WORK CAPABILITY AND PHYSIOLOGICAL EFFECTS IN $He \cdot O_2$ EXCURSIONS TO PRESSURES OF 400·800·1200·1600 FSW

PREDICTIVE STUDIES IV

Institute for Environmental Medicine
University of Pennsylvania

NOTE:

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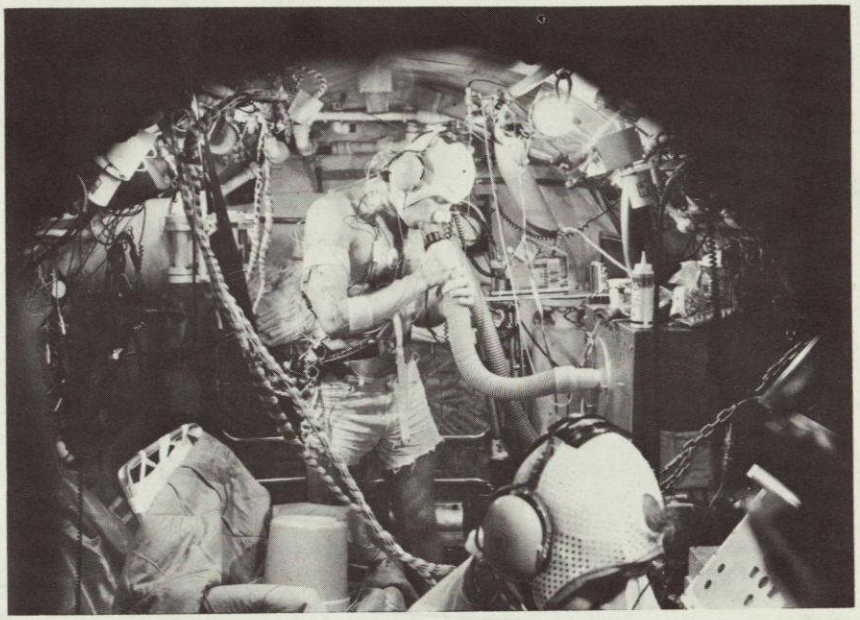
PREDICTIVE STUDIES IV

WORK CAPABILITY AND PHYSIOLOGICAL EFFECTS IN $He-O_2$ EXCURSIONS TO PRESSURES OF 400-800-1200 AND 1600 FEET OF SEA WATER

A COLLABORATIVE INVESTIGATION

EDITED BY
C.J. LAMBERTSEN, R. GELFAND AND J.M. CLARK



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UNIVERSITY OF PENNSYLVANIA MEDICAL CENTER
PHILADELPHIA, PENNSYLVANIA

1978

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SPONSORSHIP OF THE PREDICTIVE STUDY

This extensive program of studies was made possible only through the informed and interested sponsorship by a unique combination of academic, federal and industrial organizations. These were brought together under the established philosophy of the Institute for Environmental Medicine that national aims involving extensions of human capability can be best met by the cooperation and combined assets of the three major working systems concerned.

Specific sponsors included:

Academic

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Federal

U.S. Navy--Office of Naval Research - Naval Medical Research and Development Command (Contracts N00014-67-A-0216-0026, N00014-67-A-0216-0037 and N00014-75-C-1124)
National Institutes of Health--Heart, Lung and Blood Institute (Grant HL-08899-11)
National Aeronautics and Space Administration--Manned Spacecraft Center (Grant NSG-9011)

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SCIENTIFIC PARTICIPATION

COLLABORATION IN PLANNING

To provide international awareness of plans and to encourage scientific interchange in advance, specific communication between the Institute for Environmental Medicine and other laboratories was accomplished before the commencement of experimental activity in this Predictive Study.

Such detailed advance communication occurred in the Institute with members of UCLA School of Engineering and Applied Science, Texas Medical Center, Tarrytown Laboratories, Duke University School of Medicine, British Royal Naval Physiological Laboratory, U.S. Naval Medical Research Institute, Manned Spacecraft Center of the National Aeronautics and Space Administration, Compagnie Maritime d'Expertises, and U.S. Naval Experimental Diving Unit.

The Institute is grateful to each for the free access to experience provided by this exemplary interchange.

COLLABORATING LABORATORIES

The scope and specific investigative purposes of the program plan provided opportunity for direct participation by selected investigators from many laboratories or institutions. Participating laboratories included:

University of Pennsylvania

Institute for Environmental Medicine

Bioengineering	Dermatology	Pharmacology
Chemical Engineering	Medicine	Physiology
Clinical Chemistry	Neurology	Psychiatry
Dental Medicine	Otorhinolaryngology	Radiology

Other Participating Institutions

U.S. Naval Medical Research Institute
U.S. Naval Submarine Medical Center
NASA Manned Spacecraft Center
University of Florida at Gainesville
Virginia Mason Hospital at Seattle

INDIVIDUAL PARTICIPANTS

Correlated physiological studies during multi-week saturation exposures of men in laboratory pressure chambers at high pressures involve the efforts of numerous individuals for preparation of the many related control, instrumentation and life support systems of environmental chambers; for construction, assembly and installation of experiment instrumentation; for operations and safety; for experiment data acquisition and reduction; for medical care; and for logistics and general support. It is the purpose here to acknowledge the dedicated individual participants who made the experiments and Predictive Study possible.

An especially critical and valuable role was played, after the experiment phase had been completed,by a small group of individuals who devoted themselves, over the several years necessary, to provide with the scientific editors the collation, organization and documentation of the results obtained across the entire Study. This support group was composed of M.E. Fletcher as Technical Editor, E.J. Hopkin in biostatistical analysis, B.L. Hanley in data integration, L. Medved in graphic presentations and S. Dechert in technical communications. Their sustained and dedicated contributions were equivalent to the effort of any project in the experiment program.

Direct participation in experiment and preparation is indicated in individual project reports and cited by functions on the following pages.

Subject-Divers

The study depended upon the intelligent cooperation, the initiative and the keen interest of the six industrial divers who volunteered to service as experimental subjects. They had the capacity to learn in a relatively brief time the many specialized technical tasks which made them not only subjects, but also "inside" members of the investigative team. They showed exceptional initiative in preparing for and executing the many necessary investigative procedures that made this study an integrated physiological investigation. They had personal interest in contributing to the advancement of manned undersea activity. Their

responsiveness and awareness in continuing experiment procedures even during periods of symptomatic distress was a large factor in the success of the program.

These Subject-Divers, and their affiliations at the time of the study, were:

Craig R. Carlson	Samson-Ocean Systems, Inc.
Louis W. Jenkins	Sub Sea International, Inc.
Gerard B. McHale	International Underwater Contractors, Inc.
Michael R. Phillips	Samson-Ocean Systems, Inc.
Frank H. Sayle	Oceaneering International, Inc.
William A. Schwab	Seaway Diving A/S

Investigative Staff

The institutional affiliations of the individual investigators are indicated in the specific reports of component projects. The investigative group included:

A.J. Bachrach	K.M. Greene	R.E. Peterson
J.D. Bevilacqua	R.E. Hammond	W.P. Potsic
J.M. Clark	H. Hollien	C.D. Puglia
J.R.M. Cowley	J.A. Kinney	H.B. Rothman
J.O. Donaldson	C.J. Lambertsen	F. Russo
N. Egawa	C.S. Leach	K.H. Smith
A. Findling	L.D. Lowry	J.W. Spencer
D.E. Fletcher	D.J. Montabana	M.T. Troell
R. Gelfand	R. Overlock	R.H. Wilcox

Experiments Support Staff

The blending of Institute staff and student-research associates from the University at large and the Medical School provided the experiment and operations support required. Many served multiple functions, day by day or by night. The many roles played by each individual cannot all be reflected here. Critical functions are indicated on the following page.

Environmental Chamber Operations-Maintenance Team

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V. Tecco	B.J. Kitchens	R.C. Schwarze
D. St. Clair		

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M.L. Cole	R.D. Moorman

Photographic Recording

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W.J. Herr	W.M. Fooshee	A. Ranade
C.M. Hunt	C. Allegra	M.J. Margolies
D.M. Sammaritano	M. Knesevitch	B.A. Haas
G.J. Francis	A.L. Humphrey	R.W. Muldawer

PREFACE

The range of environment in which man can use his peculiar mental and physical assets is being expanded beyond earth's naturally accessible mountainous, desert and arctic regions to include previously inaccessible extremes of the lunar surface and the ocean bottom. The attempt to penetrate the undersea regions, now even beyond the continental shelves, has required a more detailed study of man and his reactions to physiological stress than has the entire program of manned earth-orbiting flight and lunar exploration. Thus far, the great forces and multiple influences of increased ambient pressure encountered under the sea have been overcome practically while still exposing man directly to these pressures. As these forces become greater with increasing depth it will eventually become impractical to expose man directly to the higher ambient pressures, and he will have to be provided with protective compartments in which to perform his undersea work.

A beginning has been made in the effort to improve complex (and still potentially hazardous) mechanical means of providing full mobility while protecting body functions against the direct and indirect influences of hydrostatic and gas pressure. However, it is important now and in the future to realize that this protection approach will always be a special supplement to, and cannot be a full substitute for, direct manned undersea work in diving. At this stage, and undoubtedly for the entire future of diving, work will continue to be done by man exposed to water and pressure--in shallow, in intermediate, and even in deep operations. It will also involve transitional situations in which the assets of diving itself are blended with work performed from within atmospheric submersible vehicles or stations. Such combined situations will provide for the lockout of effective individuals to carry out the intricate work man is endowed to accomplish with his own mind, eyes and hands.

The circumstances in diving will continue to be entirely different from those adopted for manned lunar exploration, and the differences explain the extensive and continuing requirement for study of biomedical effects of increasing environmental stress. It tends to be forgotten that, unlike

the diver, the astronaut-explorer is subjected to essentially no acute physiological stress: he has been protected from the beginning by engineering accomplishments, in a form-fitting "environmental chamber" space suit, against unacceptably low ambient pressure or respiratory gas pressure, or extremes of temperature. He is not narcotized by inert gas, suffocated by the density of the respired medium, poisoned by respired oxygen or by his metabolically produced carbon dioxide, cooled by extremely low temperatures, modified in voice by the gas he breathes, limited in mobility by the viscosity or force of currents in his fluid environment or blinded by the turbidity of the medium in which he works. Quite the opposite, his environment and circumstances are free and even psychologically buoyant.

In spite of the complex stresses encountered in diving and the resulting greater hazard, these have progressively and in part been overcome by research. This has happened to a degree that has made open sea diving practical and useful at depths much greater than considered attainable only a few years ago, and has opened the possibility of still further practical advance. It is now necessary to proceed with further investigations of human tolerance to the pharmacological and physical stresses of pressure, temperature and respired gases, and to improve both operational effectiveness and safety in the process.

The present program, Predictive Studies IV, represents one of a planned series of such investigations, designed to explore human physiological and performance limitations to compression and prolonged exposure to high ambient pressures. At the time these correlated investigations were made, open sea and laboratory chamber studies of men doing practical work underwater had reached a maximum pressure of 636 feet of sea water. The results of Predictive Studies IV have indicated that man is physiologically competent and possesses the ability to perform practical work underwater at pressures equivalent to at least 1600 feet beneath the sea surface.

C.J. Lambertsen
R. Gelfand
J.M. Clark

ABSTRACT

For the fourth in the series of "Predictive Studies", experiments at the Institute for Environmental Medicine during the summer of 1975 exposed men in chambers, breathing helium with oxygen, to progressive increases of pressure equivalent to 400-800-1200-1600 feet of sea water (fsw). Rates of compression were selected to purposely induce physiological derangements associated with both initial exposure to compression and exposure to stable high pressure. Goals included: a) determination of the specific character and time course of onset of physiological and performance decrements during the intentionally rapid compressions, and determination of rates of adaptation on reaching stable elevated pressure; b) investigation of accelerated methods for decompression in deep saturation-excursion diving; and c) determination of competence in practical work performed in water at pressures equivalent to the extreme diving depths of 1200 and 1600 fsw.

To permit discrete measurement of multiple functions during actual compressions, methods were devised to provide repetitive tracking of mental, sensory, speech, neurophysiological, thermal, cardiovascular and respiratory/pulmonary functions while pressure was changed during excursions of 0 to 800, 800 to 1200, and 1200 to 1600 fsw.

Compression rates studied were such that a pressure of 1200 fsw was reached in about half the time previously explored, and 1600 fsw was reached in about 27 hours, on the second day, or approximately 5 days sooner than in previous exposures.

Subjects who developed prominent pressure-induced symptoms following initial compression to the depth equivalent of 1200 feet over a period of three hours recovered enough, within an additional two hours, to continue working. Subjects compressed from 1200 to 1600 feet in 20 minutes on the following day were able to perform detailed investigative functions normally throughout the excursion. Although detailed examinations by electrophysiological and cognitive monitoring methods revealed the persistence of some physiological changes, subjects adapted promptly and were able to carry out intricate technical

work on this occasion and on repeated excursions from a pressure equivalent to 1200 to 1600 fsw.

Decompression after 20-minute, 400-foot compression from the 1200- to the 1600-foot pressure equivalent, with 55-minute actual exposure at the 1600-foot level, was accomplished in one and one-half hours. This is approximately one-tenth of the conventional decompression time from a 400-foot, one-hour compression with helium-oxygen, beginning at and returning to sea level.

Practical work was performed underwater at pressures to 1610 fsw, using pre-trained and timed dismantling and reassembly of oil wellhead components installed in the water-filled chamber as the task. Practical work underwater in chambers or in the open sea before this Predictive Study had been limited to pressures of about 636 fsw. Following validation of physiological adaptations and evaluation of decompression procedures in this program, men worked underwater in stable saturation conditions at pressures of 1210 and 1360 fsw, and at 1610 fsw on 20-minute excursions from 1200 fsw. Work capacity and technical competence were not grossly less than in prior calibration trials at sea level. The results therefore represented an approximately 1000-foot extension of practical working diving depth in water.

Predictive Studies IV has indicated the time course of onset and adaptation to effects of rapid compression and has shown the existence of a capacity for more efficient decompression in excursions during deep saturation than in shallow excursions from sea level. It has demonstrated the ability of men to perform useful scientific, physical and technical work at the high pressures of deep undersea exposures.

PREDICTIVE STUDIES IV

WORK CAPABILITY AND PHYSIOLOGICAL EFFECTS IN $He-O_2$ EXCURSIONS TO PRESSURES OF 400-800-1200 AND 1600 FEET OF SEA WATER

A. INTRODUCTION--THE PREDICTIVE STUDIES CONCEPT

The integrated basic and applied investigations of rapid compression, deep saturation-excursion diving represented by Predictive Studies IV exemplify a necessary but uncommon approach to solution of major national technical problems. For entirely practical reasons of specific research purpose and limited individual assets, biomedical and life-support investigation has traditionally been carried out in many isolated studies by different laboratories with extremely varied goals. Communication is sought, but it is slow and fragmented, considering the massiveness of the general research effort. Integration and correlation of results under these ordinary conditions are extraordinary tasks and rarely practical or complete. Documentation itself is usually partial, segmented and scattered. As a result of these normal obstacles, the return from very large numbers of individual research efforts tends to be limited in the rate at which it can be assimilated and utilized for basic and applied purposes.

