many years. It is a fact which has been recognized by the majority of the people of the United States for many years.

12

Biological Explorations in the Mid-Ocean Realm: Food Webs, Particle Flux, and Technological Advancements

*Marsh J. Youngbluth, Thomas G. Bailey
and Charles A. Jacoby*

| | |
|--|-----|
| I. Introduction | 191 |
| II. Species Diversity Discovery | 193 |
| III. Vertical Distribution Patterns | 193 |
| IV. Particle Flux | 194 |
| A. Fecal Pellet Transport and Zooplankton Behavior | 195 |
| B. Marine Snow Aggregates and Gelatinous Plankton | 196 |
| V. Metabolism of Pelagic Fauna | 198 |
| VI. Submersible as a Mobile Laboratory | 198 |
| VII. Some Recommendations | 201 |
| VIII. Facilities, Programs, and Projects | 202 |
| IX. Summary and Conclusions | 203 |
| Acknowledgements | 204 |
| References | 204 |

I. INTRODUCTION

The ocean realm covers 99% of this planet by volume; therefore, it is not surprising that models developed to predict cycles of biological production lack rigor (Childress, 1983). Part of the uncertainty stems from the available base of oceanographic data. For decades, biologists have relied on collections made with plankton nets and water bottles to obtain fundamental insights about the behavior, distribution and abundance of

pelagic organisms. Information gained from this ship-based methodology has been important, but it lacks the dimension of direct observation (Hamner, 1977; Hamner *et al.*, 1987). Submersibles overcome this constraint by providing *in situ* opportunities to distinguish where fauna are concentrated and how they interact with their environment, their prey or their mates.

Both manned and remotely operated vehicles have been used to conduct undersea investigations in the water column and near the sea floor (Allmendinger, 1982). The history and hardware of manned submersibles are well reviewed in an exhaustive handbook (Busby, 1976). Deep-sea craft under development have been described in other publications (Earle, 1986; Takagawa, 1987). Commercial news magazines, *Sea Technology*, *Subnotes*, *Underwater Systems Design*, highlight the activities of submersibles and the availability of undersea accessory equipment. *In Situ News*, a quarterly pamphlet issued by the University of Rhode Island, serves to summarize recent scientific accomplishments, describe new tools, and announce upcoming meetings.

Undersea vehicles used for scientific purposes today vary considerably in their complexity and capabilities (Hanson and Earle, 1987; Rechnitzer, 1986). They range from special purpose craft that conduct photographic surveys or perform retrieval tasks to multi-purpose submersibles that utilize state-of-the-art instruments (Tietze and Clark, 1986; Tusting, 1986; Youngbluth, 1984). In the last decade, manned submersibles working in water column environments have enhanced substantially the ability to observe, capture and experiment with pelagic fauna at all depths of the ocean (Alldredge *et al.*, 1984; Barnes *et al.*, 1976; Smith and Laver, 1981; Youngbluth *et al.*, 1988). One of the most important contributions has been the ability to examine critical trophic and behavioral relationships among pelagic fauna on the appropriate spatial scales (cm to m) (Harbison, 1987; Janssen *et al.*, 1987; Mackie and Mills, 1983; Mills, 1987). These accomplishments signal that continued use of submersibles and the technically advanced instruments they carry, will help oceanographers decide where and when to perform measurements and conduct experiments.

This paper provides examples of how deep-diving, manned submersibles facilitate investigations of zooplankton at mid-ocean depths. Gelatinous zooplankton are featured in some detail because they represent a group of ecologically important animals that are sampled poorly by other methods (Alldredge, 1984). Most of the information presented has been obtained with the JOHNSON-SEA-LINK submersibles. Ongoing projects in water column environments are reviewed and topics as yet unstudied are suggested.

II. SPECIES DIVERSITY DISCOVERIES

The limited, but exciting water column work with manned submersibles has demonstrated clearly that more kinds of gelatinous zooplankton exist in the deep-sea than recognized previously. Some of the most conspicuous midwater animals are carnivorous ctenophores, medusae and siphonophores. New species of ctenophores have been recognized (Madin and Harbison, 1978a; Madin and Harbison, 1978b; Mills, 1987) and more than a dozen other specimens, which have been photographed and collected at depths ranging to 1000 m, are undescribed (Harbison, 1985; Harbison pers. comm.). Many of these comb-jellies are often relatively numerous ($>1\text{ m}^{-1}$) and one unnamed, crimson cydippid-like species makes daily vertical migrations of at least 300 m in the canyons south of Georges Bank.

Mills and colleagues (Mills *et al.*, 1987) have classified a new species of coronate scyphomedusa using specimens captured from a submersible. Larson and colleagues (Larson, *et. al.*, in press) have captured and described a large (60 cm bell diameter), unusual ulmarid scyphomedusa. Another ten undescribed species of mesopelagic hydromedusae have been collected (C. Mills, R. Larson, pers. comm.).

At least six new species of siphonophores are known to exist under tropical seas (Pugh and Harbison, 1987; Pugh and Youngbluth, 1988a; Pugh and Youngbluth, 1988b). Several other unusual and infrequently collected physonect siphonophores have not yet been described (Pugh, pers. comm.) In addition to these taxonomic studies, the morphology and behavior of a physonect species *Lycnagalma utricularia*, have been noted in some detail (Pugh and Harbison, 1986). This study represents one example of the fact that many animals, previously considered quite rare based on net tow data, are in fact commonly encountered on submersible dives to mesopelagic depths.

Several new or poorly documented deep-sea appendicularians in the genera *Oikopleura*, *Pelagopleura* and *Fritillaria* have been collected and are being described (R. Fenaux, pers. comm.). Furthermore, it is likely that the genus *Bathochordaeus* will be revised on the basis of new specimens obtained with submersibles. One conclusion to be drawn from studies of gelatinous zooplankton is that the composition of food webs in midwater environments is still being defined.

III. VERTICAL DISTRIBUTION PATTERNS

In situ observations have also shown that many zooplankton live within certain depth zones; zones much narrower than have routinely sampled by tow nets (Mackie, 1985). For example, prior to 1981, the giant

appendicularian *Bathochordaes charon* was known from less than 20 specimens collected over a period of 80 years (Galt, 1979). All samples had been obtained from continuously open nets and consequently the vertical distribution of this species was not well documented. Since 1981, several specimens of this appendicularian have been observed and collected *in situ*, some near 400 m but mostly from 45-65 m within the Gulf Stream (Youngbluth, 1984). The presence of numerous *B. Charon* (up to 9-m^{-1}) in their 30-cm diameter, mucoid filter-houses, has always coincided with a strong pycnocline and a high standing biomass of chlorophyll and detritus. Examinations of particles found in the guts of this appendicularian have revealed biogenic debris from phytoplankton and crustaceans.

In situ work with the one-person vehicle WASP off southern California showed that stage V copepodites of *Calanus pacificus californicus* become torpid and overwinter in 20-m bands extending for kilometers at a depth of 450 m during non-upwelling periods. Presumably, this is a response to low food availability (Alldredge *et al.*, 1984). Densities of these diapausing copepods averaged ca. 10^7 individuals $\cdot\text{m}^{-3}$, about three orders of magnitude higher than abundance estimates made using nets in surface waters. Both of these distribution patterns indicate that there is more biological structure in the pelagic environment than suspected from remote sampling.

IV. PARTICLE FLUX

Particulate matter is ubiquitous in the oceans. One of the major objectives in oceanographic sciences has been to chronicle and quantify processes that regulate the distribution and sedimentation of biogenic particles in the sea (Platt and Sathyendranath, 1988). Of particular interest, in terms of mechanisms and rates, is the relationship between primary production in the euphotic zone and the downward flux of surface-derived, organic detritus to the deep sea. Large particles, such as the fecal pellets and marine snow aggregates produced by zooplankton, are principal vectors for vertical transport (Knauer *et al.*, 1984). Fecal pellets can sink rapidly ($10\text{-}1000\text{ m}\cdot\text{d}^{-1}$) (Fowler and Knauer, 1986). Marine snow aggregates fall more slowly ($10\text{-}150\text{ m}\cdot\text{d}^{-1}$) (Alldredge and Gotschalk, 1988). Variations in settling rates are related to a host of physical and biological factors, e.g, advection, viscosity, diet and decomposition. As a result, mathematical models developed to predict regional and global flux rates of biogenic material require detailed information about production and distribution of these important particles (Hargrave, 1985).

Most existing data about particle transport events have been based on *in situ* sampling with sediment traps, pump-supported equipment, optical

techniques or scuba (Alldredge *et al.*, 1987; Bishop *et al.*, 1985; Lampitt, 1985; Uerrey and Knauer, 1981). Data obtained from sediment traps and optical devices can be misleading in that the amount of material measured may be a mixture of particles transported by vertical sinking, lateral advection and periodic resuspension. Similarly, information derived from filtration systems may include both suspended and fast-sinking particles. Scuba assessment of the sizes, sources and sinking of particulate material are depth limited. Direct observation and sampling from submersibles can overcome some of these constraints.

A. Fecal Pellet Transport and Zooplankton Behavior

Quantification of the production, sinking rate, distribution, and abundance of large particles need to be considered in conjunction with feeding and migratory behaviors of zooplankton (Gilmer and Harbison, 1986; Harbison and Gilmer, 1986). Observations and sampling with submersibles have begun to ascertain the depths at which such particles are concentrated. For example, euphausiid fecal pellets can stall at pycnoclines, and this adds to the complexity of predicting sinking rates. Dives made within the Gulf of Maine and the canyons south of Georges Bank revealed that high densities of fecal pellets ($50\text{--}325\text{ particles}\cdot\text{m}^{-3}$) accumulated at night in 5-24 m thick layers coincident with the pycnocline (15-30 m) (Youngbluth *et al.*, 1988). These cylindrical (0.2 mm OD x 3-10 mm long) particles sank rapidly (ca. $200\text{ m}\cdot\text{d}^{-1}$) and could transport substantial amounts of organic matter ($7\text{--}12\text{ mg C}\cdot\text{m}^{-2}\cdot\text{d}^{-1}$) to the bottom. Vertically migrating euphausiids *Meganyctiphanes norvegica* produced the pellets. These individuals represented only part of enormous aggregations (up to $10^4\text{ individuals}\cdot\text{m}^{-3}$) of adults (25-35 mm long) that remained within 10 m of the seabed day and night and appeared to forage in the benthic boundary region (Greene *et al.*, 1988; Youngbluth *et al.*, 1988). Direct observations of *M. norvegica* near the sea floor also support scattered, anecdotal data from trawls and submersibles (Bigelow, 1928; Mauchline, 1980; Peres *et al.*, 1957; Tregouboff, 1961) which have suggested that epibenthic aggregation and near-bottom feeding by this species occurs in nearshore waters from the western North Atlantic to Mediterranean seas. If consumption of biogenic detritus in the epibenthic region is common among these euphausiids, this species may account for considerable repackaging and bioturbation. Assuming that epibenthic populations feed more or less continuously, those individuals that migrate vertically each night may introduce recycled biogenic and inorganic materials back into the mixed layer when they release fecal pellets. These discoveries are significant because they demonstrate the extent to which environmental factors and zooplankton behaviors can interact to influence the rate and amount of particle transport.

Fecal pellets and carcasses of gelatinous zooplankton called salps have also been implicated as significant sources of particle flux. An occasionally cited, but as yet unquantified observation, was made several years ago from the ALVIN submersible. Cacchione and colleagues (1978) reported that "windrows" of salp bodies were numerous at ca. 3000 m along the sea floor of Hudson Canyon. Presumably these aggregations were formed by *Salpa aspera*, a diel-migrating, midwater species known to attain very high densities (Wiebe *et al.*, 1979). Knowing also that salps can grow and reproduce rapidly and that they can produce fast-sinking fecal pellets (Madin, 1982) suggests that this species must contribute substantially in several ways to the export of particles from the photic zone.