Recognizing the need for interdisciplinary and interlaboratory investigations of man and his environment, the University of Pennsylvania established the Institute for Environmental Medicine as an open laboratory to serve as a focus for integrated study of undersea and other environmental stresses upon human activity. In its initial design, organization and supporting systems, the Institute was provided with means for aggregating the individual skills and technical assets of this University and other institutions toward accomplishment of investigations of a scope not practical in one agency alone.

A series of collaborative investigations designated as "Predictive Studies" was initiated to provide opportunity and stimulus for advancement of manned undersea activity. These studies began with determinations of prolonged,

saturation exposure to increased nitrogen pressures (Predictive Studies I and II) (6,7). They continued with investigation of the limits of human tolerance to extremes of increased respiratory gas density at elevated ambient pressures (Predictive Studies III) (4,5).

The present program, Predictive Studies IV, was a timely and natural outgrowth of emerging world interest in deep undersea exposures and of the convergence of fundamental scientific, practical economic, political and technical operational circumstances. The abrupt imposition of an oil embargo by Arab countries and the nearly as abrupt awakening to the limits of world energy sources both came at a time of exceptional opportunity for scientific investigation of means to increase man's capability to perform direct work deep in the sea. The Institute's Predictive Studies III had just demonstrated that man could be fully effective mentally and physically to a pressure equivalent of 1200 feet of sea water (fsw) if he proceeded to this depth slowly. Laboratories in France and England, and in the U.S. Navy, had found distinct difficulties in exposures close to and deeper than 1200 feet, with indications that the difficulties were related to neurological effects of the compression (1-3,8-11).

To meet both scientific and operational challenges, the Institute developed plans and invited participation in the fourth Predictive Study, to encompass detailed investigation of compression tolerance limits and practical extension of manned undersea activity to pressures of 400, 800, 1200 and 1600 fsw. The plan required committed, close collaboration among scientific departments of the University of Pennsylvania and other universities; support by federal agencies, including Navy, National Institutes of Health and NASA; and support by the industrial groups involved in offshore oil and gas production. These included the oil, diving, and gas and chemicals industries, all which use the results of such investigations.

Drawing together for common scientific p these very different institutions and agencies, all [illegible] different goals and motives, proved to be possible, albeit laborious. The extensive effort and direct communication involved in establishing collaborative support and identification with the research program was aided by the

recognition of the unique advantages and opportunities provided by University-Federal-Industrial collaboration.

For the University and the Institute, this collaboration provided important outlets for the results of continuing fundamental research. For the Navy, NIH and NASA, exceptionally extensive information could be expected from the unique blending of programs they supported. For the diving industry, clearer guidelines were expected for more practical and safe conduct of offshore construction and support operations. For the oil and chemical industries, an opportunity existed to aid in making possible the deeper explorations and oil well completion work that is now required in the search for energy sources offshore.

It has become even clearer since the completion of Predictive Studies IV that for support of either general research or major interdisciplinary investigations toward advance of deep or intermediate depth undersea work:

> "the time has come for the users to invest in obtaining the critically important information, sought after in step-by-step investigations of immense technical difficulty. The desired next phases of research and application must be carried out with continuous investment by industry, aiding itself by this means."[1]

Behind this now evident conclusion must remain the awareness that the continuity of basic and applied investigations and the development of individuals dedicated to conduct the research also require the appropriate blending of prominent supporting roles by the federal health-related agencies, energy agencies, ocean agencies, and the operating navy.

It is expected that the nature and results of the present program will be useful background for those faced with consolidating these aims.

[1] C.J. Lambertsen. Preface. In: Underwater Physiology V. Proceedings of the Fifth Symposium on Underwater Physiology. Bethesda: FASEB, 1976.

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B. BACKGROUND

Status of Undersea Operations

Approaches to Undersea Activity

- Nonsaturation, Limited-Duration Diving from the Surface, with Decompression to Atmospheric Pressure
- Stable, Multi-day "Saturation" Exposures to the Actual Working Depth
- "Saturation-Excursion" Exposures
- Submergence Within Protective Compartments

Status of Physiological Investigations

- Separation of Narcosis, Density and Pressure Influences in Man
  - Helium Narcosis and Excitation
  - Effects of Respiratory Gas Density
  - Varied Approaches to Investigation of Compression Syndrome

B. BACKGROUND

STATUS OF UNDERSEA OPERATIONS

Between 1965 and 1975, in the decade before this Predictive Study, striking advances in manned undersea work had resulted from a composite of factors. These included imaginative investigation in several countries, circumvention of some physiological limitations, and sensible supportive engineering development (e.g., of submersible and deck chambers) to permit more effective use of the diver. A major force in progress was the evolution of important purposes for improved manned undersea activity. These advances were made across an extremely broad front of scientific and technical gains and provided varied pathways for the continued improvement which has since occurred.

When the Institute's environmental chamber systems were designed in 1964, commercial working diving was with difficulty being conducted to about 250 feet; limitation to approximately 300 feet was imposed upon U.S. Naval diving due to need for further decompression research; and the important concept of saturation diving had only begun its required decompression research and trials at sea (9,18,42,52).

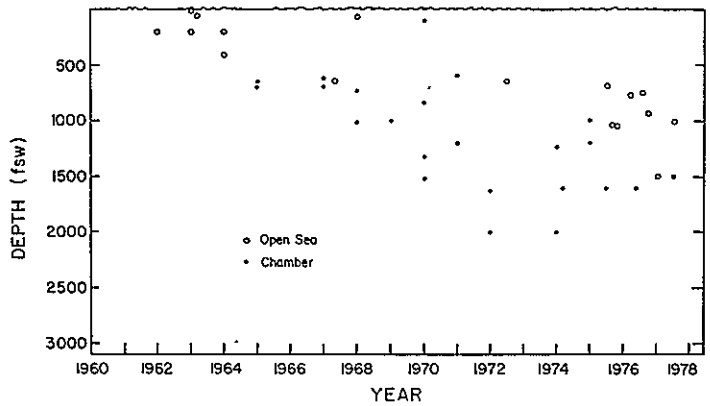

FIG. 1. Evolution of Laboratory and Open-Water Pressure Exposures. The diagram shows representative activities in research and successful open-water applications. Unsuccessful trials or trials involving serious accident are not included.

Figure 1 summarizes the slow, laborious advance of diving and deep diving research effort during the past decades, and it clearly indicates that the present is the time of most rapid change. The specific purposes and the methods employed in major episodes shown are elaborated in the individual reports of this study. It is the intent to point out here that at the time of Predictive Studies IV (1975), commercial open sea diving work was generally still limited to depths of about 450 feet, even though laboratory studies had indicated man's capacity for extreme physical work in dry chambers at 1200 feet (30) and for exposure to and return from helium pressure equivalent to up to 2001 feet (43, 45) (see Fig. 2). Performance of practical work underwater, even in laboratory systems, had not exceeded 636 feet (38).

In the year immediately following completion of this study, open-sea penetration by divers was progressively made to depths of 1148 fsw (55). In 1977, practical work in the open sea was carried out to 450 meters (approximately 1500 fsw) (16).

APPROACHES TO UNDERSEA ACTIVITY

Each of the various methods of compression which have evolved and improved strikingly during the past years can be expected to be useful indefinitely, for operational purposes from the surface or from lockout submersibles, and as the bases for the fundamental investigations required to consolidate the recent gains. These methods include: a) nonsaturation, limited-duration diving from the surface, with decompression to atmospheric pressure; b) stable, multi-day "saturation" exposures to the actual working depth; c) "saturation-excursion" exposures; and d) submergence within protective compartments.

NONSATURATION, LIMITED-DURATION DIVING FROM THE SURFACE, WITH DECOMPRESSION TO ATMOSPHERIC PRESSURE

Most open-sea work at shallow and intermediate depths is carried out in this manner, whether with air or with

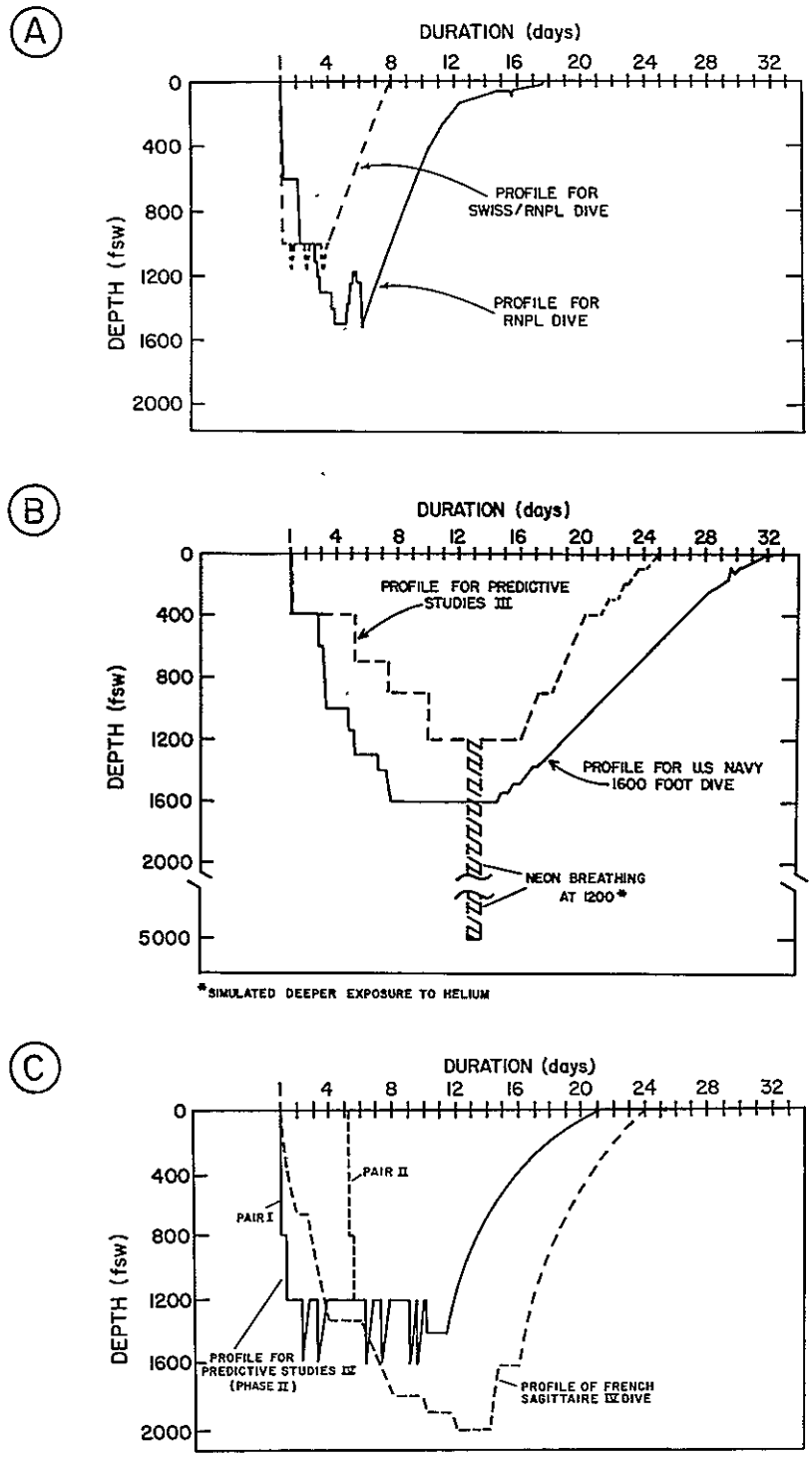

FIG. 2. A,B,C. · Helium Saturation Exposures in Laboratory Chambers. Comparison of Pressure-Duration Profiles of Major Chamber Studies. A, Swiss/RNPL dive (17); RNPL dive (3). B, Predictive Studies III (32); U.S. Navy 1600-foot dive (48); C, Predictive Studies IV, Phase II(31); French Sagittaire IV dive (47).

helium-oxygen or other mixtures. Applications of this method to the limits of continental shelf operations have been made practical by development and use of submersible and deck decompression systems (18,52). For deep diving from the surface, the final phases of decompression are now being rationally treated as decompression from an intermediate depth saturation exposure.

STABLE, MULTI-DAY "SATURATION" EXPOSURES TO THE ACTUAL WORKING DEPTH

Multi-day experimental saturations in the open sea progressively extended in duration and depth from the initial exposures of 100 feet and 30 feet, through 432 feet (9,18,42) to practical trials of saturation at a 615-foot depth (38), to the current 1978 commercial working saturation diving at 1000 feet (50). Since the saturation methods result in equilibration of all body tissues with the partial pressure of the inert gas breathed, rate of eventual decompression is limited by the rate of pressure decrease that will allow proper elimination of inert gas from those tissue sites least well-perfused with blood. Because nearly a day is required for each 100 feet of such decompression, approximately a week is used for decompression from any time period, from a day to many weeks, of saturation with helium at pressures close to 1000 fsw (17,32).

"SATURATION-EXCURSION" EXPOSURES

This approach, applicable to shallow, intermediate and deep diving, offers the possibility of transient descent (excursion) to a depth greater than the pre-selected, stable level of saturation with the respired gas. The inert gas and the oxygen concentration are selected as appropriate for the circumstances. Nitrogen-oxygen mixtures can be used in relatively shallow depths, while helium is required for deep diving. Excursion from and back to the saturation level provides opportunity for repeated short exposures to extreme depths, with decompression requirements pertinent to the excursion. This is followed eventually by the

slower decompression to 1 ata from the relatively shallower saturation depth from which the excursion diving occurred. It was this well-known concept of saturation-excursion pressurization that was used in Predictive Studies IV to allow investigation of the influences of rapid, deep compression and rapid decompression from excursions at high pressures.

SUBMERGENCE WITHIN PROTECTIVE COMPARTMENTS

The sealed atmospheric compartment and rigid diving suit are emerging to an appropriate role in very deep penetration, offering some capacity for visibility and manipulation. Their inherent problems of structural integrity, fouling, thermal protection, carbon dioxide removal and oxygen control are engineering problems, not biomedical. Such methods and direct diving are considered complementary, not mutually exclusive, and will continue to be useful alone and together. This combination is exemplified by the versatile diver-lockout submersible.

The background combination of investigative, engineering and operational advances in diving has been described here to indicate general circumstances relative to the design of Predictive Studies IV in particular, and to emphasize the rapidity of application and advance generated by the composite of investigation and development.

STATUS OF PHYSIOLOGICAL INVESTIGATIONS

In the early years of diving and diving research, the physiological problems encountered were induced by compressed air, and at relatively low pressures of one to two hundred feet of sea water (5). They included the incapacitating mental confusion of nitrogen narcosis, the superimposed mental and respiratory effects of carbon dioxide accumulation due to inadequate ventilation of the helmet, and the severely damaging consequences of decompression sickness (5). Oxygen toxicity subsequently became recognized as a limitation of both diving and therapy with air, more obviously limiting in pure oxygen diving and in the use of oxygen in decompression (51).

The introduction of helium in practical diving (44,51) removed the prominent narcotic influence of nitrogen, although it introduced substantial interference with voice communication, due to its effect on acoustic velocity in the vocal tract, and exaggeration of heat loss because of its high thermal conductivity.

Through these years and stages of practical diving and research, until less than 10 years ago, hydrostatic pressure presented no evident problems, except by way of its influences upon gas density, thermal conductivity, pharmacological effects of increased gas partial pressure, and problems secondary to inert gas uptake and elimination. Physical effects of hydrostatic pressure itself, unrelated to respired or ambient gases, had not been recognized in diving.

As diving research extended to higher pressures, different investigators encountered manual tremor and central nervous system changes in man with confusion and performance decrements not characteristic of narcosis (8,13,21,22). The phenomena have appeared to represent central nervous system excitation rather than narcosis, and it became confirmed in small mammals compressed to extreme pressures in helium that tremor persisted in conscious animals (11,24,36, 37,39,40) disappearing without evident residue on decompression (24,39). The composite of symptoms and observable effects came to be designated as "high pressure nervous syndrome" (1,13). At first, and to some extent still, the neurological excitations were generally ascribed to effects of helium, even referred to as "helium tremors" (2,53). However, the many basic studies of pressure effects on life processes have made it evident that high hydrostatic pressure, unrelated to gases, has deleterious effects on neuromuscular functions of aquatic animals and amphibians, on growth and metabolism of bacteria and even on enzymatic processes involved in metabolism (2,20,24,33,34,54). These direct hydrostatic effects had initially tended to be generally ignored by diving physiologists, and attention remained focused on helium, because the pressures at which most studies on cell systems and cold-blooded animals had been conducted were higher than those expected to prove tolerable to man on other grounds. Even with helium as the respired gas, it had been predicted that narcotic or respiratory limitations (due to high gas density and associated work

of breathing) would progressively prevent human activity at pressures less than 1800 fsw (2,29,35).

As laboratory pressure studies in man and animals continued it became obvious that small mammals rapidly compressed encountered severe central nervous system electrophysiological effects (12), even at pressures within the range of probable respiratory competence. Study of this in isolated nerve and across synaptic neural junctions indicated depression of transmission (23,28) at pressures no greater than 2000 fsw, the highest pressure to which man had been exposed (43). As such findings became more generally known it also became obvious that man should be considered susceptible to similar effects.

SEPARATION OF NARCOSIS, DENSITY AND PRESSURE INFLUENCES IN MAN

Any compression of man or other mammal breathing helium must involve a) the composite influences of hydrostatic pressure, b) all pharmacologic and/or biophysical effects of dissolved helium on any structure or chemical process and c) the physical effects of increased gas density upon respiratory/pulmonary function and intrapulmonary gas diffusion.

Helium Narcosis and Excitation

As a step in separating these factors, Predictive Studies III effected dose-response comparisons of nitrogen, neon and helium during slow, multi-day, stepwise increases in pressure over the range of 100, 200, 400, 700, 900 and 1200 fsw (30) (see Fig. 2B). These studies indicated no evident depressant effect of helium (or neon) on higher mental functions of man at pressures to the 1200-fsw maximum pressure of exposure. Nor, with the slow compressions employed, was prominent tremor or electrophysiological change encountered, even when helium and crude neon, or nitrogen-helium mixtures and helium, were alternately breathed at high pressure. For these reasons it was concluded that helium itself induced neither narcotic nor excitatory effects upon neural functions in either chronic

or acute elevation of partial pressure in man (30). On the basis of "dose-response" characteristics of the observations with nitrous oxide, nitrogen, neon and helium, as illustrated in Fig. 3, it was further concluded that helium effects should not be detectable even at pressures nearly twice those used in Predictive Studies III (30). If this is so, effects of absolute hydrostatic pressure and rate of compression can be investigated in man breathing helium.

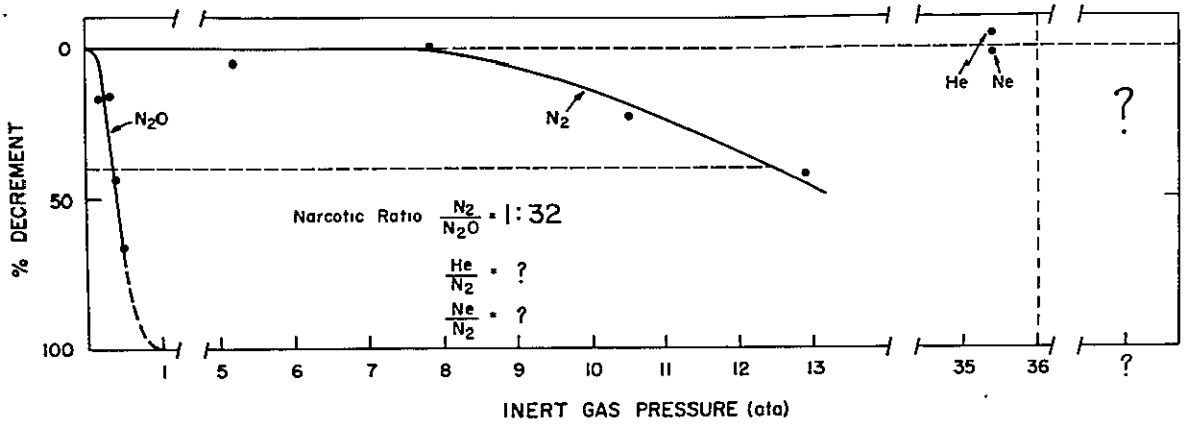

FIG. 3. Comparative Narcotic Effects of N_2O, N_2, Ne and He on Mental Function (Arithmetic Index). After Lambertsen (30).

Effects of Respiratory Gas Density

By administering crude neon at 400, 700, 900 and 1200 fsw, with density close to five times that of helium, it was possible in Predictive Studies III to simulate the pulmonary function and pulmonary respiratory consequences of helium breathing at ambient pressures to 2000, 3000, 4000 and 5000 fsw (30). Similar use of neon at increased pressure in studies of pulmonary function (10) and of narcosis (25) had been made earlier. Maximum ventilatory capacity and maximum expiratory/inspiratory flow rates were progressively reduced by increasing gas density (Fig. 4). However, these reductions, even in the most severe situation (simulation of helium breathing at 5000 fsw), still provided alveolar ventilation without evidence of hypoxia or dyspnea at rest or in mild exertion (32). It has been therefore proposed (32) that pulmonary function and alveolar-arterial gas exchange should not be considered seriously limiting even at pressures up to the maximum

(2001 fsw) pressure experienced by man (15,43). If this is true, then it should be possible to study exposure to increased hydrostatic pressure, or to rapid compression, based upon the observed projections of density-pulmonary function relationships, without concern for incapacitating symptomatic dyspnea at rest or in mild exercise on compression.

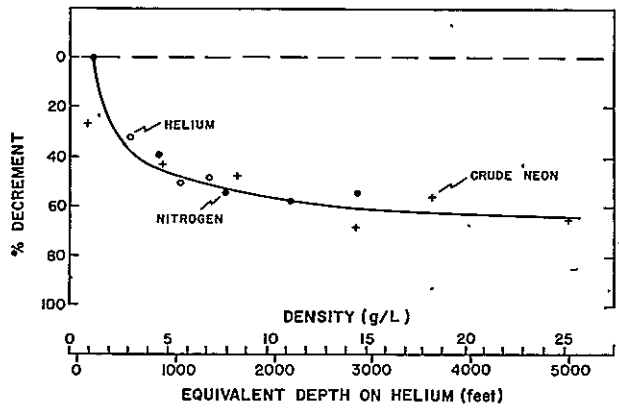

FIG. 4. Influence of Increased Gas Density Upon Human Pulmonary Function. Percent decrement in maximum voluntary ventilation in two subjects of Predictive Studies III (32).

In spite of these predictions and projections, dyspnea has been reported in mild exertion at 1600 fsw (49) and in leg exercise at 1400 fsw (19) (see Section F). These symptomatic observations led the design of Predictive Studies IV to select the pressure of 1600 fsw as a parameter of study and to include investigation of respiratory control and respiratory muscle function, as well as pulmonary airway mechanics.

Varied Approaches to Investigation of Compression Syndrome

During the past five years of expanded pressure research activity, three different approaches to investigation of the Compression Syndrome have dominated. These have been:

a. Slow, multi-day compression to high absolute pressures of 1600 and 2001 fsw; with many days at high pressure, using helium with near-normal P_{O_2} as the respired atmosphere (45,47,48). These studies have provided indications

of persistent, if slight, neurophysiological effects, even after prolonged residence at high ambient pressure.

b. Rapid, transient compressions to pressures of 1000 to 1500 fsw, using helium and nitrogen together as the inert gas diluent for oxygen in the respiratory atmosphere (4,6, 7,26). Such trials of the three-gas mixture were based on a concept, derived from animal studies of hydrostatic pressure-narcotic interactions (14,27,41), that use of a narcotic gas or other narcotic might counteract any neurological effects of high helium pressure, and they have entailed early withdrawal from pressure exposure rather than the tracking of exaggeration or adaptation to high-pressure effects. The use of nitrogen has not been found to be selective in preventing neurological effects, when compared with helium-breathing controls (46).

c. Rapid compression, with helium-oxygen only as the respired atmosphere, with monitoring of deterioration and/or adaptation during sustained exposure (31). This approach is represented by the studies reported in detail herein.

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C. PURPOSES

C. PURPOSES

At the stage of planning for this fourth Predictive Study, men had already been exposed in dry chambers, by slow, multiday compression, to helium pressures equivalent to the range of 1000, 1200, 1500, 1600 and 2000 feet of sea water (fsw) (1-3,5,7-9,11,12,14). More rapid compressions, mostly to lesser pressures, had also been accomplished (4,6). However, without precise cross-relationship of method, or quantitation in measurement, or use of heavy physical work stress to magnify changes, correlation of most of the important observations of the several large studies involved in these many efforts has been generally impractical. Without full reporting of findings, even important isolated observations could not be understood.

Three specific interpretations of these prior pressurization studies were made as a guide to determining the most useful direction for Predictive Studies IV. These were:

a. After slow attainment of 1200 feet in Predictive Studies III, exposure to helium revealed no important abnormalities of mental, neurophysiological, physical or respiratory/pulmonary function (7).

This 1200-foot pressure and the data obtained were therefore selected as one baseline or reference condition for the design of Predictive Studies IV.

b. In exposure to 2000 feet (621 meters) in helium-oxygen, even when the pressure was attained gradually over seven days (9,10), electroencephalographic abnormalities were found to persist in the subjects until decompression had lowered the ambient pressure. This, and a respiratory limitation reported in moderate exertion at the 2000-foot pressure equivalent, led to selection of a lower maximum pressure of exposure for Predictive Studies IV.

c. In another study (12,13), slow compression over several days to 1600 feet in a helium-oxygen atmosphere induced detectable effects on balance, with reports of a degree of respiratory distress not to be expected from the projections of pulmonary function derived from the Institute's investigations of neon breathing at a 1200-foot pressure equivalent in Predictive Studies III (7). This pressure of 1600 feet, studied in stable saturated state (12), was therefore selected as a second reference level for investigations of more rapid compression.

The intentional cross-relation of the planned studies with major previous investigations of 1200- and 1600-foot saturation exposures, and the methodological linkage to the higher, 2000-foot pressure study, allowed for investigation of rate of compression within a range of absolute pressures bracketed by studies in the saturated state in which positive effects of compression had been reported. It is urged that this approach of pre-planning for correlation of results be exploited as a normal procedure in such technically extensive investigations as those performed on man at extreme pressures.

GOALS OF THE STUDY

QUANTITATION OF COMPRESSION EFFECTS IN MAN

A central purpose of the program was to perform quantitative measurements in man of changes induced by compression itself in fundamental physiological processes and overall performance.

INVESTIGATION OF UNMODIFIED EFFECTS

Considering that the previous Predictive Study (III) had indicated no detectable severely limiting effect of helium at pressures equivalent to 1200 fsw and pressures projected well beyond this pressure, helium as a respired gas was considered an incidental and noncritical component of the compression environment.

There were additional reasons for using a helium atmosphere, unmodified by other inert gases. It was not the purpose of the studies to attain high pressures

without producing derangements or while in any way masking unknown effects of compression. Considering that the effects of rapid compression with only helium as the respired gas had not previously been well-defined, the Predictive Study was designed to obtain the unmodified responses to such compression.

INDUCTION OF DERANGEMENTS, ENABLING STUDY OF ONSET AND ADAPTATION

It was additionally considered that the greatest return in the study could be obtained by purposely inducing prominent effects of compression, both to allow them to be quantitatively measured and to permit tracking of the pattern of onset and any adaptation that might occur. This purpose required selection of rates of compression for each step in absolute pressure, which would induce detectable changes without producing incapacitation or requiring withdrawal from the study before any possible adaptation occurred. Selection of these rates was accomplished by detailed appraisal of the previously reported compressions of men to pressures greater than 600 fsw.

INVESTIGATION OF INTERRELATED EFFECTS

A further purpose of the program was to continue concurrent investigation of related physiological and performance alterations, as in the previous Predictive Studies. Since many of the influences of compression were expected to involve neural, neurophysiological, neuromuscular and membrane functions, much emphasis was placed upon quantitation of mental function, sensory acuity, psychomotor function and coordinated performance, discrete and purposeful work capacity, and integrity of respiratory and circulatory control systems. The earlier Predictive Studies showed that pulmonary airway and pulmonary mechanical functions, at rest and during severe exercise, do not limit human activity when compression to 1200 fsw is carried out over several days. Nevertheless, studies of respiratory control and pulmonary function at rest and in exercise were planned for the present investigations of more rapid

pressurization for several reasons: as a safeguard, as a practical definition of respiratory-pulmonary competence, and as a basic investigation of effects of any respiratory neural, neuromuscular or bronchomotor influences which might be induced by changes in hydrostatic pressure.

STUDY OF PRACTICAL UNDERWATER WORK CAPABILITY IN DEEP EXCURSIONS

Because of the continuing interest of laboratory and operating groups in linking fundamental physiological investigations to modern practical purposes, detailed studies of metabolic and control functions in exercise at extreme pressure in the helium environment were followed by practical, operations-oriented studies conducted underwater at high pressures. These concerned performance of specific skilled and strenuous underwater work functions related to deep oil wellhead diving. This phase of the program was conducted only after assurance of physiological competence in rapid, deep compression. It was performed in the Institute's water-filled pressure chamber on work equipment provided by the offshore industry.