B. Marine Snow Aggregates and Gelatinous Plankton.

The term marine snow has been applied to describe large (1 mm to 1.5 m in diameter or length), aggregated mucoid particles, many of which are formed by gelatinous zooplankton to collect small (down to 0.1 μm) particulate food. Microscopic and chemical examinations of marine snow have indicated that aggregates serve as important sites of nutrient regeneration by microbes, primary production by autotrophs, and phagotrophic growth by microflagellates (Alldredge and Cox, 1982; Caron *et al.*, 1982; Knauer *et al.*, 1982). This information prompted oceanographers to propose that microbe-enriched aggregates constitute a major contribution to the primary level of oceanic food webs. Furthermore, these aggregates may flux within the water column or sink to the sea floor (Asper, 1986).

Because marine snow aggregates are extremely fragile and easily disintegrated, the only successful means of assessing the abundance and obtaining recognizable samples of marine snow aggregates has been with *in situ* methods, either by scuba (Alldredge *et al.*, 1986; Shanks and Trent, 1980) or submersibles (Alldredge and Youngbluth, 1985; Barham, 1979; Silver and Alldredge, 1981). Ecological investigations, which relate marine snow aggregates to the gelatinous zooplankton that produce the aggregates, are rare (Davoll and Silver, 1986). Field observations from submersibles at midwater depths have identified at least three recognizable sources for globular, sheet-like, and string-shaped aggregates. For example, many mucoid globs and sheets, 1-30 cm in greatest dimension, are the discarded filter-houses produced by appendicularians. Masses of these ruptured aggregates typically occur just below the subsurface chlorophyll maximum layer of the Gulf Stream, from 50 to 100 m. Depending on the prevailing currents, these masses can intrude onto the continental shelf of the South Atlantic Bight within plumes of upwelled water. Microbial activity associated with appendicularian filter-houses produced below 200 m may act to recycle biogenic particles in this region of

the water column (Davoll and Youngbluth, 1984). Further investigations of these zooplankton are needed to improve predictions about particle enrichment and flux.

Dense concentrations of mucoid material in the form of strings, 3-90 cm long, have appeared at depths of 90-120 m in the Bahamas. These strings have also been noted to occur from 5-600 m. Most strings were oriented vertically and many remained attached to the filamentous rhizopodia of the foraminiferan *Hastigerina pelagica* (Youngbluth, 1984). Estimates of foraminiferan abundance ranged from 5-100 individuals·m⁻³.

Mucous feeding webs of pseudothecosome pteropods (Lalli and Gilmer, 1989), 5 to 80 cm in diameter, can be numerous at depths of 90-120 m in the Bahamas. The rates at which these strings and sheets are produced, sink, and decompose are unknown.

These records of appendicularians, foraminiferans and pteropods are but a few of many observations which indicate that gelatinous zooplankton can produce masses of marine snow. The biological contribution of the particulate material to pelagic and benthic communities is a subject of ongoing investigations.

Recent observations of gelatinous phytoplankton provide yet another example that relates to particle transport. During a series of dives made along the coast of Florida in the summer of 1987, gelatinous colonies (0.5-3 mm in diameter and 5-100 mm in length) of the diatom *Thalassiosira subtilis*, reduced visibility from the submersible to less than 1 m at depths ranging from the base of the thermocline (ca. 5 m) to the sea floor (30-80 m). The dense clusters of colonies (up to 300/liter with dry weight biomass reaching 2 mg·liter⁻¹) appeared only in the nutrient-rich, subsurface waters of the Gulf Stream that upwelled over the continental shelf. In a 10-day period these mucoid algal colonies began to decompose, forming globular and comet-shaped aggregates. Preliminary calculations, based on the vertical distribution and standing stock of the colonies and on the area covered by the upwelling event (1.3×10^3 km²), indicated a biomass of ca. 10⁷ kg C. Within a two-day period, a shift in wind direction deepened the mixed layer and forced the intrusion off the shelf into the Gulf Stream. This regional, but nonetheless large-scale mass exchange between coastal waters and the open ocean must affect the productivity of the open ocean (Rudakov, 1987). *Thalassiosira* spp. are known to occur in upwelled waters around the world (Elbrachter and Boje, 1978; Jimenez, 1981). On a global scale, the magnitude of organic flux associated with the growth of these colonial diatom species will elude satellite reconnaissance because the bulk of primary production occurs deeper than such remote sensing can detect.

V. METABOLISM OF PELAGIC FAUNA

A fundamental requirement for comprehending the dynamic nature of food webs in oceanic regimes is quantification of energy use by major faunal components. These rates can be estimated from measurements of metabolism, since this process generally accounts for the largest fraction of an animal's energy use (Childress, 1977). To date, physiological studies of gelatinous zooplankton taken from mesopelagic environments are rare principally because these fragile animals are difficult to collect and maintain with conventional techniques. The first measurements of oxygen consumption by the midwater ctenophore *Bathocyroe fosteri* (Youngbluth *et al.*, 1988), have revealed rates about half those of shallow water ctenophores (Kremer *et al.*, 1986). However, the accuracy of these results remains unknown. Individuals were collected carefully in special chambers from a submersible but metabolic data were recorded in a shipboard laboratory. Stresses associated with removing an animal from its natural habitat were unquantified and argue for developing an *in situ* approach: e.g., change in pressure and temperature, exposure to light, and transfer from collecting to experimental chambers. Some experiments of this type have been conducted with free-vehicles placed in bathypelagic environments using the manned submersible ALVIN (Smith and Baldwin, 1983).

VI. SUBMERSIBLE AS A MOBILE LABORATORY

Behavior of captive animals is abnormal, and their survival under laboratory conditions is usually limited. However, although nature cannot be duplicated in a laboratory, it is possible to take laboratory tools into the field to facilitate investigations. In this context, significant progress in pelagic research will depend in part on the extent direct visual observations from submersibles can be enhanced with recording devices.

One important trend for future research with manned submersibles, and to an extent remotely operated and autonomous underwater vehicles, will be to expand and extend the quality and range of perception. Human vision is the primary sensor for information in many undersea studies. Records of what can be seen have been made primarily with high definition still and video cameras (Miller and Pawson, in press; Youngbluth, 1984). To date, most images are still collected under bright, white light. Optical equipment of the future will take advantage of low light, filtered light, laser light and stereoscopic technology to provide more realistic and unobtrusive assessments of animal behavior as well as particle abundance and morphology.

The newest color CCD cameras, have high line resolution (up to 700 lines horizontal) and lack ghosting image distortion. The latter quality is essential for critical examinations of shape and movement. Some new video recorders are capable of 400-500 lines, about double the usual limit of playback quality. Experimental stereo-imaging video systems, capable of tracking complex swimming and feeding movements of relatively large zooplankton (>3 cm), are undergoing field trials in an effort to provide a basis for analyzing the adaptive importance and energetic costs of specific behaviors (Hamner *et al.*, in press; Price *et al.*, 1987). A laser-based video system with high resolution (ca. $15\text{ }\mu\text{m}$) is being tested (R. Strickler and G. Paffenhofer, pers. comm.). Optical gear that can record particle size, density and movement over small (mm to cm) distances (e.g. laser holography, Betzer *et al.*, 1987; camera/transmissometer profiler, Gardner *et al.*, 1987), if adapted to submersibles, would also help to define how the behaviors of zooplankton effect the cycling of organic matter at physical chemical interfaces.

If currently available electronic image intensifiers can be improved and used in concert with low-light SIT and ISIT cameras (Wood and Potts, 1987), observations and measurements could be made at ambient, dim-light conditions within the photic zone. This practice would reduce unnatural phototactic responses associated with using incandescent light in the deep sea and could document a wide variety of normal swimming, feeding and breeding behaviors. ISIT video recordings made in midwater from the submersible DEEP ROVER have shown that bioluminescent displays produced by animals *in situ* differed considerably from displays emitted by captive individuals (Widder *et al.*, in press). This result clearly illustrates that measurements and experiments must be conducted *in situ* to obtain unbiased understanding of the communication value of these signals between and among species. In the latter and previous studies, bioluminescent records indicated that a large proportion of such emissions were produced by organisms smaller than 0.5 mm. Identification of these organisms awaits development of imaging equipment with better magnification and resolution.

Manned submersible diving involves being isolated from a high pressure environment inside a capsule. There is no smell, no taste, no sound from the environment. In an effort to compensate for the absence of such information, environmental data, such as depth, temperature, conductivity, and turbidity can be recorded automatically by a sensor and data logger package (Voyles and Clayton, 1986). Two new sensors have recently been added to the JOHNSON-SEA-LINK submersible system: (1) a SeaTech fluorescence sensor, capable of measuring chlorophyll *a* concentrations ranging from $0.05\text{-}50\text{ mg}\cdot\text{m}^{-3}$, and (2) a novel radiometer

(F. Caimi pers. comm.), that can measure photosynthetically active flux to 500 m. The radiometer works within a spectral range of 400-700 nm and provides nearly instantaneous readings over seven decades of illumination ($.003$ to $3000 \mu\text{E}\cdot\text{s}^{-1}\cdot\text{m}^{-2}$) with spatial integration over nearly a 2π -steradian field. Chemical nutrients ($\text{NO}_3\text{-N}$, $\text{SO}_4\text{-S}$, SiO_4 , $\text{PO}_4\text{-P}$) have been measured from manned submersibles by modified flow injection analysis (Johnson *et al.*, 1986). Access to some or all of these environmental parameters during the course of a dive and the period between consecutive dives is often crucial for planning sampling strategies and conducting *in situ* experiments on a given cruise.

Both the panoramic range and the distance component of visibility are limited from submersibles in direct proportion to the absorption of light in seawater. Another way to interrogate the surrounding environment is with acoustics. Sonar systems can scan large volumes of water in a short period of time. For example, high frequency (420 kHz to 1 MHz) dual-beam acoustical equipment holds promise for obtaining quantitative data on the size and abundance of zooplankton. These data can complement visual records of distribution and migration patterns of individuals and populations (Jefferts *et al.*, 1987) and extend them up to 60 m from the submersible (Green *et al.*, in press). This acoustic technique mitigates two important sampling problems: first, biases associated with animal avoidance of sampling gear (in this case a submersible) and, second, labor intensive enumeration of animals in different size classes. The Japanese are reported to be working on a sonar system that will display video images of targets within a range of 200 m (Tagawa, 1987).

Near real-time detection of zooplankton distribution patterns should clarify how pelagic animals select patchy food resources. In this context, pre-dive shipboard surveys of current shear zones, which can influence the distribution of microzooplankton (Townsend and Cammen, 1985), may be possible with an acoustic doppler profiler (Flagg, 1987).

As the use of *in situ* incubation equipment becomes routine, highly versatile and dextrous manipulator arms will improve the efficiency of deep-sea investigations. In this respect, two features of existing arms deserve special mention. A tool interchange mechanism has been designed to accommodate flexible jaws, clam-shell grab or a cutting blade (C. Tietze, pers. comm.). The advantages of this mechanism include: 1) the ability to index a tool in any of 360 degrees axially, 2) the capability of holding a tool whether or not power is maintained to the arm, and 3) the versatility of using hydraulic power or air pressure to exchange tools. Acoustical signals incorporated into at least one manipulator arm (Hawkes, 1984) indicate force and texture. Knowledge of these factors can be critical when grasping instrumented packages.