INVESTIGATION OF ACCELERATED DECOMPRESSION IN DEEP EXCURSION-SATURATION EXPOSURES

Growing recognition that tolerance of rapid decompression is greater at high pressure than near atmospheric pressure led to inclusion of deep excursion-decompression exposures in this Predictive Study. Decompression schedules selected for stepwise investigation between pressures of 1200 and 800 fsw, and 1600 and 1200 fsw, were approximately one-tenth the duration of decompression for the equivalent 400-foot excursion from atmospheric pressure.

SUMMARY OF GOALS

1. Fundamental quantitative investigation of observations that deep excursions, rapid pressurizations, and especially the combination of deep and rapid

pressurizations lead to neurophysiological derangement, with changes in:

Mental performance--brain electrical activity;
Sensory function--balance and coordination;
Muscular competence--neuromuscular function;
Metabolic state.

2. Study of onset, degree and adaptation of detectable changes due to high pressure and rapid compression.

3. Utilization of the baseline finding in the previous study of this series that neither narcotic, performance, neuromuscular, nor respiratory-pulmonary limitations are imposed by sustained exposure to helium at pressure equivalent to 1200 fsw.

4. Utilization of the baseline finding in the previous study of this series that physical aspects of respiratory-pulmonary function at rest and in moderate physical exertion should not be limiting during helium breathing even to depths of several thousand feet of sea water.

5. Investigation of U.S. Naval and French observations of detectable dyspnea in moderate exertion during long exposure at helium pressures equivalent to 1600 and 2000 fsw.

6. Investigation of practical work capability in the underwater environment in deep stable saturation and on rapid excursion to high pressures.

7. Evaluation of temperature tolerance and thermal balance at high helium pressures, with control of temperature effects in deep excursion pressurization.

8. Evaluation of laryngeal speech function in relation to helium pressure and to extreme rates of pressure change.

9. Demonstration of accelerated decompression on return from excursions to pressures greater than saturation exposure.

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D. EXPERIMENT PLAN FOR PHYSIOLOGICAL AND PERFORMANCE STUDIES

Scope of Related Investigations

Compression Rate Plan

Pressure-Duration Profiles and Subject Pairs

Measurement Module System

Subject-Divers and Training

Organization for Collaborative Experiments

Decompression System

Underwater Work Plan

Operational Procedures

Instrumentation

D. EXPERIMENT PLAN FOR PHYSIOLOGICAL AND PERFORMANCE STUDIES

SCOPE OF RELATED INVESTIGATIONS

While primary effects of rapid compression were expected to be neural and neuromuscular in expression, the magnitude and duration of overall physiological stress required design for examination of multiple functions. The component parts of this Predictive Study, intricately related to its predecessors (I, II and III), are summarized in Table I as areas of interest. The purposes and methods of investigation varied among these component projects, depending largely upon whether a measurement pertained to acute effects of rapidly changing pressure, or adaptation to sustained high pressure. The table reflects the special attention to influences on neural and membrane functions.

To provide for obtaining of control values and for training in safe and effective use of the methods involved, an approximately six-month training and development period was used, during four months of which the subjects served as technical aides in control studies and in adaptation of methods to the chamber environment. The subjects were therefore not naive concerning the objectives and methods of the study.

Certain control measurements of performance time, metabolic cost of work and ventilation were obtained underwater. Except where specifically indicated, the study was conducted in the respirable ambient environment, at one or more atmospheres of pressure and during transit from one ambient pressure to another.

Details of the methods utilized for each component project are contained in the specific section describing it (Section E). The summary of methods which follows here is offered as introduction and background for the subsequent general description of measurement integration.

TABLE I. Scope of Specific Studies

- Performance
  - Perceptual
  - Memory
  - Cognitive
  - Psychomotor
- Underwater Work
  - Performance
  - Metabolic Cost
  - Ventilatory Cost
- Neurological-Neuromuscular Function
  - Electroencephalogram
  - Evoked Potentials, Visual and Somatosensory
  - Tremor-Postural and Intentional
  - Postural Balance
  - Electromyography
  - Muscle Strength
- Visual Function
  - Acuity
  - Color Vision
  - Fields
  - Eye Movement
- Audio-Vestibular Function
  - Electronystagmometry
  - Audiogram
  - Vestibular Reactivity
- Vocal and Speech Function
- Metabolism
  - O_2 Consumption
  - CO_2 Production
  - Respiratory Exchange Ratio
  - Weight
- Respiratory Function
  - Resting Ventilation
  - Exercise Response
  - Diaphragmatic Electromyograph
  - End-Tidal P_{CO_2}, P_{O_2}
- Pulmonary Function
  - Maximum Voluntary Ventilation
  - Forced Vital Capacity
  - Inspiratory Capacity
  - Lung Compliance
  - Esophageal (Intrathoracic) Pressure
- Cardiovascular Function
  - Electrocardiograph
  - Cardiac Rate and Output
  - Exercise Response
  - Orthostatic Reflex
- Renal Function
  - Urine Volume
  - Electrolyte Composition
  - Endocrine Composition
- Hematology and Blood Chemistry
  - Cells, Hemoglobin
  - Electrolytes, Enzymes
- Endocrine Function
  - Serum Hormones
- Temperature
  - Deep Body
  - Skin
  - Environmental

PERCEPTUAL, MEMORY, COGNITIVE AND PSYCHOMOTOR PERFORMANCE

A minicomputer was programmed to administer selected sequences of Psychomotor Tests, Perceptual Tests, and Memory and Cognitive Tests, which were presented in fixed time sequences, separately to the "rest" subject and the "exercise" subject of each compression pair. The subject's responses to the tests were stored for concurrent scoring and subsequent statistical analysis and printout.

EXERCISE

Following determination of physiological responses to exercise up to "maximal work capacity" during air breathing at one ata, a level of approximately one-third maximal on a bicycle ergometer was chosen: a) for determination of muscular coordination and joint effects in compression; b) for physiological examination of respiratory-cardiac-metabolic relationships; and c) as a work level reference for subsequent practical underwater work performance.

MUSCULOSKELETAL FUNCTION

Muscle strength of coordinated respiratory musculature was measured by intra-esophageal pressure transducer as maximum inspiratory force and expiratory force generatable in voluntary effort. Grip strength of the hand was also employed.

Documentation of joint dysfunction in pressurization was by symptomatic appraisal.

ELECTROENCEPHALOGRAPHY

Multi-electrode recording on strip chart and on magnetic tape was continuous during all periods of compression, in exercise and rest subjects of each compression pair, using adhered EEG electrodes in the

0-800-1200-foot pressurizations and needle electrodes in the 0-800-1200- to 1600-foot phase.

VISUAL AND SOMATOSENSORY EVOKED RESPONSES

Studies of visual and somatosensory evoked responses employed the EEG electrodes for measurement of cortical evoked potentials.

TREMOR

Intentional tremor was recorded as movement of the middle finger pressing with fixed force against a strain-gauge sensor. Postural tremor measurement (accelerometer) recorded finger movement with the arm held horizontally and unsupported.

Electromyography used to monitor for spontaneous "tremor" of functioning muscle included recording of diaphragmatic action potentials in the "exercise" subject (via esophageal electrode) and search for tremor of ocular muscles during electronystagmometry.

VISUAL FUNCTION

Measurements of visual function which were made during periods of stable pressure, prior to or following a compression (e.g., at 0-800-1200-1600 fsw), and not during dynamic change in pressure, utilized standard methods adapted as necessary for monitored self-administration by the subjects.

AUDITORY FUNCTION

Audiogram determination was performed at stable pressures during periods of silenced chamber activity. Bone conduction methods were used to avoid influences of pressure (density) upon the gas conduction auditory systems of the subject and on electroacoustic transducers (earphones) (15).

BALANCE AND VESTIBULAR FUNCTION

In order to detect changes in vestibular stimulus threshold, the balance function study combined measurement of postural sway in the subject a) while standing in the erect position on a statometer (balance board), with eyes open and with eyes closed, and b) during electrical stimulation of vestibular apparatus while standing on the statometer. During compression, electronystagmometry was continuous. Responses to caloric and rotatory stimulation of vestibular function were performed at one ata before and after exposure phases of the study.

SPEECH FUNCTION

Speech, as an index of complex neuromuscular and centra function was studied during stepwise compression by evaluation of operational intercommunications. Speech intelligibility over the range of 200 to 1600 fsw was evaluated by analysis of the intelligibility of preassigned work lists recorded via wide bandwidth condenser microphones.

RESPIRATORY FUNCTION

Respiratory relationships to metabolic activity levels and alveolar gas composition were measured at rest and during exercise in order to determine influences of compression upon a) sensors concerned with respiratory control, and b) metabolic cost of external work of the voluntary neuromuscular system. External work was accomplished with a bicycle ergometer and ventilation was measured with a high-capacity pneumotachograph. Measurements of breathholding capacity and ventilation, alveolar gas composition and gas exchange at stable pressures from 1200 fsw to 1 ata were included to examine effects of pressure on the control of respiratory drive.

PULMONARY FUNCTION

Using intrathoracic pressure measurement (esophageal sensor) and a "dry" spirometer, recording of pulmonary ventilatory and mechanical functions provided measures of respiratory muscle function during acute compression, and of changes in function during prolonged exposure. Measurements made throughout multi-day decompression after adaptation to acute effects of compression provided a density-related reference for evaluation of acute effects.

CARDIOVASCULAR FUNCTION

The electrocardiogram, atrial conduction and cardiac ejection times, and cardiac rhythmicity were monitored continuously in both rest and exercise subjects during changing pressures. In addition, the electrical impedance method was used to measure cardiac stroke volume. Monitoring of these variables after abrupt postural change from seated to standing position provided an index of orthostatic (baroreflex) response to gravitational stress. Monitoring during exercise related cardiac, respiratory, metabolic and work functions.

METABOLIC ACTIVITY

Metabolic adaptation to the thermal stress of high helium pressure at rest and metabolic cost of physical work were studied by measurement of oxygen consumption and carbon dioxide production. For metabolic and ventilatory costs of practical work underwater, measurements of oxygen consumption, carbon dioxide production and respiratory minute ventilation were made, prior to and after compression studies in subjects performing the same work task underwater.

TEMPERATURE

With controlled ambient temperature during compression, rest and exercise subjects were studied by measurement of

deep body and skin temperatures. Thermal stress in relation to caloric balance was determined for stable saturation exposure periods.

HEMATOLOGICAL, ENDOCRINE AND ELECTROLYTE FACTORS

Before, during and after saturation periods, examination of blood cellular and chemical composition was made. Measurements encompassed proteins, electrolytes, endocrine and clotting factors. For selected 24-hour periods of the overall pre-, during and post-compression period, urine volume and composition were measured, aiding study of electrolytes and sympathetic-adrenal function.

UNDERWATER WORK PERFORMANCE

Assembled components of an undersea oil wellhead installed in the water-filled chamber provided the test system for underwater work performance at pressures equivalent to sea level, 1210, 1360 and 1610 fsw. Timing and motion were photographically documented for comparison with prior measurements at sea level. Work level, at predetermined levels of oxygen consumption and ventilation, was related to exercise capacity measured on each subject prior to compression. Ventilatory gas was supplied by low-resistance open-circuit respiratory systems.

DECOMPRESSION

Accelerated decompression times required for physiological and performance studies during the 400-fsw excursion-compressions were derived by projection from existing observations of smaller excursions to lesser pressures. Validation of the concept was by stepwise increase in exposure duration in excursions from 800 to 1200 fsw. The procedure derived for 800- to 1200-fsw excursions was employed for the deeper 1200- to 1600-fsw excursions. Monitoring during decompression included Doppler sensing for bubble transit in the heart and large peripheral veins.

SPECIAL ANCILLARY STUDIES

Study of isobaric counterdiffusion in animals was performed as a cross-link to the previous observation of cutaneous gas lesions encountered during crude neon breathing by human subjects at 1200 fsw [isobaric inert gas counterdiffusion, Predictive Studies III (5,8,9)]. Circumstances equivalent to those in which the isobaric counterdiffusion syndrome was encountered in man were provided for study of the cutaneous gas lesion phenomenon in piglets breathing crude neon during stable exposure to 1200 fsw. The study, aided technically by the diver-subjects within the chamber, included measurement of neon passage across the skin as well as direct visual observation of dermal and vascular changes.

COMPRESSION RATE PLAN--
CHOICE OF THE EXCURSION-SATURATION PROCEDURE

STUDY PHASES I AND II

The Predictive Studies Program was planned for investigation of effects of rapid compression and adaptation to compression effects. This dual purpose required both excursions and prolonged stays at increased pressure. Selection of an appropriate excursion-saturation pattern was therefore a major element of the investigations. Choice of actual profiles was made essentially as follows:

a. Pressure for the final, prolonged, deep saturation period was selected first, to be the 1200-fsw pressure equivalent intensively studied in the previous Predictive Study (III) (8).

b. Since no rapid excursions to pressures higher than 1200 fsw had been made previously, it was elected to design the overall experimental sequence in two parts, to be performed in two steps over several months. The first part was planned as an initial investigation of excursion, also to a maximum of 1200 fsw. This was intended to provide cross-relation of any observed changes during or following rapid compression to the essential absence of effects on slow attainment of this saturation pressure (7).

c. Uncertainty concerning rate of adaptation to the desired rapid initial excursion depth of 1200 fsw, together with interest in the practical concept of saturation-excursion diving, led to decision to use the lesser pressure of 800 fsw as a pressure level which would likely be a symptom-free base for saturation, and at which prior investigation elsewhere had indicated that initial compression effects during saturation should not be severe. From this pressure, any adaptations during repeated excursions to the 1200-fsw pressure could be studied.

d. Plans and detailed experimental procedures were prepared in full for a subsequent and separate exposure series equivalent in design, but to involve remaining at 1200 fsw on acute compression, with repetitive excursion thereafter to the 1600-fsw pressure equivalent. Choice of the 1600-fsw pressure was made to provide exaggeration and an opportunity for tracking of effects observed by others at approximately this pressure during slower compression or saturation exposure (2,3,10,12).

e. Plans for return to saturation levels after each of the repetitive 400-fsw excursions provided for monitoring the long-term, day-by-day adaptations.

For the overall Predictive Studies IV program, therefore, two separate and related compression study phases were designed. These are designated Phases I and II.

Phase I

0-400-800-1200 fsw excursions in contiguous sequence, with return from 1200-fsw to 800-fsw saturation pressure, followed by repeated excursions from 800 to 1200 fsw.

Phase II

0-400-800-1200 fsw excursions, with repeated further excursions from 1200 to 1600 fsw and return to saturation pressure of 1200 fsw.

The initial sequence, Phase I, was performed not only as a full investigation in its own right, but also as a preliminary validation of the appropriateness of compression profiles, the precision of experimental methods and the

adequacy of decompression plans for the greater pressures of Phase II.