VII. SOME RECOMMENDATIONS

Significant progress in water column research will depend on contemporaneous use of submersibles, ships and satellites. In order to implement this strategy, manned submersibles must incorporate at least the following features and equipment to be effective for biological investigations at midwater depths:

1. Variable-speed, ultra-quiet thrusters to rapidly and precisely regulate fine-scale movements in horizontal and vertical directions.
2. A variable ballast system to quickly adjust trim and pitch throughout the depth range of the submersible.
3. High resolution still cameras. High definition video cameras (color and low light, both with zoom capability) mounted on pan-and-tilt platforms. Real-time overlay of alpha-numeric data on the video monitor should become standard, e.g., date, depth, time, environmental parameters.
4. Multiple sampling chambers that can capture delicate, slow-moving creatures as well as more robust, fast-swimming zooplankton.
5. Data logging systems (preferably solid-state) for real-time and recorded measurements of environmental parameters, i.e., depth, temperature and salinity (via conductivity), and the option to adapt additional sensors, e.g., turbidity, chlorophyll, light intensity, oxygen and nutrients, as required by research projects.
6. Efficient, long-life battery system or capability of battery change-out to allow a 12-h term of operation at sea per day.

A factor often overlooked, but critically needed, for effective operation of a submersible is a proper support vessel (Tagawa, 1987). The ship should be capable of operating and launching the submersible in unsettled sea conditions. The concept of using ships or platforms of opportunity, while attractive from the perspective of reduced cost and geographic mobility for the submersible, may, in practice, limit the success of a given mission for several reasons. Possible problems include: a crew's lack of experience in handling the submersible, inadequate cranes for launch and retrieval, disturbance of the submersible's acoustical communication/location/data acquisition gear by ship-generated noise, lack of spare parts, a ship's lack of station-keeping and submersible-tracking equipment, and lack of scientific laboratory space. More attention should be given to blending the facilities of a modern oceanographic ship (dynamic positioning system, winches, laboratories, temperature-controlled rooms) and the features of a submersible tender (launching gear, tracking equipment) to maximize the use of expensive seetime.

VIII. FACILITIES, PROGRAMS AND PROJECTS

Presently, few national or international opportunities exist for long-term research with manned submersibles. Excursions into the midwater realm with manned submersibles are still relatively infrequent, principally because of the number of adequate, currently active vehicles is limited (JOHNSON-SEA-LINKS, DEEP ROVER, PISCES IV & V, CYANA, NAUTILE, ALVIN). In the last five years, federal research support has come primarily from the National Oceanic and Atmospheric Administration (NOAA), the National Science Foundation and the Office of Naval Research, along with occasional funding from Minerals Management Service, Department of Energy and Sea Grant. Funding has increased in direct proportion to the reliability and versatility of submersibles. The recent establishment of regional National Undersea Research Centers by NOAA (in Connecticut, North Carolina, St. Croix and Hawaii and the likely creation of at least one more Center on the west coast of the United States) should increase the number of investigators and programs that focus on *in situ* investigations. The Harbor Branch Oceanographic Institution has promoted science from submersibles, both manned and unmanned, since 1971 by fostering interdisciplinary cooperation among scientists, engineers and operators. The Monterey Bay Aquarium Research Institution (MBARI), founded in 1987, has adopted a similar, multi-talented team approach and plans to conduct projects in midwater (B. Robison, pers. comm.). MBARI, however, has chosen to concentrate on the development of an unmanned, deep-diving vehicle (HYSUB ROV). As the goals and objectives of *in situ* pelagic research broaden among the current and future generations of oceanographers in the United States, it is likely that international collaboration will be based on research initiatives with Japan (JAMSTEC, Mayama, 1987) and France (IFREMER, Grandvaux, 1986).

Current multi-investigator programs in the Gulf of Maine, South Atlantic Bight and the Great Lakes exemplify how manned submersibles can serve fundamental roles as technological resources (Babb and DeLuca, 1988). Projects for the future, that will require *in situ* approaches, have been addressed at several workshops. In a meeting devoted to outlining the properties of aggregated particles and the mechanisms responsible for their formation, it was clear that the importance of these particles to organic flux must be based on *in situ* assessments of their distribution, abundance and utilization (Alldredge *et al.*, 1986). At a symposium on zooplankton behavior, it was concluded that answers to major questions concerning the evolution of natural responses to environmental conditions can be best explored by applying novel

optical and acoustic technology *in situ* (Price *et al.*, 1987). Recently, ocean scientists emphasized that optical and acoustical methods (see section on Submersible as a Mobile Laboratory) can produce complementary information about the identity, abundance and distribution of pelagic fauna (Remote Optical and Acoustic Mapping of Ocean Waters, Monterey Bay Aquarium, 20-21, May 1988, B. Robison, pers. comm.). Integration of both methods is a goal for the future and will involve various schemes for the collection, analysis and storage of large data streams.

In an effort to define unsolved problems and chart new directions, studies of marine zooplankton were segregated by time-scale strategies (Marine Zooplankton Colloquium, Lake Arrowhead, 18-22 April 1988, G. Paffenhofer, pers. comm.). One set of recommendations from this colloquium encouraged *in situ* deployments of instruments capable of recording high volumes of data over brief or extended periods to investigate behaviors of individuals and populations. At an earlier conference (22-23 October 1986, Doelling and Harding, 1987), alliances between scientists and engineers from academia and industry were encouraged to facilitate the development of advanced instruments for undersea applications, e.g., robotic tools with artificial intelligence and manipulator devices with tactile sensing systems.

IX. SUMMARY AND CONCLUSIONS

In the last decade, undersea studies with manned submersibles have produced unprecedented records of natural behavior for well-known and undescribed pelagic animals and have shown that many species live in specific depth zones, zones much narrower than have been measured by traditional methods. Such distributional patterns probably reflect preferred feeding areas in many cases and suggest that there is considerably more biological organization in the pelagic ecosystem than recognized previously. These discoveries have indicated new problems to study in the dark, cold and hyperbaric environments that characterize the deep sea.

Over the next decade new discoveries about the behavior and distribution of zooplankton will undoubtedly emanate from submersible-based investigations, principally because of the high potential for coupling direct visual observations of behavior (individuals and populations) with simultaneous measurements of environmental variables. Fragile, gelatinous zooplankton, in particular, require more *in situ* studies to determine their roles in processes associated with biogeochemical cycling of particulate matter.

The conclusion to be drawn from this review is that undersea investigations with manned submersibles need to be an integral part of pelagic

research programs, not a separate component. Submersibles have investigated phenomena on temporal and spatial scales not addressable with remote sampling from ships or satellites. Continued use of these vehicles will help oceanographers to optimize where and when to perform ecological investigations in water column environments. Problems of broad significance can be addressed if target organisms in specific geographical regions are selected carefully and a repertoire of physical, chemical and biological factors are studied concurrently.

ACKNOWLEDGEMENTS

This work was supported in part by grants from the National Undersea Research Programs, NOAA/OUR, NOAA/UCAP, NOAA/HURL, from the National Science Foundation OCE-8600278, and from the North Atlantic Treaty Association 0262/88. R. Cooper and E. Finkle generously shared information about research submersibles and program development. We appreciate assistance at sea from ship and submersible crews aboard the R/V SEWARD JOHNSON, R/V JOHNSON, R/V SEA DIVER and R/V KILA. C. Tietze and M. Young provided unflagging mechanical engineering expertise. Contribution No. 000 of the Harbor Branch Oceanographic Institution.

REFERENCES

- Allredge, A.L. The quantitative significance of gelatinous zooplankton as pelagic consumers. In: M.J.R. Fasham, ed. *Flows of Energy and Materials in Marine Ecosystems*. New York, NY: Plenum Press, p. 407-433, 1984.
- Allredge, A.L. and J.L. Cox. Primary productivity and chemical composition of marine snow in surface waters of the Southern California Bight. *J. Mar. Res.* 40:517-527, 1982.
- Allredge, A.L., B.H. Robison, A. Fleminger, J.J. Torres, J.M. King, and W.M. Hamner. Direct sampling and *in situ* observation of a persistent copepod aggregation in the mesopelagic zone of the Santa Barbara Basin. *Mar. Biol.* 80:75-81, 1984.
- Allredge, A.L. and M.J. Youngbluth. The significance of macroscopic aggregates (marine snow) as sites for heterotrophic bacterial production in the mesopelagic zone of the subtropical Atlantic. *Deep-Sea Res.* 32:1445-1456, 1985.
- Allredge, A.L., J. Cole, and D.A. Caron. Heterotrophic production of bacteria inhabiting macroscopic organic aggregates (marine snow) from surface waters. *Limnol. Oceanogr.* 31:68-78, 1986.
- Allredge, A.L. and E.O. Hartwig. *Aggregate Dynamics in the Sea*. Workshop Report, Office of Naval Research. Washington, D.C.: American Institute of Biological Sciences, 1986.
- Allredge, A.L., C.C. Gotschalk, and S. MacIntyre. Evidence for sustained residence of macrocrustacean fecal pellets in surface waters off southern California. *Deep-Sea Res.* 34:1641-1652, 1987.
- Allredge, A.L., and C. Gotschalk. *In situ* settling behavior of marine snow. *Limnol. Oceanogr.* 33:339-351, 1988.
- Allmendinger, E. Submersibles: Past, Present, Future. *Oceanus* 25:18-29, 1982.

- Asper, V.L. *Accelerated Settling of Particulate Matter by 'Marine Snow' Aggregates*. Ph.D. Thesis, Woods Hole, MA: Woods Hole Oceanographic Institution/MIT, 1986.
- Babb, I. and M. DeLuca, eds. *Benthic Productivity and Marine Resources off the Gulf of Maine*. National Undersea Research Report 88-3, 1988.
- Barham, E.G. Giant larvacean houses: observations from deep submersibles. *Science* 205: 1129-1131, 1979.
- Betzer, P.R., K.L. Carder, D.K. Costello, R.H. Byrne, and R.W. Young. *In situ* laser holography: insights for basic processes/applications for global ocean flux. *EOS, Trans. Amer. Geophy. Un.* 68:1715, 1987.
- Bigelow, H.B. Plankton of the offshore waters of the Gulf of Maine. *Bulletin of the United States Bureau of Fisheries*. Washington, D.C.: U.S. Government Printing Office.
- Bishop, J.K.B., D. Schupack, R.M. Sherrell, and M. Conte. A multiple unit large volume *in situ* filtration system (MULVFS) for sampling oceanic particulate matter in mesoscale environments. In: Zirino, A., ed. *Mapping Strategies in Chemical Oceanography, Advances in Chemistry Series 209*, Washington, D.C.: American Chemical Society, p. 155-175.
- Busby, R.F. *Manned Submersibles*. Washington, D.C.: Office of the Oceanographer of the Navy, 1976.
- Cacchione, D.A., G.T. Rowe, and A. Malahoff. Submersible investigation of outer Hudson submarine canyon. In: D.J. Stanley and G. Kelling, eds. *Sedimentation in Submarine Canyons, Fans, and Trenches*. Stroudsburg, PA: Dowden, Hutchinson and Ross, Inc., 1978.
- Caron, D.A., P.G. Davis, L.P. Madin, and J. McN. Sieburth. Heterotrophic bacteria and bacterivorous protozoa in oceanic macroaggregates. *Science* 218:795-797, 1982.
- Childress, J.J. Physiological approaches to the biology of midwater organisms. In: N.R. Anderson and B.J. Zahuranec, eds. *Oceanic Sound Scattering Prediction*. New York, NY: Plenum, p. 301-324, 1977.
- Childress, J.J. Oceanic biology: lost in space? In: P.G. Brewer, ed. *Oceanography, the Present and Future*. New York, NY: Springer-Verlag, p. 127-135, 1983.
- Davoll, P.J., and M.J. Youngbluth. Production and heterotrophic activity of macroaggregates in mesopelagic regions. *EOS, Trans. Amer. Geophy. Un.* 65:904, 1984.
- Davoll, P.J. and M.W. Silver. Marine snow aggregates: life history sequence and microbial community of abandoned larvacean houses from Monterey Bay, California. *Mar. Ecol. Prog. Ser.* 33:111-120, 1986.
- Doelling, N. and E.T. Harding, eds. *Undersea Teleoperators and Intelligent Autonomous Vehicles*. Cambridge, MA: MIT Sea Grant 87-1, 233 p., 1987.
- Earle, S.A. Microsubmersibles: putting more scientists in deep water. *Sea Technol.* 27:14-21, 1986.
- Elbrachter, M. and R. Boje. On the ecological significance of *Thalassiosira partheneia* in the Northwest African upwelling area. In: R. Boje and M. Tomczak, eds. *Upwelling Ecosystems*. New York, NY: Springer-Verlag, p. 24-31, 1978.
- Flagg, C.W. Can the RDI acoustic current profiler measure zooplankton abundance? *EOS, Trans. Amer. Geophy. Un.* 68:1717, 1987.
- Fowler, S.W. and G.A. Knauer. Role of large particles in the transport of elements and organic compounds through the oceanic water column. *Prog. Oceanogr.* 16:147-194, 1986.
- Galt, C.P. First records of a giant pelagic tunicate, *Bathochordaeus charon* (Urochordata, Larvacea) from the eastern Pacific Ocean, with notes on its biology. *Fish Bull.* 77:514-519, 1979.
- Gardner, W.D., I.D. Walsh, and V.L. Asper. Comparison of large-particle camera and transmissometer profiles. *EOS, Trans. Amer. Geophy. Un.* 68:1716, 1987.

- Gilmer, R.W. and G.R. Harbison. Morphology and field behavior of pteropod molluscs: feeding methods in the families Cavoliniidae Limacinidae and Peraclididae (Gastropoda: Thecosomata). *Mar. Biol.* 91:47-57, 1986.
- Grandvaux, B. Recent and future developments in undersea survey and intervention. In: *Advances in Underwater Technology, Ocean Science and Offshore Engineering*, Vol. 5. *Submersible Technology*, London: Graham & Trotman, Ltd., p. 97-118, 1986.
- Greene, C.H., P.H. Wiebe, J. Burczynski, and M.J. Youngbluth. Acoustical detection of high-density krill demersal layers in the submarine canyons off New England. *Science* 241:359-361, 1988.
- Greene, C.H., P.H. Wiebe, and J. Burczynski. Analyzing zooplankton size distributions using high-frequency sound. *Limnol. Oceanogr.* in press.
- Hamner, W.M., S.W. Strand, G.I. Matsumoto, and P.P. Hamner. Ethological observations on foraging behavior of the ctenophore *Leucothea* n.s.p. in the open sea. *Limnol. Oceanogr.* 32:645-652, 1987.
- Hamner, W.M., C.T. Prewitt, and E. Kristof. Quantitative analysis of the abundance, swimming behavior, and interactions of midwater organisms. In: I. Babb, M. DeLuca, eds. *NOAA National Undersea Research Program, Research Report 88-4*, in press.
- Hanson, L.C. and S.A. Earle. Submersibles for scientists. *Oceanus* 30:31-38, 1987.
- Harbison, G.R. On the classification and evolution of Ctenophora. In: S.C. Morris, J.D. George, R. Gibson and H.M. Platt, eds. *The Origins and Relationships of Lower Invertebrates*. Oxford: Oxford University Press, p. 78-100, 1985.
- Harbison, G.R. Direct observation in plankton ecology. In: R.A. Cooper and A.N. Shephard, eds. *Scientific Applications of Current Diving Technology on the U.S. Continental Shelf*, Vol. 2, Washington, D.C.: U.S. Department of Commerce, p. 85-92, 1987.
- Harbison, G.R. and R.W. Gilmer. Effects of animal behavior on sediment trap collections: implications for the calculation of aragonite fluxes. *Deep-Sea Res.* 33:1017-1024, 1986.
- Hargrave, B.T. Particle sedimentation in the ocean. *Ecol. Model.* 30:229-246, 1985.
- Hawkes, G.S. Manipulators, past, present and future. In: *Proceedings of SUBTECH 1983 Symposium*. London: Society of Underwater Technology, p. 319-329, 1984.
- Janssen, J., G.R. Harbison, and J.E. Craddock. Hatchetfishes hold horizontal attitudes during diagonal descents. *J. Mar. Biol. Ass. U.K.* 66:825-833, 1987.
- Jimenez, R. Composition and distribution of phytoplankton in the upwelling system of the Galapagos Islands. In: Richard, F.A. ed. *Coastal Upwelling*, Washington, D.C.: American Geophysical Union, p. 327-337, 1981.
- Jefferts, K., J. Burczynski and W.G. Pearcy. Acoustical assessment of squid (*Loligo opalescens*) off the central Oregon coast. *Can. J. Fish. Aquat. Sci.* 44:1261-1267, 1987.
- Johnson, K.S., C.L. Beehlee, C.M. Sakamoto-Arnold, and J.J. Childress. *In situ* measurements of chemical distributions in deep-sea hydrothermal vent field. *Science* 231:1139-1141, 1986.
- Knauer, G.A., D. Hebel, and F. Cipriano. Marine snow: major site of primary production in coastal waters. *Nature* 300:630-631, 1982.
- Knauer, G.A., J.H. Martin, and D.M. Karl. The flux of particulate organic matter out of the euphotic zone. In: *Global Ocean Flux Study, Proceedings of a Workshop* (Anonymously edited). Washington, D.C.: National Academy Press, p. 136-150, 1984.
- Kremer, P., M.F. Canino, and R.W. Gilmer. Metabolism of epipelagic tropical ctenophores. *Mar. Biol.* 90:403-412, 1986.
- Lalli, C.M. and R.W. Gilmer. *Pelagic Snails, The Biology of Holoplanktonic Gastropod Mollusks*. Stanford: Stanford University Press, 1989.
- Lampitt, R.W. Evidence for the seasonal deposition of detritus to the deep-sea floor and its subsequent resuspension. *Deep-Sea Res.* 32:885-897, 1985.

- Larson, R.J., L.P. Madin, and G.R. Harbison. *In situ* observations of deepwater medusae in the genus *Deepstaria*, with a description of *D. reticulum* sp. nov. *J. Mar. Biol. Ass. U.K.*, in press.
- Mackie, G.O. Midwater macroplankton of British Columbia studied by submersible PISCES IV. *J. Plank. Res.* 7:753-777, 1985.
- Mackie, G.O. and C.E. Mills. Use of the PISCES IV submersible zooplankton studies in coastal waters of British Columbia. *Can. J. Fish Aquat. Sci.* 40:763-776, 1983.
- Madin, L.P. The production, composition and sedimentation of salp fecal pellets in oceanic waters. *Mar. Biol.* 67:39-45, 1982.
- Madin, L.P. and G.R. Harbison. *Thalassocalyce inconstans*, new genus and species, an enigmatic ctenophore representing a new family and order. *Bull. Mar. Sci.* 28:680-687, 1978a.
- Madin, L.P. and G.R. Harbison. *Bathocyroe fosteri*, gen. nov., sp. nov.: a mesopelagic ctenophore observed and collected from a submersible. *J. Mar. Biol. Ass. U.K.* 58:559-564, 1978b.
- Mauchline, J. The biology of mysids and euphausiids. Part 2. The biology of euphausiids. *Adv. Mar. Biol.* 18:372-623, 1980.
- Mayama, T. The Japan Marine Science and Technology Center (JAMSTEC). *Oceanus* 30: 27-29, 1987.
- Miller, J.E. and D.L. Pawson. An analysis of swimming behavior in four species of bathyal holothurians. *Smithson. Contrib. Mar. Sci.*, in press.
- Mills, C.E. *In situ* and shipboard studies of living hydromedusae and hydroids: preliminary observations of life-cycle adaptations to the open ocean. In: Bouillon *et al.*, eds. *Modern trends in the Systematics, Ecology, and Evolution of Hydroids and Hydromedusae*. Oxford: Clarendon Press, p. 197-207, 1989.
- Mills, C.E. Revised classification of the genus *Euplokamis* Chun, 1880 (Ctenophorea: Cydippida: Euplokamidae n. fam.) with a description of the new species *Euplokamis dunlapae*. *Can. J. Zool.* 65:2661-2668, 1987.
- Mills, C.E., R.J. Larson, and M.J. Youngbluth. A new species of coronate scyphomedusa from the Bahamas, *Atorella octogonos*, new species. *Bull. Mar. Sci.* 40:423-427, 1987.
- Peres, J.M., J. Picard, and M. Ruivo. Resultats de la Campagne de recherches du Bathycaphe F.N.R.S. III. *Bull. Inst. Oceanogr. Monaco* 54:1-29, 1957.
- Platt, T. and S. Sathyendranath. Oceanic primary production: Estimation by remote sensing at local and regional scales. *Science* 241:1613-1619, 1988.
- Price, H.J., G.A. Paffenhofer, C.M. Boyd, T.J. Cowles, P.L. Donaghy, W.M. Hammer, W. Lampert, L.B. Quetin, R.M. Ross, J.R. Strickler, and M.J. Youngbluth. Future studies of zooplankton behavior: Questions and Technological Developments. In: H.J. Price and G.A. Paffenhofer, eds. *Proceedings of Symposium on Zooplankton Behavior*, Savannah, Georgia, 13-16 April 1987, in press.
- Pugh, P.R. and G.R. Harbison. New observations on a rare physonect siphonophore, *Lycnagalma utricularia* (Claus, 1879). *J. Mar. Biol. Ass. U.K.* 66:695-710, 1986.
- Pugh, P.R. and G.R. Harbison. Three new species of prayine siphonophore (Calycophorae, Prayidae) collected by submersible, with notes on related species. *Bull. Mar. Sci.* 41:68-91, 1987.
- Pugh, P.R. and M.J. Youngbluth. A new species of *Halistemma* (Siphonophora, Physonectae, Agalmidae) collected by submersible. *J. Mar. Biol. Ass. U.K.* 68:1-14, 1988a.
- Pugh, P.R. and M.J. Youngbluth. Two new species of prayine siphonophore (Calycophorae, Prayidae) collected by the submersibles 'Johnson-Sea-Link' I and II. *J. Plank Res.*, 10:637-657, 1988b.
- Rechnitzer, A.B. On the upward trend in manned submersible use. *Sea Technol.* 27:10-13, 1986.