COMPRESSION RATES

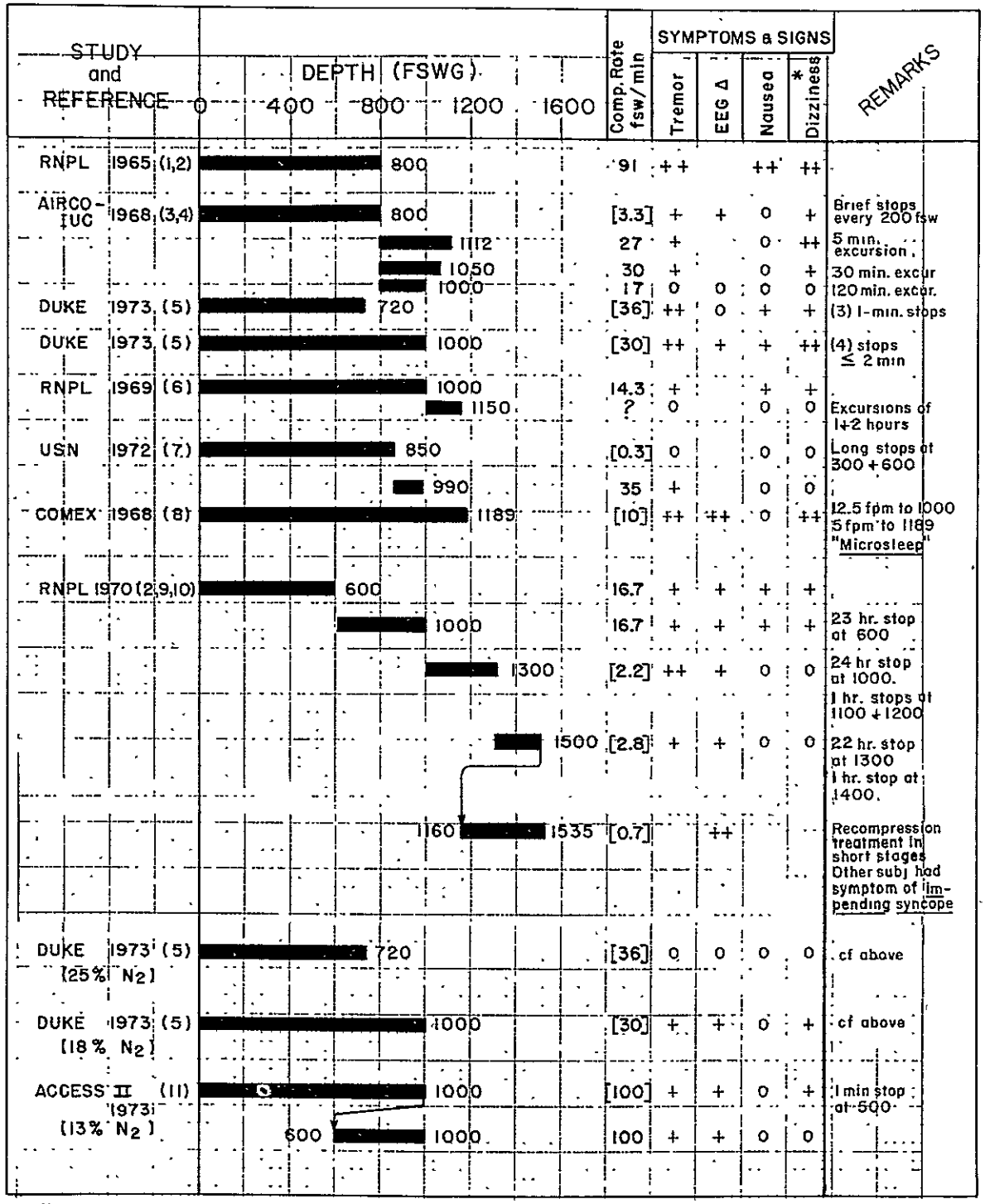
Appraisal of effects induced in man in short-term (excursion) and long-term (saturation) exposures to increased pressure was based upon existing published and unpublished documents, clarified by extensive discussions with individuals who had direct experience with high-pressure exposures. The special direct contributions to be acknowledged include those of X.R. Fructus [studies at Comex--Compagnie Maritime d'Expertises (10,11)], P.B. Bennett and H.V. Hempleman [studies by Royal Naval Physiological Laboratory (2)], R.W. Hamilton [studies by Samson-Ocean Systems, Inc. (6)], and W.H. Spaur [studies by U.S. Navy Experimental Diving Unit (13)].

Table II with its accompanying references summarizes the status of information available, during the planning of Predictive Studies IV, concerning compression effects upon man. From this and from extensive information at

TABLE II. Numbers in parentheses represent sources of information relating to specific compression studies which encountered compression effects. Indications of presence of effects (0, +, ++) represent occurrence but not degree.

References: (1) Bachrach,A.J. and P.B. Bennett. Tremor in diving. Aerosp. Med. 44:613-623, 1973. (2) Bennett, P.B. and E.J. Towse. Performance efficiency of men breathing oxygen-helium at depths between 100 feet and 1500 feet. Aerosp. Med. 42:1147-1156, 1971. (3) Galerne, A. International Underwater Contractors, Inc. Personal communication. (4) Proctor, L.D., C.R. Carey, R.M. Lee, K.E. Schaefer and H. van den Ende. Electroencephalographic changes during saturation excursion dives to a simulated seawater depth of 1000 feet. Report 687. Groton, Ct.: Naval Submarine Medical Research Laboratory, 1971. 24 pp. (5) Bennett, P.B., G.D. Blenkarn, J. Roby and D. Youngblood. Suppression of the high pressure nervous syndrome in human deep dives by He-Ne-O_2. Undersea Biomed. Res. 1: 221-237, 1974. (6) Buhlmann,A.A., H. Matthys, G. Overrath, P.B. Bennett, D.H. Elliott and S.P. Gray. Saturation exposures at 31 ata in an oxygen-helium atmosphere with excursions to 36 ata. Aerosp. Med. 41:394-402, 1970. (7) Greene, K.M. Data on file in International Decompression Data Bank, Institute for Environmental Medicine. (8) Brauer, R.W. Seeking man's depth level. Ocean Ind. 3: 28, 1968. (9) Bennett, P.B., J. Morrison and J. Eaton. Interim report on some physiological studies during 1500 ft. simulated dive. Royal Naval Physiological Laboratory Report IR 1/70. (10) Leitch, D.R. Medical aspects of a simulated dive to 1,500 feet (458 metres). Proc. R. Soc. Med. 64: 1273-1276, 1971. (11) Hamilton,R.W., Jr., T.C. Schmidt, D.J. Kenyon and M. Freitag. Access: Laboratory dives to 1000 feet using both direct compression from the surface and excursions from saturation. Environmental Physiology Laboratory Technical Memorandum CRL-T-773. Tarrytown, N.Y.: Union Carbide Technical Center, 1974. 122 pp.

TABLE II. Summary Results of Major Compression Studies Used in Design of Predictive Studies IV. (See legend on preceding page for references to studies.)



KEY:

A. BRACKETS AROUND COMPRESSION RATES INDICATE EFFECTIVE RATE WHEN STOPS WERE USED

B. SYMPTOMS AND SIGNS: 0 = OBSERVED TO BE ABSENT, + = MILD TO MODERATE, ++ = MARKED

C. REFERENCES LISTED IN LEGEND

* DIZZINESS REFERS TO LIGHTHEADEDNESS AND DISEQUILIBRIUM, BUT NOT VERTIGO

lesser pressures it was judged that: a) even extremely rapid compression to a pressure equivalent to 400 fsw, such as that used in investigating submarine escape procedures, would induce no consistently measurable or unmanageable effects; b) rapid compression to an 800-fsw pressure equivalent does produce changes and, therefore, continuous compression to still higher pressures would necessarily induce still more prominent effects; and c) rapid, symptom-free compression to an intermediate pressure such as 400 to 800 fsw should contribute to the development of derangements in any immediately subsequent excursion-compression to a higher pressure.

Desiring to induce demonstrable compression effects of definite and measurable degree, it was planned to establish compressions in increments of 400 fsw, changing rates of pressurization during these compressions to allow reaching the high maximum pressures selected. Rates of compression were chosen to provide the probability that prominent but tolerable effects would develop. The capacity of the subjects to tolerate induced disruptions was considered necessary to allow study of adaptation and to avoid any necessity for cancellation of the planned investigative programs due to effects of excessive rate or degree of compression.

Table III shows the compression pattern selected initially for Phase I (400-800-1200 fsw). Since these rates were considerably faster than those previously employed for compression to 1200 fsw, it was expected that they would possibly be excessive for subsequent application in the Phase II Study. It was eventually learned, due to the minimal degree of compression effect on the four subjects of Phase I, that a still faster compression to 800 fsw was desirable to produce effects prominent enough for investigative purposes. The table also shows the compression rates selected on this basis for Phase II.

PRESSURE-DURATION PROFILES AND SUBJECT PAIRS

Figure 1 shows the overall profiles of compression and the time scales for the two Phases of the study. Two subjects could be monitored at once. Therefore, to provide

TABLE III. Compression Rate Profiles for Phase I and Phase II

DEPTH (fsw)	PHASE I		PHASE II			
	COMPRESSION RATE (ft/min)	COMPRESSION DURATION (min)	COMPRESSION RATE (ft/min)		COMPRESSION DURATION (min)	
0-400	20	20	32		12.5	
400-600	10	20	16		12 5	
600-800	5	40	8		25	
	2 HOUR HOLD		2 HOUR HOLD			
800-1000	20	10	20		10	
1000-1100	10	10	10		10	
1100-1200	5	20	5		20	
			22 HOUR HOLD			
			EXCURSIONS		EXCURSIONS	
			INITIAL	LATER	INITIAL	LATER
1200-1400	—	—	(20)	40	(10)	5
1400-1500	—	—	(10)	20	(10)	5
1500-1600	—	—	(5)	10	(20)	10

The dual profiles for Phase II in excursion from 1200 to 1600 fsw represent the planned "initial" compression rates (used for the first Subject Pair on its first 1200 to 1600 fsw excursion) and the faster "later" rates selected for all subsequent excursions when it was learned that the "initial" rate induced no prominent symptoms.

for increased number of subjects and for investigation of each subject during initial compression and adaptation over the first three days of exposure, each Phase of the study was performed twice. This was accomplished by designated Subject Pairs (one "rest" subject and one "exercise" subject constituting a Pair). For example, in Phase II one Subject Pair was compressed and studied in initial excursions for three days, then held at the stable saturation pressure in a living compartment while the experiment chamber was brought to atmospheric pressure and then again used for a three-day compression study of the second Subject Pair. Thereafter, throughout the saturation exposure and decompression, the subjects followed a predetermined schedule of measurements. This procedure, overlapping two compression studies in each Phase, was utilized to expand the subject population to four in Phase II, as well as in Phase I.

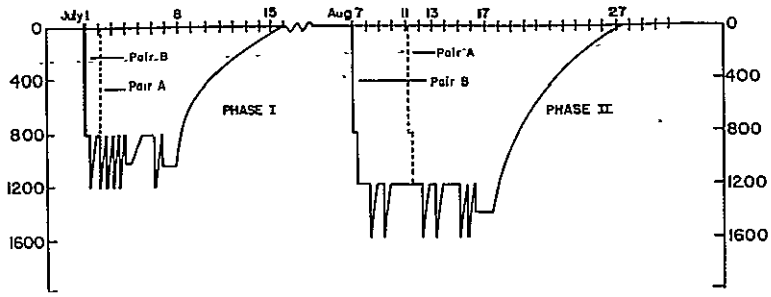


FIG. 1. Pressure Duration Profile Plan for Phase I and Phase II of Predictive Studies IV. Deep excursions represent compression for monitoring Subject Pairs. Additional excursions represent deviation from plan, to accomplish therapy of decompression incidents.

MEASUREMENT MODULE SYSTEM

To study effects of compression as they developed and during adaptations at stable pressures required means for making measurements rapidly and repeatedly throughout and following the periods of rapid compression. This was accomplished by designing measurement systems and experiment procedures for integration into a 17-minute-long "Measurement Module" (Module 1, Fig. 2) which encompassed most of the investigations shown in Table I. Additional measures, to be carried out only during periods of stable pressure, were combined into an "Ancillary Module" 12 minutes long (Module 2, Fig. 2). Combined training of subjects, investigators and operations support staff provided successful timed repetition of the entire measurement pattern with subjects of each Pair appropriately sharing and alternating methods, apparatus and investigator attention. In this manner, each "Rest Subject" and each "Exercise Subject" were tracked, step by step, throughout compressions, stable excursion pressures and decompressions, for investigation of the dynamic influences of compression.

The pattern for performing the modules of measurement is shown in Fig. 3 as the rectangles superimposed on the pressurization profile of the first three days of Phase II. It was mentioned above that in each Phase (0-800-1200 fsw and 0-800-1200-1600 fsw) it was considered that the early

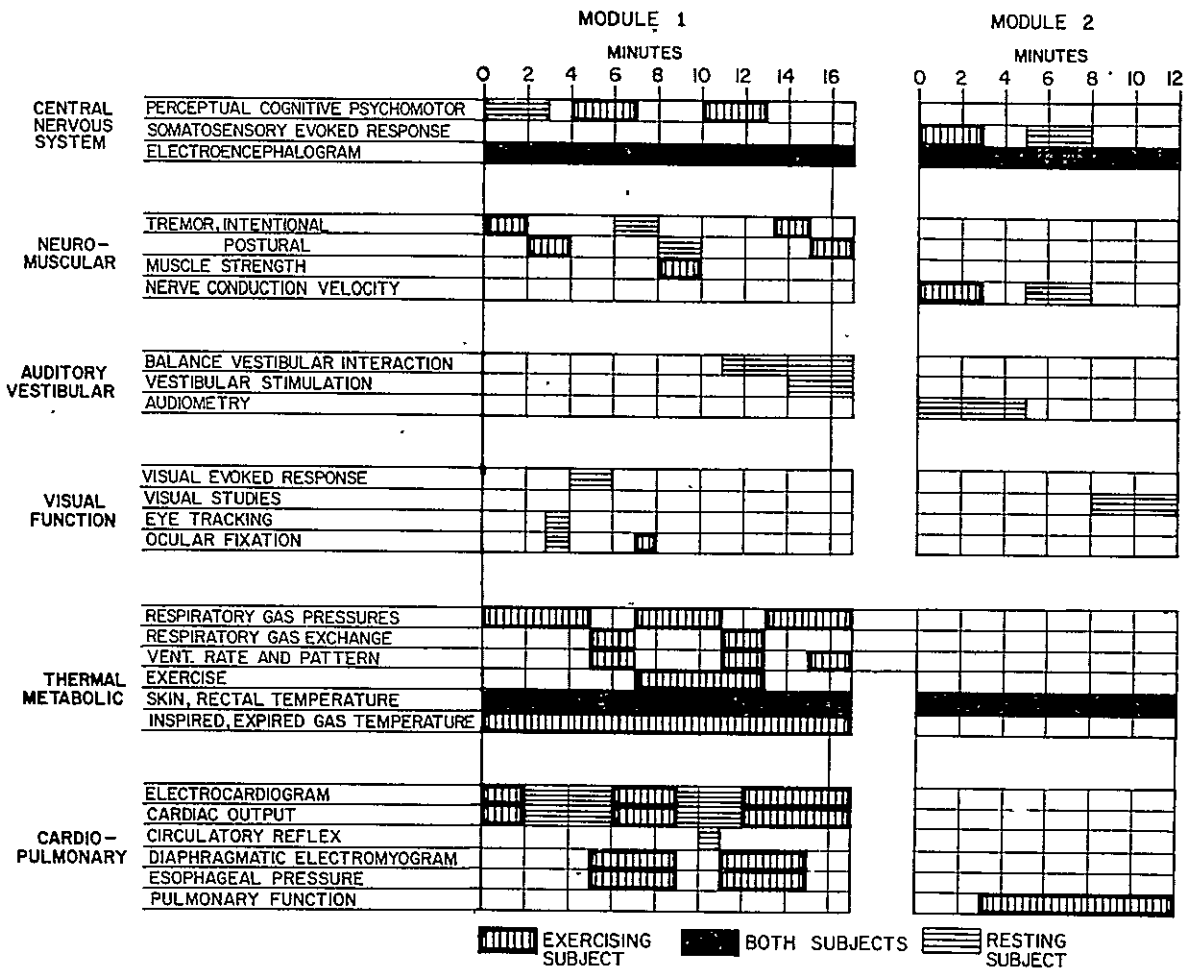


FIG. 2. Synchronization of Experiments by Use of Measurement Modules. Measurements performed during excursions are shown under Module 1. Studies requiring sustained attention and/or active procedures by the subject were performed under Module 2 in holding periods at constant pressures (800, 1200, 1600 fsw pressure equivalents).