- Rudiyakov, Yu.A. Ecosystems of coastal waters as a component of the biological structure of the ocean. *Oceanol.* 27:479-481, 1987.
- Silver, M.W. and A.L. Alldredge. Bathypelagic marine snow: deep-sea algal and detrital community. *J. Mar. Res.* 39:501-530, 1981.
- Shanks, A.L. and J.D. Trent. Marine snow: sinking rates and potential role in vertical flux. *Deep-Sea Res.* 27:137-143, 1980.
- Smith, K.L. Jr. and M.B. Laver. Respiration of the bathypelagic fish *Cyclothone acclinidens*. *Mar. Biol.* 61:261-266, 1981.
- Smith, K.L. Jr., and R.J. Baldwin. Deep-sea respirometry: *in situ* techniques. In: E. Gnaiger and H. Forstner, eds. *Polarographic Oxygen Sensors*. Berlin: Springer-Verlag, p. 298-319, 1983.
- Takagawa, S. Deep submersible project (6,500 m). *Oceanus* 30:29-32, 1987.
- Tietze, R.C. and A.M. Clark. Remotely operated tools for undersea vehicles. In T. McGuinness, ed. *Current Practices and New Technology in Ocean Engineering*. New York, NY: American Society of Mechanical Engineers, p. 19-223, 1986.
- Townsend, D.W. and L.M. Cammen. A deep protozoan maximum in the Gulf of Maine. *Mar. Ecol. Prog. Ser.* 24:177-182, 1985.
- Tregoubouff, G. Prospection biologique sous-marine dans la region de Villefranche-sur-Mer en juillet-aout 1960. *Bull. Inst. Oceanogr. Monaco* 58:1-14, 1961.
- Tusting, R.F. Non-conventional techniques for sampling and collecting marine organisms. *Proc. Pac. Cong. Mar. Tech. PACON '86*. MRM1/12-18, 1986.
- Voyles, Q. and D. Clayton. A submersible-based data display and data logging system. In: D. Steiger, ed. *Proceedings of 4th Working Symposium on Oceanographic Data Systems*. San Diego, CA: IEEE Computer Society, p. 191-195, 1986.
- Urerre, M.A. and G.A. Knauer. Zooplankton fecal pellet fluxes and vertical transport of particulate organic material in the pelagic environment. *J. Plank. Res.* 3:369-387, 1981.
- Widder, E.A., S.A. Bernstein, D.F. Bracher, J.F. Case, K.R. Resenbichler, J.J. Torres, and B.H. Robison. Bioluminescence in the Monterey Submarine Canyon: Image analysis of video recordings from a midwater submersible. *Mar. Biol.*, in press.
- Wiebe, P.H., L.P. Madin, L.R. Haury, G.R. Harbison, and L.M. Philbin. Diel vertical migration by *Salpa aspera* and its potential for large-scale particulate organic matter transport to the deep sea. *Mar. Biol.* 53:249-255, 1979.
- Wood, J.W. and G.W. Potts. Low-light level video system for use in underwater research. *Int. Underwater Sys. Des.* 9:22-26, 1987.
- Youngbluth, M.J. Manned submersibles and sophisticated instrumentation: Tools for oceanographic research. In: *Proceedings of SUBTECH 1983 Symposium* (Anonymously edited). London: Society of Underwater Technology, p. 335-344, 1984.
- Youngbluth, M.J. Water column ecology: *in situ* observations of marine zooplankton from a manned submersible. In: N.C. Fleming, ed. *Divers, Submersibles and Marine Science*. Mem. Univ. Newfld. Occ. Pap. Biol. Vol. 9, p. 45-57, 1984.
- Youngbluth, M.J., P. Kremer, T.G. Bailey and C.A. Jacoby. Chemical composition, metabolic rates and feeding behavior of the midwater ctenophore *Bathocyroe fosteri*. *Mar. Biol.* 98:87-94, 1988.
- Youngbluth, M.J., T.G. Bailey, P.J. Davoll, C.A. Jacoby, P.I. Blades-Eckelbarger and C.A. Griswold. Epibenthic krill impact particle fluxes and food webs: Detection by submersible. In: I. Babb and M. DeLuca, eds. *Benthic Productivity and Marine Resources of the Gulf of Maine*. National Undersea Research Report 88-3, p. 205-214, 1988.

13

U.S. Navy Deep Submergence — 88

Michael R. Popovich

| | |
|-------------------------------------|-----|
| I. Introduction | 209 |
| II. History | 210 |
| III. Mission Profile | 211 |
| IV. Equipment | 211 |
| A. Electrical | 211 |
| B. Hydraulics | 212 |
| C. Sonars | 212 |
| D. Photographic Equipment | 212 |
| V. Interior | 213 |
| VI. Exterior | 213 |
| VII. Data Interfaces | 213 |
| VIII. Recovery | 214 |
| IX. Operations in 1988 | 214 |
| X. Summary and Conclusion | 217 |

I. INTRODUCTION

United States Navy Deep Submergence Vehicles SEA CLIFF (DSV-4) and TURTLE (DSV-3) are homeported in San Diego, California as units of the Commander, Submarine Development Group One. They, along with two Navy Deep Submergence Rescue Vehicles and the Navy Unmanned Vehicles Detachment, reside at the Naval Air Station on North Island, California. Their crewmembers, 3 officers and 13 enlisted, are qualified Navy submariners who are serving these unique billets for 2 or 3 year tours.

The submersibles must be transported to their dive sites via surface support ships. The support ship can be one of the two that the Navy currently has on hire (the M/V Transquest and the M/V Laney Chouest) or another ship of opportunity can be used, provided it has some means of launching and recovering the vehicles.

SEA CLIFF is a 31 ft, 58,000 lb., three man submersible capable of operating at depths down to 20,000 ft. SEA CLIFF, a military asset, is used primarily for deep ocean search, recovery and scientific research. A manned, non-combatant, untethered submersible, SEA CLIFF has access to over 98% of the world's ocean floor and is the nation's deepest diving manned submersible. SEA CLIFF's sister submersible TURTLE is a 26 ft, 52,00 lb., 3 man submersible capable to 10,000 ft, which permits it access to 26% of the seafloor.

SEA CLIFF and TURTLE are powered by silver-zinc batteries that energize electrical systems and side pod propulsion motors. These side pods are trainable to 360 degrees and provide excellent maneuverability. The batteries also provide power to 2 hydraulic power plants that provide stern propulsion and a variety of other functions. Three plexiglass viewports can be used for direct viewing or in conjunction with hand-held video cameras for scientific observation. Two electro-hydraulic manipulators, extended from the front of the vehicle, can be used to recover objects from the ocean floor, attach line reels or collect core samples.

The DSVs are equipped with collection baskets, line reels, television cameras, video recorders, lights, 35 mm cameras, obstacle avoidance and high resolution sonars, gyrocompasses, fathometers and surface/submerged communication devices. The life support systems far exceed the expected length of a mission dive.

The Navy DSVs have several features which enable the vehicle to return to the surface safely in the event of an emergency. These emergency systems can jettison all external weights, the manipulators and both battery boxes. Batteries within the personnel sphere provide power for life support and communication. Any component or system that affects the vehicle safety is rigidly controlled via the U.S. Navy Deep Submergence "Scope of Certification." Exacting, nondeviation design and maintenance standards ensure the reliability of these systems.

II. HISTORY

Initially designed and built by Electric Boat Division of General Dynamics Corporation at Groton, CT, SEA CLIFF (DSV-4) and sister submersible TURTLE (DSV-3) were launched on 11 December 1968. Following initial shakedown and acceptance by the Navy, the DSVs became operational units of Submarine Development Group One in San Diego, CA and conducted missions as deep as 6500 ft for over 10 years.

In 1980, TURTLE completed an overhaul at Mare Island Naval Shipyard that upgraded her depth capability to 10,000 ft. In 1984, SEA CLIFF was similarly upgraded to 20,000 ft and featured a new titanium personnel

sphere and vehicle frame. Following sea trials, SEA CLIFF relieved TRIESTE II (DSV-1) as the nation's 20K deep ocean recovery asset.

III. MISSION PROFILE

The DSVs carry an operational crew of 3 to the ocean floor. Military dives include a pilot, copilot and observer. During scientific dives, a single pilot and 2 scientists, one qualified as Equipment Operator, typically make the dive. The dive is the culmination of the efforts of the entire crew during maintenance and pre-dive checks. The DSV personnel spheres resemble space capsules being just under 7 ft in diameter. They are packed full of equipment necessary to control and monitor submersible operation.

The pre-dive checks take about 2 hr. Prior to the dive, the observer is briefed on the safety/casualty procedures and operational necessities including insulated clothing and sanitary waste disposal. When this has been completed, the crews of the support ship and the DSV take their launch stations and the dive crew enters the vehicle prior to launch.

During the descent, as well as throughout the dive, the pilot monitors all systems for proper operation. The vehicle conserves battery power while descending under the weight of externally mounted descent weights. At approximately 1000 ft above the bottom, the descent weights are dropped and the DSV is ballasted for neutral trim. A bottom approach is then conducted using the electrically powered side pods. (Note, *TURTLE* is not presently equipped with ascent/descent weight system and must use side pods for ascending/descending.) Endurance on the bottom can vary, but is usually limited to 6-8 hr. Longer dives require longer preparation times and reduce the number of dives that can be performed per week. Full battery capacity permits dives of 16 hr at a vehicle speed of 1.5 knots. When the mission is completed or when diminishing battery power dictates, ascent weights are released and the DSV starts its ascent to the surface. Once on the surface and recovered by the support ship, the deep submergence crew performs a series of post-dive checks and scientists can conduct data/specimen analysis.

IV. EQUIPMENT

A. Electrical

The vehicles are powered by 30 and 60 volt batteries. Adequate electrical power is available, but space inside the personnel sphere is severely limited. Available voltages are:

- 60 vdc 120 amps (varies from 51.35 to 70.68 depending on battery charge.
- 30 vdc 100 amps (varies from 25.65 to 35.34 as above; regulated 30 v available)
- 5 vdc
- 120 vac 60 Hz single phase

Pressure resistant penetrators pass electrical signals through the hull via pressure compensated, oil filled cables and junction boxes.

B. Hydraulics

Hydraulic pressure is used to operate devices external to the personnel sphere. A spare mission hydraulic port, controlled by one double solenoid, three position, four way valve, is available for auxilliary use. Applicable parameters are:

- Pressure — 3000 psi
- Flow Rate — 0.5 gal/min
- Fittings — 0.25 in. — forward of the personnel sphere

C. Sonars

Frequencies (kHz) and types of onboard equipment are:

| | |
|--------|---|
| 23 | Fathometer |
| 72-87 | CTFM Sonar |
| 40-55 | Transponder mode of CTFM Sonar |
| 37 | Marker mode of CTFM Sonar |
| 7 | DSV Transponder interrogation |
| 9 | Alternate DSV Transponder interrogation |
| 12-17 | DSV Transponder response |
| 330 | Mesotech Sonar |
| 8.087 | Underwater telephone (UQC) |
| 9.337 | Transponder frequency of UQC in TIPE mode |
| 10.084 | Interrogation frequency of UQC in TIPE mode |
| 14.829 | Pinger frequency of UQC in TIPE mode |
| 14.829 | Echo Sounder freq of UQC in TIPE mode |

D. Photographic Equipment

SEA CLIFF and TURTLE are equipped with a pan and tilt video camera, video tape monitor/recorder, 35 mm still camera and associated

strobe system. The DSVs also employ various CCD mini-cameras for selective mounting and downward looking low light television cameras. Video from any of these cameras can be viewed on a monitor inside the personnel sphere. Additionally, an externally mounted strobe can be triggered by a handheld 35 mm camera when taking pictures from inside the submersible.

The 35 mm camera has data (depth, heading, hours, minutes, seconds, dive number) recorded on the film and displayed in the personnel sphere. Pictures can be taken manually or in automatic. The camera uses 100 ft spools, containing approximately 800 exposures. Pertinent video/photo data:

Pan and Tilt Video Camera

- SIT or CCD TV
- 600 TV lines — horizontal resolution
- Video bandwidth — DC to 8 mHz minimum
- Field of view — 340 deg horizontal and 910 deg vertical (normally limited by mounting position)

The 35 mm camera has the following characteristics:

- Benthos
- 100 ft spool, 800 frames
- 28 mm, f/3.5 Nikkor (water corrected)
- Aperture f/3.5 to f/22
- Viewing angle 50 deg in water

V. INTERIOR

Space in the DSV personnel sphere is extremely limited. A scientific equipment rack, measuring 18 x 12 x 6 inches, is sometimes available depending on other mission requirements. Handheld equipment is kept to a minimum. The 22 inch access hatch precludes the entry of any oversized items. All equipment is checked to ensure that no electromagnetic interference from sphere equipment exists.

VI. EXTERIOR

External equipment must be tested to ensure that it will not implode at working depth. The Navy has strict safety requirements for pressure testing and maintaining applicable documentation. Requests for externally mounted gear are forwarded to the appropriate Officer in Charge.

VII. DATA INTERFACES

Currently there is no available interface to any of the standard equipment such as navigation, communications, sonar, photo, television, or manipulators. The DSVs are scheduled to receive the Submarine Navigation

Integrated Data System, in early 1989, which is estimated to make some of these interfaces possible. Plots of the vehicle's transit are maintained on the surface support ship, as are records of communication.

VIII. RECOVERY

DSV manipulators are located at the forward end of the vehicle, in view of the forward viewport. SEA CLIFF's manipulator motions are toggle controlled by the pilot for picking up objects, connecting line reels or taking specimen samples. TURTLE has an advanced forced feedback manipulator that is capable of more dextrous work. (Upgraded manipulators for SEA CLIFF have been funded and are expected to be installed in 1989). DSV manipulators can be fitted with various tools including parallel jaws, grabber, scissors, drill, and cable cutter.

IX. OPERATIONS IN 1988

SEA CLIFF's year started off with excitement. On 22 Dec. 87, the Unmanned Vehicles Detachment of Commander Submarine Development Group One, while searching for a lost US Marine Corps aircraft, was forced by bad weather to cut the tether line on their deep diving remotely operated vehicle (ROV), the Surface Towed Search System (STSS). The 2000 pound STSS came to rest, upright, in 4200 ft of water with 6000 ft of tow cable trailing behind. SEA CLIFF was dispatched on 4 Jan. 88 to recover the lost STSS.

On 6 Jan. 88, during a brief period of good weather, SEA CLIFF located the STSS on the first dive and cut away the interfering tow cable. After a quick repetitive dive check and a change of manipulator tools, SEA CLIFF dove again with the intent of using a hook, in the starboard manipulator, to attach a kevlar lift line to the STSS termination block (a cone shaped object on the centerline of the ROV). Preparations were also made for an alternative "snare" method using a wire noose held in the port manipulator.

Inadequate clearance between the STSS termination block and its protective fiberglass fairing prevented the attachment of the metal hook and required the SEA CLIFF pilot to opt for the "snare" method. After several painstaking hours, the pilot was finally able to sling the termination block and cinch the noose for a tight fit. The connecting kevlar line was part of a line reel on the front of SEA CLIFF which paid out as the DSV ascended.

Once on the surface, at about 12:30 AM on 7 Jan. 88, the kevlar line was passed to a fleet tug so that the actual lifting could begin. The recovery from 4200 ft was slow and tedious, but not eventful. The 3/8 inch

kevlar line broke once, but managed to wedge itself in the gunnel of the tug, rather than sinking to the bottom with the STSS. The line was reattached to the recovery winch and operations continued.

At about 4:30 AM, the STSS was a mere 50 ft below the surface of the water. Fearing the possibility of losing the quarry at the air-water interface, SEA CLIFF deployed scuba divers to attach safety lines to the STSS, which were ultimately connected to a 2200# buoyancy bag. Thus, if the primary lift line broke, then the deepest the STSS could sink was 50 ft, a substantial improvement over 4200 ft! At 5:00 AM on 7 Jan 88, STSS was hauled aboard the tug in remarkably good condition. Within 10 days, after the installation of new tow cable, the STSS was back in service.

On 24 Jan. 88, SEA CLIFF located a lost Navy ASW torpedo target in about 4000 ft of water in the Southern California operating areas. The target was broken into three major pieces; the forward sections imploded. SEA CLIFF recovered a propeller from the target and conducted a thorough video survey.

After viewing SEA CLIFF's video, TURTLE was asked to recover the stern portion of the target. On 15 Feb. 88, using a "clamp type" torpedo recovery tool, TURTLE attached a lift line to the 1000 pound after portion of the ASW target and the target was hauled aboard the support ship a short time later. TURTLE also recovered 6 deep ocean transponders that were used previously by the Unmanned Vehicles Detachment for the Marine Corps aircraft search. Additionally, TURTLE conducted dot recovery and dot relocation work for the Navy Civil Engineering Laboratory (NCEL).

On 20 Feb. 88, SEA CLIFF was tasked to locate and survey 3 lost, air launched torpedoes in the Southern California area. One torpedo was located intact in 950 ft of water resting upright with an easy attachment point protruding from the nose. Some days you just have all the luck.

On 22 Feb. 88, SEA CLIFF conducted an Underway Material Inspection, with the U.S. Navy Board of Inspection and Survey. The results were satisfactory as SEA CLIFF performed well during her demonstration dive. This dive was combined with a brief, but unsuccessful search for a circa 1920 aircraft, requested by the Smithsonian Institution.

From 21 Mar. until 15 Apr. 88, TURTLE conducted operations in the local Southern California operating area. During this time TURTLE recovered 4 deep ocean transponders and an imploded ASW torpedo target, similar to the one recovered earlier. TURTLE also performed survey work for the NCEL and an indoctrination dive for a member of the Woods Hole Oceanographic Institution. TURTLE also recovered an underwater current meter string for the Naval Ocean Systems Center (NOSC).

On 17 May 88, the new Deep Submergence Vehicle Support Ship (DSVSS) M/V Laney Chouest arrived in San Diego. The new support ship is chartered by the Navy from Edison Chouest Offshore, Inc. from Galliano, Louisiana. The Laney Chouest is a 240 ft, 14 kt ship equipped with an "A-Frame" system for DSV launch and recovery. The new support ship has excellent accommodations and is equipped with expansive wet and dry scientific labs, including installed personal computers for scientific use. The Laney Chouest distributes 12,735 horsepower among three stern propellers and three thrusters giving it excellent station keeping capability. It has a heavy lift system for deep ocean salvage and is outfitted with Loran C, Transit, Global Positioning, Microwave and Phrognav navigation systems. The Laney Chouest also has Sonatrack II and Trackpoint II acoustic systems for long baseline and ultrashort baseline underwater tracking, respectively. Additionally, the support ship has a Sea Beam multi-beam seafloor mapping system which is used for precise bottom contouring.

After initial validation trials on the new support ship, SEA CLIFF conducted 4 dives during the period 24-27 Jun. 88 including a deep dive to 10,000 ft. Based on the positive results of this dive series and a thorough review of quality assurance records, SEA CLIFF was authorized to conduct a 20,000 ft. recertification dive.

On 7 July 88, after a 1300 mile transit to the Murray Fracture Zone located about halfway between San Diego and Hawaii, SEA CLIFF commenced a long descent into 19,000 ft of seawater. Arriving on the bottom at a depth of 19,185 ft SEA CLIFF performed a series of system checks similar to those conducted on seatrials. With only a few exceptions, SEA CLIFF's systems performed flawlessly and the vehicle returned to the surface after a 3 hour ascent.

On 30-31 July 88, TURTLE recovered a failed acoustic hydrophone from the Southern California Acoustic Range in response to tasking from the Naval Facilities Command, Chesapeake. TURTLE performed 2 dives in 5000 ft of water to search, locate and recover the hydrophone and its attached cabling. The dive crew included a member from the parent command as an expert observer during the mission dives. The hydrophone was hauled aboard a support ship, repaired in place and then replanted successfully within 24 hours.

At 1200 h, 26 Aug. 88, SEA CLIFF, embarked on M/V Laney Chouest, departed on a scientific expedition to an area 100 miles west of Northern California known as the Gorda Ridge. Working in support of the Gorda Ridge Technical Task Force (GRTTF), a combined Federal and State agency, SEA CLIFF, accommodating scientists from the USGS, NOAA and various academic institutions (including University of Hawaii) was

tasked to locate areas of underwater hydrothermal venting and map the associated polymetallic sulfide deposits in pursuit of geologic and biologic objectives.

Despite consistently poor weather, SEA CLIFF was able to accomplish 4 dives during the first leg of the expedition in the Northern Escanaba Trough of the Gorda Ridge. During this dive series, SEA CLIFF located numerous sulfide deposits and relocated a hydrothermal vent field discovered by DSV ALVIN earlier in the year. Additionally, SEA CLIFF collected an entire vent colony of biota. Weighing in at approximately 50 lbs, this is believed to be the largest biologic sample ever obtained from a hydrothermal vent field.

Eventually, the worsening weather caused an early end to the first leg dive series and the expedition paused for 6 days in Eureka, California.

The second leg commenced on 18 Sept. 88, but bad weather prevented diving until the 23rd. Diving in an area known as GR-14, in the Northern Gorda Ridge, SEA CLIFF discovered the first high volume, black smoke emitting, hydrothermal vents found on the Gorda Ridge. Three total dives revealed an interesting combination of low temperature, shimmering water type vents as well as the higher volume "black smokers," all within the same general area. This was a particularly important discovery considering that the entire Gorda Ridge area is within the boundary of the U.S. Exclusive Economic Zone (EEZ) and reasonably close to the United States for ease of scientific exploration. SEA CLIFF collected various samples from this area and an impressive array of photographs and video. Two additional dives were conducted in the Northern Gorda Ridge Valley, but no active hydrothermal vents were discovered.

All in all, SEA CLIFF conducted 9 dives on the Gorda Ridge to an average water depth of 10,200 ft. The expedition terminated on 1 Oct 88 and SEA CLIFF returned to San Diego aboard M/V Laney Chouest.

Since returning to San Diego, SEA CLIFF has joined TURTLE in a maintenance and upkeep period at the Submarine Rescue Unit at Naval Air Station, North Island. Both units are expected to be operating again in the December 88/January 89 timeframe.

X. SUMMARY AND CONCLUSION

The United States Navy continues to play an active role in deep submergence in both military and scientific pursuits. The Navy's agent for deep submergence, Commander Submarine Development Group One, is located in San Diego, California, where two Navy Deep Submergence Vehicles are located. DSV TURTLE (DSV-3) and DSV SEA CLIFF (DSV-4) reside at the Submarine Rescue Unit at Naval Air Station, North Island,

when in port, and are supported by their newly acquired DSV Support Ship, M/V Laney Chouest, when at sea.

In 1988, TURTLE and SEA CLIFF combined to recover over \$7,000,000 of military equipment from the ocean floor. Additionally, the DSVs conducted numerous underwater surveys for the Navy Civil Engineering Laboratory and have participated in the planning to assist the Air Force in future recovery work.

Navy Deep Submergence hopes to continue to play a vital role in both military and scientific mission areas. With the advent of the new DSVSS, coupled with the traditional "can do" DSV attitude, TURTLE and SEA CLIFF hope to further contribute to the "Man in the Sea" pursuits.

In summary, the United States Navy Deep Submergence Vehicles SEA CLIFF and TURTLE are among the nation's deepest diving manned submersibles. Having fairly recently completed their upgraded depth capability overhauls, both vehicles possess superb capabilities for underwater search, recovery and scientific exploration. With the delivery of the new DSV Support Ship M/V Laney Chouest, the Navy DSVs now have an essentially worldwide capability. As demonstrated during 1988 to date, SEA CLIFF and TURTLE are ready to further "Man's pursuit in the Sea."

14

The Future of Man in the Sea

John P. Craven

This conference has demonstrated the continuous progress that has been made in adapting the human animal to exist for long periods of time in progressively deeper depths of the ocean. In this development process it has been necessary to construct underwater habitats, decompression chambers, personnel transfer capsules, logistical support barges, propulsion units, submersibles and remotely operated vehicles as required adjuncts of the *Man in the Sea* mission. The difficulties associated with these peripheral functions brings home the fact that the ocean environment is so different from the land environment that it would be better if humankind approached living and working in the sea the same way we would approach the establishment of life on another planet — a planet which had its own life forms and its own basis for life.

Employing this frame work we can see that progress has also been made in adapting humans to live and work in this new planet. The essential ingredients of this adaptation are the ability to construct economic, functional and attractive habitats in ocean space, the ability to supply economic, functional and comfortable transportation for goods and people, the ability to extract energy, fresh water and protein from the ocean environment, and the ability to communicate at high data rates and with clean signals.

In the past two decades the author has been associated in one way or another with projects which have been designed to accomplish one or more of these goals. The first of these has resulted from association with a number of Floating City projects — the U.S. Navy Mobile Ocean Basing System, the Hawaii Floating City Project, the Japanese Aquapolis project at the Okinawa worlds fair of 1974, and most recently the Ocean Information City Project initiated by Mitsui and Nippon Telephone and Telegraph.