(pre-adaptive) periods of compression would provide the most critical information. Therefore the two (exercise and rest subjects) under study were compressed according to the sequence illustrated in Fig. 3 for Phase II and were studied over a three-day period before they stepped aside at the saturation pressure, and the second pair of subjects was similarly compressed to duplicate the observations.

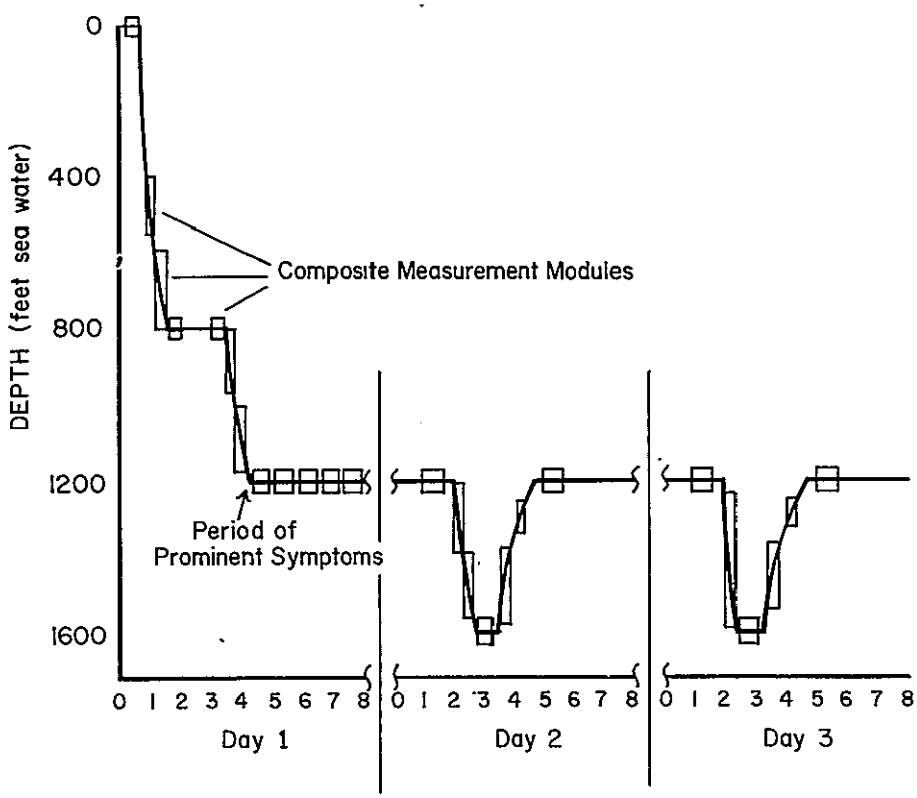


FIG. 3. Relation of Experiment Modules to Phase II Compression Profile for Subject Pair. Indicated "Period of Prominent Symptoms" represented both plan and findings.

Modules used for Phase II differed slightly in detail of sequence, but not in method, from those used for Phase I.

The Measurement Module system was also employed during experiment days of the saturation exposure. In addition, studies of thermal stress, renal, hematological, respiratory, speech and some pulmonary functions were conducted at stable saturation pressures.

SUBJECT-DIVERS AND TRAINING

Six male subjects were selected from eleven professional offshore industry diver volunteers, with the intent of using the same four primary subjects throughout all phases of the study. The two additional subjects were intended to serve as standbys in the event of illness of a primary subject. In the course of the study it became necessary to use in Phase II the two subjects who had not participated in Phase I.

Selection of subjects was based on direct observation of purposefulness and dedication in work circumstances, as well as upon medical and psychological testing. It was not based on specific physiological performance, which was determined after the selection process.

The physical and related characteristics of the six subjects are shown in Table IV. None had served previously for detailed physiological experiments, although one (FS) had been a subject for decompression trials elsewhere.

TABLE IV. Subjects

Subject	Age (yrs)	Height (cm)	Weight (kg)	Surface Area (m^2)	Participation	
					Phase	Subject Pair
WS	24	191.0	85.0	2.066	I	B
LJ	29	178.0	72.4	1.844	I	A
CC	25	180.0	82.6	1.964	I,II	A,A
FS	25	169.5	72.5	1.816	I,II	B,B
GM	23	183.0	81.0	2.036	II	B
MP	29	173.0	75.8	1.964	II	A

Training as subject-technicians covered a period of approximately four months leading up to the beginning of Phase I. This initial training was designed to provide competence in application, use and maintenance of the multiple measurement systems within the chamber; familiarization with blood sampling and subject physiological electrode and sensor systems placement; equivalent familiarity with life-support, safety, communications and other aspects of the environmental chamber systems; and practice in underwater work performance, timed execution of the measurement modules and conduct of ancillary animal experiments.

Figures 4 through 7 show aspects of subject-diver activity during the actual deep saturation-excursion exposures.

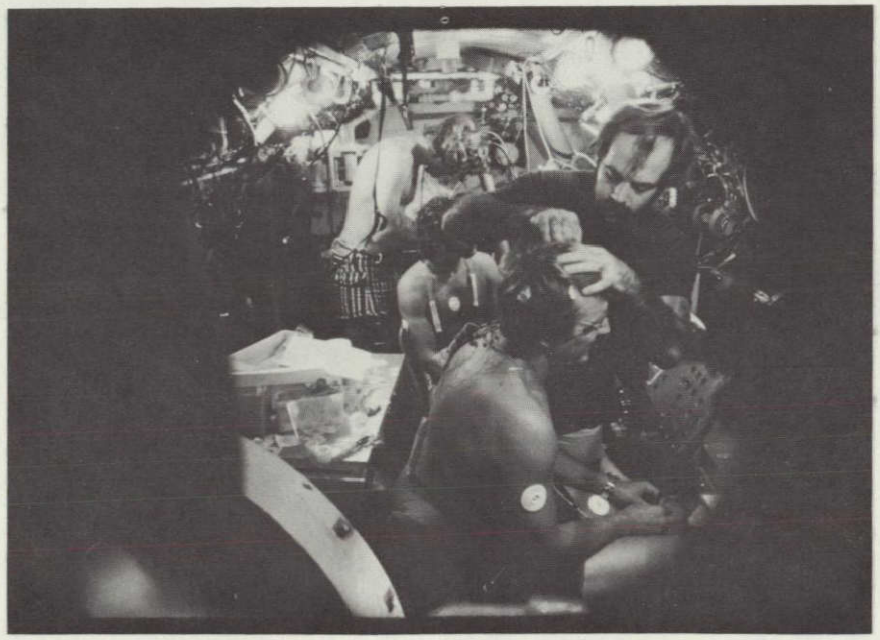


FIG. 4. Morning preparation at 1200 fsw of subjects with sensors for electrophysiological measurement during excursion from 1200 to 1600 fsw.

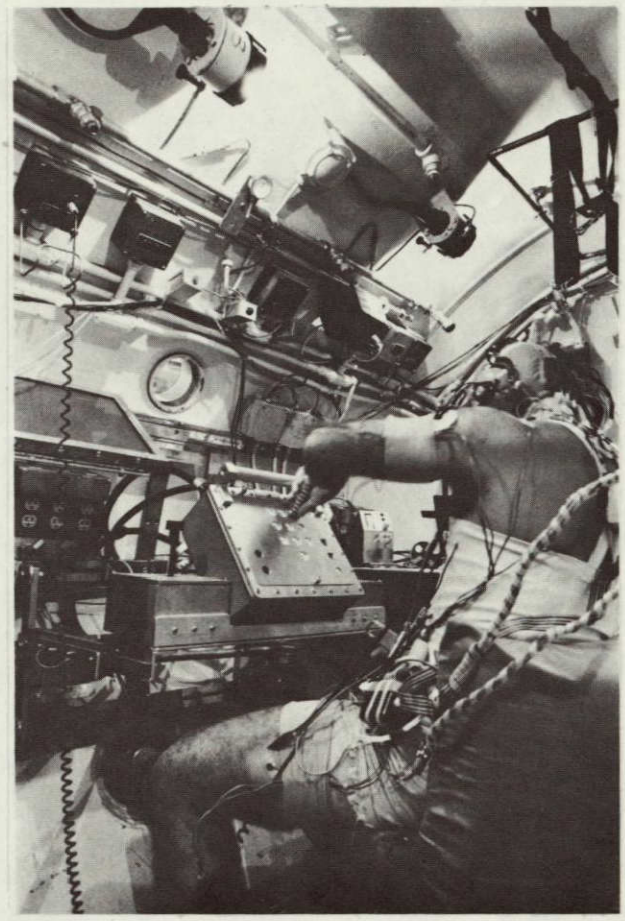


FIG. 5. Subject FS engaged in concurrent studies of perceptual, memory, cognitive and manipulative performance during compression from 1200- to 1600-fsw pressure equivalent.

FIG. 6. Subject FS performing bone conduction audiometry on excursion to 1600-fsw pressure equivalent, signaling to controlling external investigator that sound is heard. Ergometer system in background. Somatosensory evoked response being elicited in Subject GM.

FIG. 7. Subject standing abruptly on statometer platform for simultaneous measurement of postural sway and cardiac postural reflex changes, during compression from 1200- to 1600-fsw pressure equivalent.

ORGANIZATION FOR COLLABORATIVE EXPERIMENTS

For the purposes of design, development, instrumentation and training, senior investigators of the Institute staff formed a Planning Group. Each member of the Planning Group was also responsible for aiding the experiment integration and performance of studies by investigators from collaborating laboratories external to the Institute.

Related projects, including those sharing or alternating use of particular equipment, were grouped to facilitate direct contact and communication among investigative teams (Fig. 8).

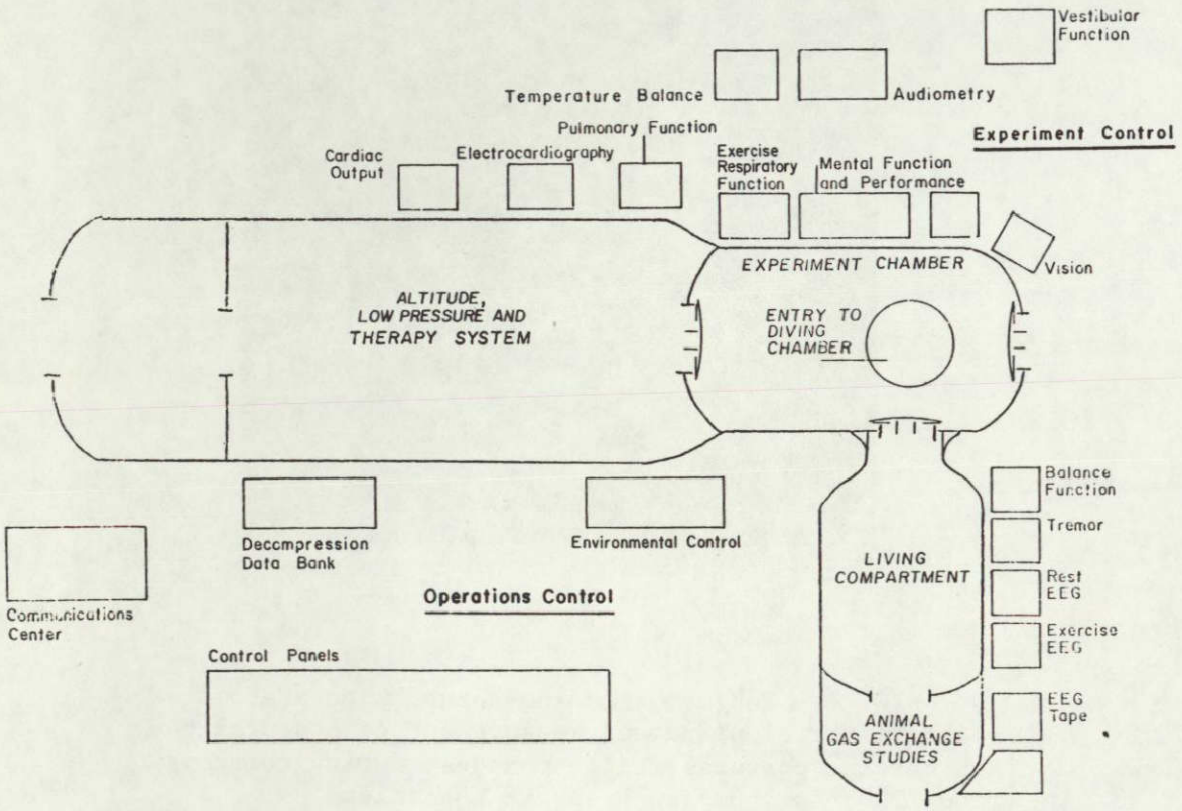


FIG. 8. Arrangement of Chamber Systems, Experiment Stations and Operations Control.

Measurement functions were located along one rim of the environmental chamber system (shown in part in Fig. 9), with operational support functions grouped separately, (Fig. 10). This permitted direct attention to measurement by the investigative teams, and to operational safety and environment control by the operations staff. Video and audio communication existed between these functions.

For all the concurrent investigations of the compression study, one individual (Experiments Coordinator) served to conduct subjects and investigators through the hours of sequential measurement procedures. A counterpart (Operations Coordinator) provided the direct control of predetermined pressurization rates, gas environmental control, life-support, safety and documentation of environmental conditions, in precise communion with the Experiments Coordinator. A senior investigator familiar with instrumentation and chamber systems (Instrumentation Engineer) freely aided other investigators and operations staff as necessary in overcoming technical problems.