The key to success of each of these projects is in the ability to design and construct large superstable modules having small waterplane areas and large underwater volumes that can be assembled and disassembled on the high seas. Most successful of these module concepts has been the triple bottle configuration which was employed in the Hawaii design and actually employed in the CONDEEP oil platforms of the North Sea. The major part of platform stability is achieved by having a very small water plane area so that the motions of the platform are, to a first order decoupled from the surface waves of the sea. The Hawaii floating city study demonstrated that for purposes of human habitation this will not be adequate. The design criteria for motion as employed in that project was to keep accelerations below the threshold of human sensory perception at 0.01 G and to be able to play billiards in a State 7 seaway.

This required, in addition to waterplane stability, the control of the underwater configuration to insure that the added mass of the structure would provide anti-resonant damping of the structure at its resonant frequency and long term dynamic ballasting to compensate for sea state build up and variations of platform load and distribution of platform load. As a result of these developments it is now possible to design large space structures for the ocean whose configuration for function is not determined by the characteristics of the sea and which will not be exposed to the large dynamic loadings which are associated with a heavy sea.

A second major problem which required resolution was that of positioning. For very large structures mooring systems which will be adequate in a storm are impractical and a major cost item in the structure. The Aquapolis was designed to slip its moor in heavy seas and was so located that the platform would be carried away from shore during storms and could be repositioned after the storm had abated. The Marine Information City is designed to be slightly negatively buoyant and to be 'soft landed' on pylons in the sea bed. This would provide a mechanism for positioning that will not require expensive foundations. The Hawaii design is predicated on dynamic positioning with the underwater structure configuration designed to permit the environment to aid in the positioning process.

The technology in place for large platforms in ocean space for a variety of functions ranging from military bases, industrial plants to floating cities, it is necessary to have a transportation system that matches the characteristics of the platform. Although a number of alternatives are possible such as hydrofoils or submersibles, the choice appears to be in the form of the SSP (Semi-Submersible Platform) or SWATH Small Waterplane Area Ship. Basically these ships consist of two underwater hulls which are connected by thin struts to a platform above the water. This is the same principle that is employed in platform design. The

United States Navy first demonstrated this concept in the 190 ton experimental platform the "Kaimalino". Commercial versions of this concept have been developed by Mitsui in the form of passenger ferries, work boats and oceanographic ships, for example, the KAIYO, the Japan Marine Science and Technology Center's (JAMSTEC) newest deep sea dive support ship. Ocean liners and military craft are now on the drawing boards in Japan and the United States and a slow but evolutionary trend toward the utilization of these craft in marine mass transit systems is apparent.

The more fundamental problems of energy, fresh water and protein have been under development at the Natural Energy Laboratory of Hawaii since 1974. The major breakthrough in resolving these problems came in the recognition that the cold deep ocean water of the tropical ocean in combination with the warm surface waters of the tropics provided the generic basis of the production of many forms of energy, including the production of foods and fuels and the generic basis for utilizing this energy for the production of fresh water.

The primary resource characteristic of deep ocean water is that it is cold. Of nearly equal importance is that it is rich in nutrients and that it is biologically pure (only a few diatoms). To understand the fundamental significance of the value of cold we should understand the fundamental nature of natural productivity in any given micro climate. Each sub region of the earth operates as a heat engine with transports of heat coming from the solar irradiation, convection through the transports of fluids such as the gases of the atmosphere and of the water in the form of atmospheric moisture, river flow, oceanic transport. At any moment in time, however, the efficiency of this heat engine is given by the Carnot efficiency $T_1 - T_2/T_1$. In desert regions where the air and ground are at the same temperature $T_1 - T_2$ is very small and the region is unproductive. In the temperate zones in the spring time when the warm spring sunshine interacts with the cold runoff from the mountain snows $T_1 - T_2$ is large and as a result there is evaporation, condensation and rain and of equal importance is the photosynthetic process which generates sugars as well as starch and proteins when the root zone is cold and the leaves are warm. Similar periods of productivity are associated with autumn. In the low tropical islands exposed to trade winds the surface of the ocean water is a cold plate and as a result the temperature of the atmosphere does not decrease until an altitude of about 10,000 feet is reached. The resulting inversion layer prevents the formation of rain. The isothermal character of the land mass and the adjacent fluids (land and water) is such that there is little natural productivity. When there are high islands and winds such as trade winds the moist atmosphere is lifted into colder regions and "orographic" rain is produced. The production of rain is a physical

process in which the heat energy of the environment is converted to mechanical energy to raise the water from the ocean to the top of the mountain. Once again $T_1 - T_2$ is large as a result of the vertical transport of the moist atmosphere.

If now we produce artificial upwelling from the deep ocean to the surface we dramatically change the temperature differences available to the natural heat engine. In regions where the water has upwelled the natural productive efficiency of a low island is increased by factors of four or more; on the high islands the leeward coasts are similarly benefited and in the open sea where there is no natural upwelling an environmental energy potential now exists in what is now a vast tropical oceanic desert. The energy required to bring the water to the surface is, of course minimal, requiring only the energy associated with the density difference resulting from the difference in temperature and salinity between the deep ocean and the surface of the sea. (Indeed the primary energy cost is in the positioning of the deep water in header tanks and reservoirs from which it may flow by gravity to the various facilities).

This realization of the fundamental change in productivity is the cumulative understanding of a wide variety of deep ocean water developments that are taking place at the Natural Energy Laboratory in Keahole Point, Hawaii. We now understand, that with intelligent intervention it is possible to achieve the natural benefits of eternal spring in the tropical oceanic regions. This means the year round production of spring crops such as strawberries, lettuce, asparagus, alpine ornamental flowers etc., the year round production at maximum growth rates of seaweeds such as nori and ogo, of shell fish such as opihi, oysters, lobster, shrimp, the year round production of kelp, of abalone, of trout, salmon, sea urchins, the year round high volume production of sophisticated algae such as spirulina, dananiella, icosopentane, the generation of closed cycle electrical energy without risk of biofouling and with the use of low cost aluminum heat exchangers, the flash evaporation of surface water and condensation with the use of deep ocean water to produce fresh water as a byproduct of the open cycle process. The low cost, non-heat producing air conditioning of buildings, the elimination of chill water generators and cooling devices in industrial production. Many of these processes can employ deep ocean water which has already been used or which is yet to be used.

This technology is being followed closely in Japan and in Europe and in Britain and Canada. We can confidently predict that various forms of energy, and aquaculture plants will be available for the tropical islands in the near future. But the major significance of these developments is the hastening of the transition from fishing to aquafarming and ranching. This transition is occurring most rapidly in Japan where aquaculture now

provides about twenty five percent of the marine protein. The construction of artificial reefs is continuing at a high rate and projects for fertilizing these reefs with deep ocean water are on the drawing boards. The cage culture of salmon in the Norwegian fjords is already a major element in the world's supply of salmon. The ability to produce marine protein continuously through out the year under controlled conditions equivalent to those for the production of chickens and beef at a cost which is comparable to the cost of animal protein of comparable quality will displace the competitive fished product which is the victim of seasonal and annual variations, of the uncertainty of the hunt, of illegal competitors, of the cost of regulation, of the economics of quotas and all of the international encumbrances which make fishing such an interesting and non-profitable operation.

The pattern for the systematic use of deep ocean water as a resource is now clear. We can envision a floating platform complex in a trade wind zone which has little or no precipitation. Deep ocean water is brought to the surface at a temperature of about 6°C. The water is employed in a closed cycle thermal energy plant for the production of energy adequate for the city complex and the community and industry associated therewith. The waste cold water from this process is now at a temperature of about 9°C. (Additional cold water may have to be employed to lower the waste water to this temperature.) The water is then employed in heat exchangers for the air conditioning of the city complex, for chilling fresh water, for industrial cooling and chilling, and for changing the ground temperature of farm land for the cultivation of spring crops. The energy saved per unit volume of cold water in cooling will be an order of magnitude greater than the energy which has been created. The waste water from these processes will still be pure and rich in nutrients and can be employed for the high quality marine products which will characterize the hotel cuisine.

Some decades in the future it will be recognized, as Avery of the Johns Hopkins Applied Physics Laboratory has already recognized, that floating OTEC plants can produce ammonia or methanol at costs competitive with oil. This OTEC process will produce little if any CO₂ in the manufacture and use of ammonia and will produce little if any CO₂ in the manufacture of methanol. The greenhouse-proof advantages of these fuel sources will add a considerable bonus to the economic advantages.

Finally we concern ourselves with vehicles for the undersea and with communication. The development of low cost free flooded Remote Operated Vehicles is proceeding rapidly. The high information capability of the modern computer is the basis for control of these vehicles in the ocean environment and for the implementation of various developments in

artificial intelligence. Breakthroughs have also occurred in hull design with respect to materials (glass, ceramic, maraging steels) and structure (the pre-buckled poly cylindrical hull) which have not yet been implemented but can provide, not only a deep ocean presence, but a deepest ocean presence.

As for communications ocean people have struggled for fifty years or more with the notion that oceanic communication must be broadcast acoustically. Indeed it was hoped that the satellite in combination with surface transponders would be the ultimate in global ocean communication. This concept has been made obsolete by the fiber optic cable whose cost is a fraction of that of the satellite or acoustic network and whose band width is several orders of magnitude greater than that which can be achieved by any other mechanism.

When the remarkable accomplishments of the underwater physiology community are combined with these developments in the utilization of ocean space and the generation of fresh water, power and protein from the ocean then we see that humans are ready to enter the sea as a place in which we can live and move and have our being.

Author Index

Bailey, T.G., 191
Boyce, J.B., 105
Calder, I.M., 163
Craven, J.P., 219
Davis, J.R., 105
Edmonds, C., 149
Falke, K., 57
Hill, R.D., 57
Hochachka, P.W., 57
Hong, S.K., 73
Jacoby, C.A., 191
Kawashima, M., 117
Kindwall, E.P., 131
Krasney, J.A., 1
Liggins, G.C., 57
Lin, Y.C., 33
Mano, Y., 87
Mizuno, T., 87
Ohkubo, J., 87
Park, Y.S., 73
Popovich, M.R., 209
Qvist, J., 57
Schneider, R.C., 57
Shibayama, M., 87
Shiraki, K., 73
Takao, K., 117
Tsunosue, T., 117
Walden, B.B., 181
Youngbluth, M.J., 191
Zapol, W.M., 57

Abstract

Index

A

- Adonis Diving Recorder (ADR), 95, 96
 - diving safety, 102
- Alving, 181-189
 - accomplishments, 183
 - history, 182
 - Mid-Atlantis Ridge, 184
 - R/V Atlantis II, 186, 187
 - R/V Lulu, 182, 183
- Ama*, 73
- Anti-resonant damping, 220
- Antarctica, 58
- Arterial baroreceptors, 8, 9, 11
- Arterial gas embolism (AGE), 150, 159
 - cerebro-vascular system, 150
 - patient position, 151
 - treatment, 151
- Atrial natriuretic peptide, 17

B

- Black smokers, 217
- Breath-hold (BH) breaking point
 - conventional, 38
 - physiological, 38, 39
 - psychological factors, 38
- Breath-hold (BH) depth
 - limit, predicted, 40
 - limit, observed, 41-43, 51
 - professional divers, 41
- Breath-hold (BH) diving, 33
 - ama*, 35
 - bradycardia, 47-50
 - cardiac arrhythmia, 47
 - DCS, 44
 - depth, 40-43
 - diving reflex, 45, 47, 51
 - exercise, 48-50
 - funado*, 40
 - hae-nyo*, 35
 - head-out immersion, 47
 - hyperventilation, 36-38, 42, 45
 - hypothermia, 42-44, 51
 - kachido*, 40
 - katsugi*,
 - near-drowning, 45-47
 - oxygen consumption, 36, 37
 - physiology, 33
 - professional, 34, 39, 73
 - recreational, 34
 - record depths, 41-43
 - record durations, 37
 - shallow water blackout, 45
 - time, 35-39
 - Weddell seals, 57, 58
- Breath-hold (BH), professional, 73
 - body heat content, 75-77, 85
 - diving time, 76-79, 84
 - heat exchange, 75, 77
 - heat production/loss, 75, 76, 79-82, 85
 - hypothermia, 73, 77
 - Japanese, 73, 75-86
 - Korean, 73, 75-86
 - metabolic rate, 75, 79-82
 - rectal temperature, 75, 77-82
 - shivering, 75, 80
 - skin temperature, 77-79, 82
 - surface/dive ratio, 84, 85
 - surface time, 84
 - swimming suits, 75, 77, 81
 - thermal gradient, 77
 - water temperature, 78, 79, 81, 82, 85
 - wet suit, 77, 79, 81-85
- Breath-hold (BH) time
 - hypothermia, 44
 - limit, observed, 36, 37
 - limit, predicted, 36
 - professional divers, 39, 40, 76-79, 84

C

- Caisson decompression
 - air, 131-139, 140, 141, 147
 - continuous, 132, 133

oxygen, 135, 136, 140, 141, 143, 147
 staged, 132
 time, 135, 141

Caisson decompression tables

Autodec III, 144
 Blackpool, 132, 134, 135, 138-140
 Canadian DCIEM, 140
 French, 137, 138, 139, 140
 German, 138, 141
 Japanese, 133
 Milwaukee-air, 140
 Milwaukee-oxygen, 136, 140
 New York, 133, 134
 recommendations, 142
 tunnel vs. diving, 142
 U.S. Navy, 133, 137-139
 Washington State/OSHA, 134-141

Caisson work

aseptic necrosis, 134, 146
 bends incidence, 132
 characteristics, 132
 decompression tables, 131, 132
 exposure limits, 139
 neurophysical evaluation, 144
 saturation exposure, 146
 split shift, 132, 134
 traditional practice, 132

Cardiac nerves, 9

Cardiopulmonary receptors, 7, 8, 14

Compressed air work, 131

CONDEEP, 220

D

DCS

acute, 167, 175
 BH diving, 43, 44
 brain, 173-174
 caisson workers, 132, 134, 144
 chronic, 168-170
 CNS, 164, 176
 EEG, 140
 EVA suit, 106, 108, 110
 experimental, 166, 174, 175
 inner ear, 174, 175
 in-water recompression, 152, 158
 morphological observations, 165, 166
 myelin degeneration, 171-173

neuropathology, 163
 post-mortem, 165
 recovery from, 168-170
 secondary pathology, 152
 spaceflight, 105, 108, 110, 112
 spinal cord, 165-173
 subacute, 167
 treatment, 152
 Type II, 174, 175

Decompression

EVA protocol, 109

Diuresis

immersion, 11
 left atrial balloon, 10, 11
 mechanism, 7

Diving Accident Network (DAN), 89

Diving accidents

barotrauma, 90
 DCS, 152, 159
 depth, 90, 100
 diving experience, 89, 90
 documentation, 157
 drowning, 153, 159
 eustachian tube, 100
 fatal, 88-90
 hypothermia, 153
 infections, 155
 marine animals, 156, 160
 medivac, 160, 161
 non-fatal, 89
 novice divers, 89, 90, 100
 oxygen systems, 157
 panic, 100
 reporting, 89
 survey, 89

Diving injuries

diving experience, 90, 100
 ear pressure gradient, 95, 96
 hearing loss, 90, 91
 inner ear barotrauma, 94, 95, 100
 ruptured eardrum, 90
 sinus squeeze, 89

Diving mammals

adaptations, 58

Diving profiles (Scuba)

Adonis Dive Recorder (ADR), 95-97,
 99, 100
 descent posture, 97
 diving experience, 97, 99
 monitoring, 95-100

- Diving reflex
 free-swimming animals, 58
 restrained animals, 58
 sympathetic response, 68
- Diving Weddell seal
 adaptations, 58
 arterial P_{N_2} , 61, 64-66, 69, 70
 arterial P_{O_2} , 63, 64, 70
 arterial pH, 61
 base excess, 61
 circulating blood volume, 68, 69
 depth, 64, 65
 heart rate, 65
 hematocrit, 61, 62, 63, 68, 69
 hemoglobin, 61, 62, 63, 65-67
 lactate, 61
 nitrogen anrcosis, 65, 57
 nitrogen uptake, 65, 66
 red blood cell (RBC) storage, 67, 68
 spleen, 67-69
- Drowning, 37, 38, 45, 153, 159
 CPR, 46
 diving reflex, 45, 47
 hypothermia, 45, 46
 near drowning, 45
 shallow water, 45
- E**
- ECG
 free-swimming seal, 62
- EEZ, 217
- Envenomation
 fish spine injury, 156
 jellyfish, 156
 neuromuscular venom, 156
- Eustachian tube
 function, 91, 94, 100
 diving experience, 100
 diving position, 96-98
 opening pressure, 93, 94
 sonotubometry, 91, 92
 valsalva, 90, 91, 94, 96, 97, 100
- Extravehicular activity (EVA), 105, 106,
 108, 112, 114
 Apollo, 106
 Gemini 4, 106
 Skylab, 106
 suit, 106, 108, 109
- suit pressure, 106, 108
 system, 108
- F**
- Fluid shift
 immersion, 18
- G**
- Gauer and Henry hypothesis, 2
 Gorda Ridge, 216-217
- H**
- Haldane's rule, 110
 R factor, 110, 111
- HBO
 average number of treatments, 121
 case distribution, 121
 clinical disorders, 120, 121
 circulation disorders, 126
 effectiveness, 122, 123
 Japan, 117
 monoplance chambers, 118
 multiplace chambers, 118
 necrotizing fasciitis, 127, 128
 osteomyelitis, 123, 126
 tissue oxygenation, 125, 126
 treatment tables, 118
 vascular diseases, 126
- Head-out water immersion, 1
- Hypothermia, 42-44, 51, 73, 77
 mild, 153
 severe, 153
 treatment, 153
- Hypoxia, 38
- I**
- Immersion
 aortic transmural pressure, 11
 arterial pressure, 11, 13, 27, 28
 atrial natriuretic peptide (atripeptin),
 17
 cardiac nerves, 9-13, 16, 28

- cardiac output, 3, 4, 9, 11, 27
- cardiac volume, 3
- central venous pressure, 3
- circadian component, 17
- clinical applications, 2
- diuresis, 2, 6, 11-13, 15
- dogs, 6, 9-14, 16, 21
- duration, 7
- ECF volume, 20
- endocrine, 5
- fluid shifts, 18-24, 28
- free water clearance, 6, 12, 13, 15, 16
- head-out, 1
- heart rate, 9, 11
- humans, 6, 9, 11, 17
- hydration, 5
- ICF volume, 20
- kidney, 5
- nitrogen washout, 28
- osmolal clearance, 11, 12
- oxygen consumption, 3, 4
- plasma renin activity, 16
- plasma volume expansion, 2, 18-20, 24
- prostaglandins, 14, 16
- pulmonary changes, 24, 25
- pulse pressure, 11
- regional circulation, 25-28
- renal sympathetic nerve activity (RSNA), 8, 13, 14
- sodium excretion, 2, 6, 11-13, 17
- stroke volume, 9, 11
- thermoneutrality, 5
- vagus nerves, 15
- vasopressin, 15, 16
- water depletion/repletion, 5, 6, 11, 13, 14, 16
- weightlessness, 2
- Inert gas
 - arterial, 60, 61, 64-66
 - solubility, 66
- Infections
 - antibiotics, 155
 - coral cuts, 155
 - otitis externa, 155
 - sinusitis, 155
- Inner ear barotrauma
 - audiometer, 154
 - first aid, 154, 155
 - symptoms, 154
- Insulation
 - body, 83-85
 - suit, 77, 83-85
 - total peripheral, 82, 83, 85
- K
- Kawashima Orthopedic Hospital, 127
- Keahole Point, 222
- L
- Leptonychotes weddelli*, 58
- M
- Man in the Sa*
 - Aquapolis, 219, 220
 - Floating City, 219
 - Future, 219
 - Hawaii Floating City, 219
 - Mobile Ocean Basing System, 219
 - Ocean Information City, 219, 220
- McMurdo Station, 58
- Metabolic rate, 74
- N
- NASA, 105, 108
- Natriuresis
 - immersion, 11
 - left atrial balloon, 10, 11
 - mechanism, 7, 17
- Natural Energy Laboratory
 - aquaculture, 222
 - aquafarming, 222
 - aquaranching, 222
 - artificial upwelling, 222
 - Carnot efficiency, 221
 - deep ocean water, 221, 223
 - productivity, 222
- Neurophysical findings
 - brain atrophy, 142
 - MRI, 144-146
 - psychometric tests, 146
 - Shipley verbal IQ, 145
 - UBO, 145, 146
- Nitrogen Narcosis, 65, 67

O

- Osteomyelitis
 - infection sites, 123
 - HBO effectiveness, 124
 - microorganisms, 124
 - TcPo₂, 125
- OTEC, 223

P

- Pulmonary barotrauma
 - mediastinal emphysema, 151
 - pneumothorax, 151
 - tissue damage, 151

S

- Semi-submersible platform (SSP), 220
 - Kaimalino, 221
 - Kaiyo, 221
- Shallow water blackout, 45
- Small waterplane area ship (SWATH), 220
- Space Station Freedom, 105, 114
- Spaceflight
 - bends treatment apparatus, 112, 113
 - decompression sickness, 108, 109, 110
 - denitrogenation, 108
 - escape system, 112
 - health maintenance facility, 113, 114
 - hyperbaric medicine, 105
 - hyperbaric airlock, 114, 115
 - launch and entry suit, 112
 - manned maneuvering unit, 112
 - medical kit, 107, 108
- Spaceflight physiology
 - cardiovascular deconditioning, 107
 - fluid shift, 107
 - orthostasis, 107
 - space motion sickness, 106, 107
- Spleen
 - contraction, 68
 - RBC storage, 68
 - size, 67, 68
- Sports diving
 - expert, 87
 - Japan, 87, 88

- novice, 87
- population, 87, 88
- safety, 87

- Submersibles (DSV)
 - Alvin, 181, 198, 202
 - Cyana, 202
 - Deep Rover, 199, 200, 202
 - facilities, 202
 - Johnson-Sea-Link, 192, 199, 202
 - manned, 192, 198
 - Nautile, 202
 - new technology, 199, 203
 - Pisces, IV, V, 202
 - recommendations, 201
 - research, 192, 204
 - ROV, 192, 198, 223
 - Sea Cliff, 183, 209, 218
 - Trieste II, 211
 - Turtle, 183, 209-218
 - WASP, 194
- Superstable modules, 220

T

- Thermoneutral water temperature, 42
 - anesthesia, 5
 - dog, 5
 - human, 5, 27

U

- Underwater instrumentation
 - microprocessor monitor, 58, 59
 - blood sampling, 58-60
- U.S. Navy DSV
 - equipment, 210, 211-214
 - history, 210
 - M/V Laney Chouest
 - M/V Transquest
 - operations conducted, 214-218

W

- Water column investigations
 - algal colonies, 197
 - behavior, 192-195, 198, 202
 - fecal pellets, 194-196

- food webs, 193, 198
- marine snow, 194, 196, 197
- metabolism, 198
- organic flux, 197
- particle flux, 194, 202
- pelagic fauna, 192, 198, 200
- species diversity, 193
- specimen collection, 193-196, 198, 200,
201, 203
- trophic relationships, 192
- vertical distribution, 193
- zooplankton, 193-198, 200-203
- Weddell seal, 57, 58
- West Japan Medical Research
Institute, 127
- Woods Hole Oceanographic Institution,
182