Groups of studies (neurophysiological, performance, temperature, respiratory or pulmonary) were aided by investigator members of the Planning Group.

A major element in establishing understanding of procedures, safety measures and technical performance was the extensive training mentioned earlier, of the six-man subject group in the details of both experiment and operational support.

Following the performance of measurement sequences (modules) throughout each experiment day, a limited operations, medical and safety team monitored and maintained the subjects and chamber systems throughout the night.

DECOMPRESSION SYSTEM

Three patterns of decompression procedure were devised in support of the Predictive Study, as described in Sections G-1 and G-2. These included:

a. Emergency decompression procedures covering the possible requirement for prompt return to atmospheric

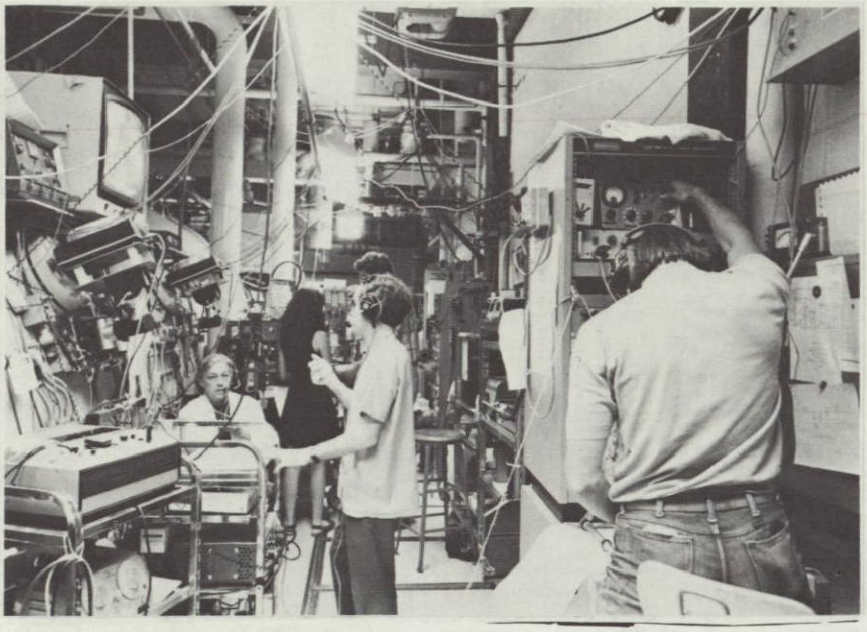


FIG. 9. Experiment Control and Recording Stations External to Chambers.

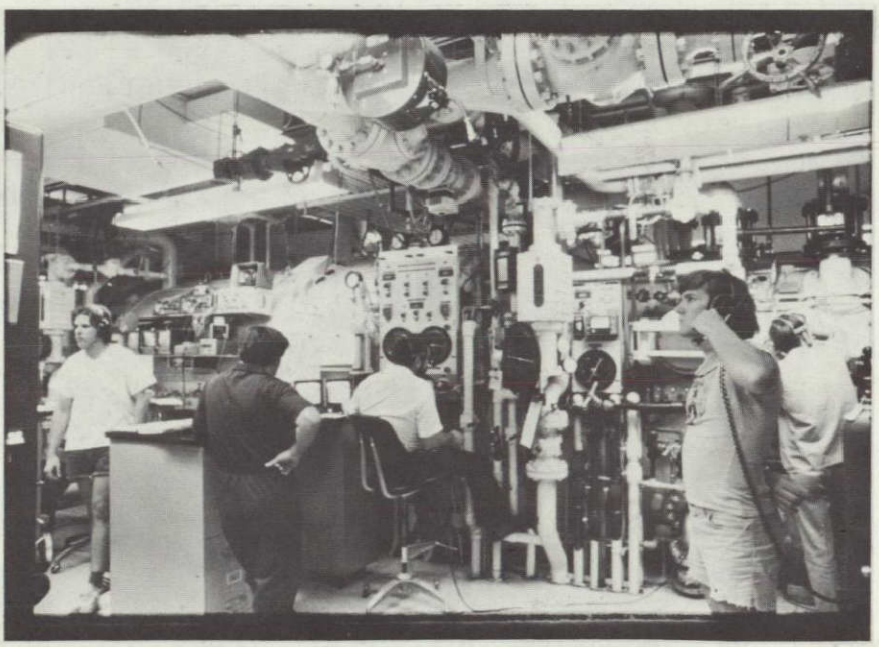


FIG. 10. Chamber Compression, Environment Control, Decompression and Operations Stations.

pressure during initial compressions from atmospheric pressure. These covered the range of 0 to 800 fsw and 0 to 1200 fsw.

b. Saturation-decompression procedures for slow, post-experiment return to atmospheric pressure from the planned periods of saturation exposure at pressures equivalent to 800 and 1200 fsw.

c. Excursion-decompression procedures for use in "deeper" excursions from a high-pressure, saturated state.

Efficient decompression from deep excursions was a requirement for performance of the compression studies. It was considered desirable to perform a full measurement sequence on successive days with a pressurization equivalent to 400 fsw. Since such an excursion from atmospheric pressure, of more than 1 hour duration, customarily requires approximately 18 hours of decompression time, application of existing decompression procedures was not practical.

The general impression has existed for many years that overt bends is less likely to develop while divers decompressing from excursions from sea level are still at high pressure than when they are approaching atmospheric pressure. Important studies of this phenomenon in the British Royal Navy (1) and the U.S. Navy (4,14) provided important empirical information on decompression tolerance from saturation exposures at increased pressures to 750 fsw. By analysis and adaptation (Section G-1), such information was used to plan procedures for study of saturation-excursion decompression in transient exposures from 800 to 1200 to 800 fsw and from 1200 to 1600 to 1200 fsw. The plans developed involved 20-minute compression and 55-minute actual working time at peak pressure, with a decompression of 1 1/2 hours, as contrasted with the 18-hour requirement for equivalent excursion from the surface (Fig. 11).

Phase I of the fourth Predictive Studies program had as a major purpose the systematic trial of this large deviation from previous decompression procedure (Section G-1). It was assumed that successful excursion-decompression between 800 and 1200 fsw would be at least

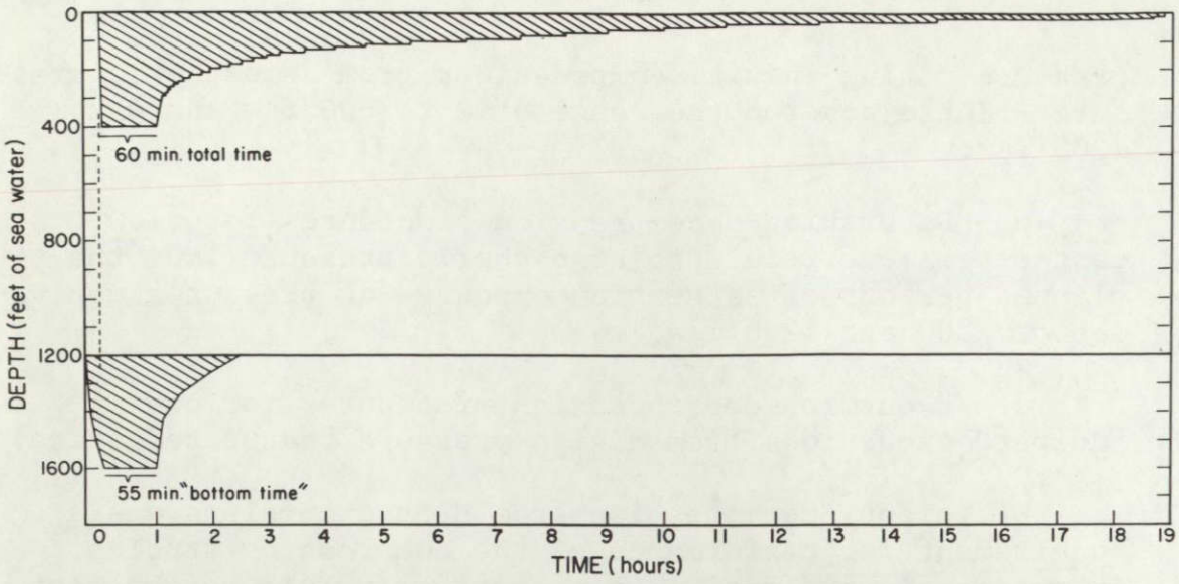


FIG. 11. Summary Illustration of Decompression Plan. Comparison of decompression in excursions from one atmosphere and excursions from high saturation pressure.

as effective in deeper excursions of 1200 to 1600 fsw planned for Phase II as in the lower pressure circumstances of Phase I.

To aid in evaluation of these novel excursion-decompressions, a Doppler ultrasonic bubble detector was employed in the 1600 to 1200 fsw decompressions to search for evidence of circulating gas bubbles (Fig. 12).

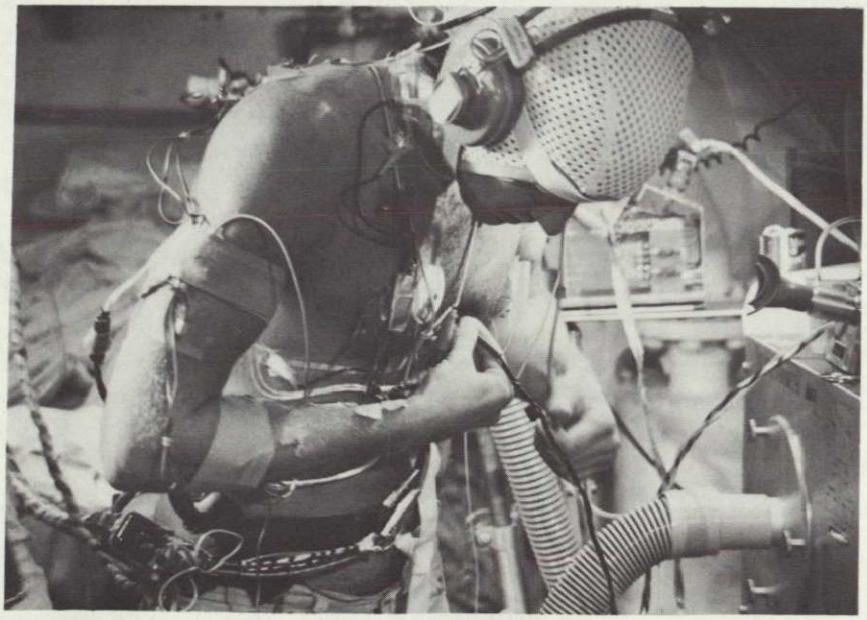


FIG. 12. Subject GM, applying Doppler transducer sensor for bubble detection over pulmonary artery, during excursion-decompression from 1600 to 1200 fsw.

experiment-related items of small size. The entrance lock of the Living Chamber was used for transferring larger items of equipment, for conducting the animal studies at 1200 fsw, and as an emergency entry into the pressurized system.

Water for washing and showering and for operation of the sanitary waste disposal system in the Experiment Chamber was provided as needed by high-pressure hot and cold water supply pumps controlled by chamber operations personnel.

OPERATIONS TEAM

The operations team for experiments consisted of an operations director, a medical supervisor, a primary chamber operator, two or more assistant chamber operators (depending on the complexity of compression procedure), and an environmental data recorder. During experiments, operational procedures had to be precisely coordinated with the experiment protocol. Because it was required that operational and investigative personnel and subjects all be constantly aware of the status of both operational and investigative maneuvers, a common or "party-line" intercommunications sytem was used. Speech originating inside the chamber and distorted by the helium environment was channeled through a helium speech processor before being mixed into the common power amplifier with the unprocessed speech originating outside the chambers. The electronically improved helium speech was thereby also returned to the subject teams in the chambers.

The operations team was responsible for control of chamber environmental conditions--compression and decompression, electrical requirements, lighting, oxygen level, carbon dioxide level, odor control, temperature and humidity control--and for the operation and maintenance of all other chamber and life-support systems relating to the needs and safety of the subjects.

ENVIRONMENTAL CONTROL: PRESSURE

Compression from one atmosphere was accomplished as follows. With the hatches secured, chambers were

pressurized to 10 fsw with air. This step provided positive closure of the hatch seals, assuring that any temporary initial leaks could not lower oxygen tension by displacement of air with pure helium. Subsequent compression was accomplished with pure helium, admitted to the chambers via a silencer for noise suppression.

Chamber pressure was manually controlled by throttling valves, with the valve input pressure set by remote-loaded high-flow reducing regulators. Helium was stored in mobile gas tube trailers located outside the building housing the pressure chambers.

Chamber pressure was measured and visually monitored by Bourdon tube pressure gauges (Heise) with calibration precision of ±0.1% of full scale (±1.8 fsw) and readability (resolution) of ± 1 fsw.

ENVIRONMENTAL CONTROL: OXYGEN

Chamber oxygen concentrations were continuously monitored by paramagnetic oxygen analyzers (Taylor-Servomex OA137). Digital voltmeters were used to give visual digital displays of 0%-10.00% or 0%-100.0%, as appropriate. Oxygen concentration of the Experiment Chambers was also recorded on a strip chart recorder and monitored by high-low limit alarms.

The gas from the chambers was sampled from within and delivered to the oxygen analyzers by gas sampling systems, via low dead space pressure-reducing regulators mounted at the chamber hulls. Gas from the chamber pressure was thereby reduced to constant low pressure for delivery to the tubing which supplied the oxygen analyzers. Response time between change of oxygen concentration in the chambers to analyzer response of 80% of full scale was approximately 35 seconds.

Makeup oxygen was controlled manually by the chamber operators. Oxygen percentage for the different conditions was maintained within predetermined limits appropriate to each pressure and purpose (Table V). To provide for prompt mixing, oxygen was introduced into the chambers by means of venturi-type jet blowers. Each liter of oxygen

immediately mixed with approximately 15 liters of chamber gas as it entered the chamber via the jet blower. The resulting mixture, containing approximately 8% oxygen, then blended further with the chamber atmosphere. This method of introducing diluted oxygen into the chamber minimized the possibility of formation of a region of high oxygen pressure/concentration, with its concomitant hazard of fire or toxicity.

Oxygen pressure during all initial compression and stable pressure phases was maintained between the limits of 0.21 and 0.23 ata. During compressions (see Sections G-1 and G-2), oxygen pressure was increased (Table V).

TABLE V. Inspired Oxygen Exposure During Compressions, Stable Pressure and Decompression. Dry Chamber and Underwater Work (Phases I and II)

Experiment Condition		Gas Breathed	Total Pressure (ata)	Oxygen Partial Pressure (atm)	Oxygen Percentage (%)
Atmospheric pressure, sea level		Air	1.0	0.21	20.94
Compression to 800 fsw		$He-O_2$	1.0-25.2	0.21	-
Saturation at 800 fsw		$He-O_2$	25.2	0.21-0.23	0.83-0.91
Excursion to 1200 fsw		$He-O_2$	37.3	0.21-0.23	0.56-0.62
Excursion-decompression 1200 to 800 fsw		$He-O_2$	37.3-25.2	1.01-0.68	2.7
Saturation at 1200 fsw	Chamber	$He-O_2$	37.3	0.21-0.23	0.56-0.62
at 1210 fsw	Diving	$He-O_2$	37.6	0.38	1.0
Excursion to 1600 fsw	Chamber	$He-O_2$	49.4	0.21-0.23	0.43-0.47
at 1610 fsw	Diving	$He-O_2$	49.7	1.00	2.0
Excursion-decompression 1600 to 1200 fsw		$He-O_2$	49.4-37.3	1.58-1.19	3.2
Saturation-decompression	Phase I	$He-O_2$	-	0.49-0.51	-
	Phase II	$He-O_2$	-	0.50-0.52	-
Diving during decompression at 1360 fsw		$He-O_2$	42.2	0.54	1.3

During compressions with pure helium, prompt and verified mixing of the incoming gas with the chamber contents was accomplished by the internal heat exchange blowers and by the external loop blowers used for carbon dioxide removal.

ENVIRONMENTAL CONTROL: CARBON DIOXIDE

Carbon dioxide concentrations in the three high-pressure chambers were continuously monitored by three infrared carbon dioxide analyzers (Mine Safety Appliances models 200 and 300) and were maintained at less than the sea level equivalent of 6 mm Hg. Chamber gas was delivered to the carbon dioxide analyzers by the same sampling systems employed for the oxygen analyzers.

Carbon dioxide was removed from the chamber atmospheres by external loops containing squirrel cage blowers (approximately 150 cubic feet/minute), canned rotor electric motors and baralyme canisters with a capacity of 40 pounds. The baralyme charge was replenished at approximately 50-hour intervals, when the continuous carbon dioxide analysis indicated a rise from the stable level.

ENVIRONMENTAL CONTROL: TRACE CONTAMINANTS AND ODORS

Odors and trace contaminants were scrubbed from the sealed chamber atmospheres by a one-hour daily substitution of Purafil-filled canisters (Purafil, Inc.) in place of the baralyme canisters in the external CO_2 scrubbing loops.

The Experiment and Living Chambers were continuously monitored for carbon monoxide (Beckman model 315A, 0-100 ppm) and for total hydrocarbons (Beckman model 108A, 0-25 ppm).

ENVIRONMENTAL CONTROL: TEMPERATURE AND HUMIDITY

The temperature-humidity control system of each chamber (except for the water-filled Undersea Simulator Chamber) was based upon two high-capacity heat exchange coils located inside the chamber. These coils could be connected as independent chiller and reheat coils for fine control of humidity and temperature, or they could be connected in parallel for maximum cooling or heating capacity, with only temperature control, during rapid compressions. Modes could be changed during compression by pneumatically activated valves; transfer was initiated by manual operation of a single selector switch.

The temperature control systems used resistance temperature sensors, electric-to-pneumatic converters, pneumatic controllers and pneumatically operated valves. A proportional control range was provided by mixing hot and cold heat exchange fluids (water-propylene-glycol mixture). The temperature controller acted on the reheat coil alone during operations in the temperature-humidity control mode, on both coils when in the maximum cooling or heating mode.

The humidity control system utilized hygroscopic moisture sensors, electric-to-pneumatic converters and pneumatic controllers. The humidity controller acted on the chiller coil alone during operations in the temperature-humidity control mode and was inactive in the maximum cooling or heating mode.

Air circulation for heat exchange was provided by blowers powered by hydraulic motors. The power units which provided hydraulic fluid (water-glycol mixture) to the motors used case-drainage collection systems which were referenced to chamber pressures, maintaining the differential pressures across the hydraulic motor shaft seals near zero. Speed control was by pneumatically activated valves in parallel with the motors.

Sources of hot and cold fluid consisted of a steam heat exchanger to provide hot fluid and a refrigeration compressor to provide cold fluid.

The water-filled chamber in which the underwater work experiment was performed (see Section F) had an external loop temperature control/filtration system consisting of a resistance temperature sensor, electric-to-pneumatic converter, pneumatic control and pneumatically activated valves, water pump, filtration system and heat exchanger.

SAFETY: SYSTEMS REVIEW AND CERTIFICATION

A complete review of the structural integrity of the pressure vessel systems and associated electrical, fire control and environmental life support systems was carried out under the auspices of the U.S. Navy System Certification Authority.

SAFETY: ELECTRICAL SYSTEMS

Electric power was supplied to the chambers via isolation transformers, which would have disconnected in case of activation of the fire protection water deluge. An emergency generator (15 kilowatts) would have come on line automatically, in case of primary electric power failure, to provide electricity to the most vital systems of light, communication and atmosphere control. A standby generator (150 kilowatts) was available in case of extended power outage. A 12-volt battery pack would have instantly provided for emergency lighting to all chamber compartments upon primary power failure and during switch-over to emergency power. Standard lighting was low voltage (24 volts) and zoned, and its intensity could be adjusted to zero.

SAFETY: FIRE PROTECTION

Fire protection for the chamber interiors consisted of a water deluge system with ancillary water hoses available for use separately under appropriate circumstances. Electric power to the chambers (except 12-volt emergency lights) would have been automatically disconnected by flow switches in the fire water lines. Water for fire fighting was stored in a hydropneumatic tank, with delivery pressure provided by compressed air. The single pressurized storage tank supplied each chamber compartment with water at the correct overpressure via pressure-reducing regulators.

Fire protection available in the chamber control and machinery area was by water hose, chemical extinguishers and carbon dioxide extinguisher, as appropriate. Compressor and equipment rooms were protected by an automatic smoke detection and alarm system, with a Halon extinguishing system.

Protection against smoke was by emergency mask and emergency breathing gas quick-connects inside the chamber for the subjects and outside for the operations personnel.

SAFETY: INTERCOMMUNICATIONS

As stated previously, a hands-free party-line intercom system was employed by operations and investigative personnel and by the subjects during the experiment phases of the simulated dives. The helium speech processor (Marconi type 023) was effective in improving the intelligibility of speech originating in the high-pressure helium-oxygen environment.

Backup systems were available for communication both into and out of the chamber. A loudspeaker intercom and a direct system were available for speaking to the subjects, in addition to the party line. Emergency microphones located inside the chamber could also be directly connected to any available helium speech processor outside the chamber in case of failure of the party-line systems. Sound-powered phones provided a final means of communication.

Visual communications between subjects and operational or investigative personnel were possible through viewports. Closed-circuit television cameras enabled outside personnel to monitor activities inside the chamber. Entertainment television was viewed by the subjects through a viewport, with sound available on a music-news loudspeaker or on headsets.

SAFETY: REDUNDANCY

For the systems involved in the pressurizations, redundancy was built in. This feature of electric power supplies, gas analyses and intercommunications has been described earlier. Chamber escape capability existed in the interconnection between the Experiment and Living Chambers. Pressurization systems were duplicated for each chamber. The oxygen makeup system of the Experiment Chamber could serve the Living Chamber, and vice versa, and gas sample systems could be rigged to serve for oxygen makeup as well. The Experiment and Living Chambers had independent carbon dioxide scrubber systems, and portable, lock-in carbon dioxide scrubbers were available.

INSTRUMENTATION

Electrical instrumentation connections were required to link the sensors and transducers located inside the Experiment Chamber, under the control of the subjects, with the electronic and electrical equipment located outside, under the control of the investigative staff. The required "through-bulkhead" connections for the study of a Subject Pair totaled 244. Appropriate assignment of the wiring location by function made it possible to avoid electrical interference between signals.

Several variables, such as cardiac stroke volume and electrocardiography were measured in rest and exercise subjects but were not measured simultaneously in both. In such cases, it was possible to reduce the required number of through-bulkhead leads by providing relay assemblies inside the chambers. Signal wires from one or the other subject could be selected sequentially (multiplexed in time) by the investigators outside the chambers.

SENSORS REQUIRING THROUGH-BULKHEAD WIRING

The following sensors, designed and selected specifically for this study, required through-bulkhead wiring from the sensors and other equipment inside the Experiment Chamber to measuring and control equipment outside:

Temperature sensors
- Skin, rectal, esophageal
- Expired gas, ambient gas
- Pneumotachograph, dry spirometer

Eye fixation lights (two sets)
Electroencephalograph and electronystagmograph electrodes (two sets)
Electrocardiograph electrodes
Esophageal pressure transducer
Diaphragmatic electromyograph electrodes
Nerve conduction velocity electrodes (two sets)
Mastoid vestibular stimulation electrodes
Condenser microphone and pre-amplifier for speech recording
Audiometer (bone conduction stimulator)

Perceptual-Cognitive-Psychomotor (P-C-P) test-response unit
P-C-P response switches (on bicycle ergometer)
X-Y coordinate position sticks (two)
Gas collection-dump bag pressure switch
Bicycle ergometer control unit
Pneumotachograph differential pressure transducer
Dry spirometer signal
Intentional tremor transducers (two)
Postural tremor transducers (two)
Balance platform (2 axes)
Airway occlusion valve
Ambient gas velocity transducer
Light source for visual acuity
Multiplex relays control signal
Electrical impedance cardiograph electrodes

SUBJECT INSTRUMENTATION HARNESSES

Each subject was directly instrumented with the multiple sensors required. The associated interconnecting wires and cables were arranged into single "umbilical cords" and secured to subject harnesses. Wires were subgrouped by function and terminated onto connectors devised from standard printed circuit "boards." After donning a harness the subject was able to quickly link his harness to the equipment outside the chamber by plugging these connectors into mating receptacles secured to the chamber overhead. The subjects, excluding communications and apparatus control systems, were instrumented as shown in Table VI.

SUBJECT ELECTRICAL SAFETY

Both subjects were simultaneously connected to several electronic instruments. It was therefore necessary to provide appropriate measures to prevent either cross-ground currents or equipment malfunction from causing excessive currents to pass through either subject. The following precautions were taken. a) Temperature probes were operated from low-voltage bridge circuits. Insulation resistance of each temperature probe was required to be 10 megohms or more, from conductors to probe surface.

TABLE VI. Instrumentation Leads

	No.
Exercise Subject	
Temperature sensors: 10 skin, 1 rectal, 1 esophageal	24
Electroencephalograph and electronystagmograph	19
Electrocardiograph	5
Electrical impedance cardiograph	4
Diaphragmatic electromyograph	3
Nerve conduction velocity electrodes	4
Total	59
Rest Subject	
Temperature sensors: 10 skin, 1 rectal	22
Electroencephalograph and electronystagmograph	19
Electrocardiograph	5
Electrical impedance cardiograph	4
Mastoid vestibular stimulation electrodes	3
Nerve conduction velocity electrodes	4
Total	57

b) The electrocardiograph, the electrical impedance cardiograph, the amplifier for the diaphragmatic electrodes, the mastoid stimulator and the nerve conduction velocity stimulator all had floating, isolated amplifiers with worst-case leakage current levels of 20 microamperes or less. c) The most critical instruments from the electrical safety aspect were connected to a single electrical branch circuit. This was a "floating" circuit monitored by a ground-fault alarm. In the event of water deluge in the chamber by the fire-fighting system, this circuit would have been automatically de-energized by a water flow switch.

Additional safety measures involved the appropriate use of portable "ultra-isolation" transformers and ground-fault interrupters.

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E. PHYSIOLOGICAL AND PERFORMANCE INVESTIGATIONS--INTERRELATIONSHIPS OF NEUROLOGICAL, PHYSICAL WORK AND PERFORMANCE STUDIES

Direct Observations

E-1. Symptoms and Overt Manifestations Induced by Rapid Compression

Neurological, Neuromuscular and Performance Studies

E-2. Electroencephalographic Changes
E-3. Visual Evoked Cortical Responses
E-4. Tremor and Somatosensory Evoked Cortical Response
E-5. Muscle Strength and Coordination
E-6. Vestibular Function and Balance
E-7. Auditory Function
E-8. Visual Function
E-9. Speech Generation and Distortion
E-10. Perceptual, Memory, Cognitive and Psychomotor Functions
E-11. Sleep Electroencephalographic Patterns

Respiratory, Metabolic and Cardiac Studies

E-12. Acute Effects of Hydrostatic Pressure on Pulmonary Function
E-13. Cardiac Electrical and Mechanical Function
E-14. Ventilatory and Metabolic Responses to Exercise During Rapid Compression to Extreme Pressures
E-15. Thermal and Metabolic Homeostasis
E-16. Ventilation at Rest During Compression and at Stable High Pressures

Biochemical, Endocrinological and Hematological Studies

E-17. Biochemical, Endocrinological and Hematological Studies

E-1. SYMPTOMS AND OVERT MANIFESTATIONS INDUCED BY RAPID COMPRESSION

C.J. Lambertsen[1], J.M. Clark[1] and C.D. Puglia[1]

The responses to rapid compression and extreme hydrostatic pressures include symptoms, overt manifestations and behavioral changes which cannot be recorded quantitatively. Nevertheless, while unmeasurable, they may represent the fine and integrated expressions of adverse biophysical and physiological effects induced by changes in hydrostatic pressure. Human subjects exposed to pressurization in studies elsewhere have experienced nausea, vomiting, dizziness, loss of alertness, indifference, disorientation, mental confusion, and somnolence (1-4,7,9-13,19-25,27). Grossly observable effects reported from such studies have included coarse tremors, motor incoordination, some decrement in balance function, muscle fasciculations and spastic movements. In animals exposed to higher pressures and more rapid compressions, the neurological influences generate gross tremor, convulsions and unconsciousness (5,6, 8,16-18). At extreme pressures cessation of cardiac action has been observed (17).

Even the symptomatic and the general neural and neuromuscular influences are important for several reasons. If they occur with sufficient severity and persist, they may be of operational significance in limiting the performance capabilities and safety of a working diver or experimental subject. Moreover, even moderate effects could be important if they do not prove to be fully reversible on ultimate reduction of pressure. Compression at faster rates and to

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still higher pressures than those investigated thus far may be expected inevitably to produce the same physical incapacitation, loss of consciousness, convulsions and even death observed in animals exposed to such extreme pressures.

In the present study it was considered important to combine measurements of physiological effects of compression with detailed documentation of direct observations of the subjects' sensations and overall activity. This made it possible to evaluate the functional importance of physiological changes which would not otherwise be interpretable. The documentation of symptoms and overt manifestations which follows was designed to serve that purpose.

MONITORING AND RECORDING

The diver-subjects were observed continuously both directly and by audio and video monitoring during the initial compressions from the surface and subsequent excursions, and essentially continually throughout the days and nights of saturation and decompression. In specific circumstances considered most critical, both video tape and motion picture recordings were also employed. Pertinent observations at all stages were recorded in written log books which included the "decompression data bank log," the "medical log" and the "investigative log." During periods of the compression-pressure profile in which symptoms, signs or behavioral changes were considered likely to exist, subjects were interviewed extensively between experiment modules, and these communications were recorded on magnetic tape for subsequent correlation with other findings. Information extracted from all of these sources was used in interpreting the results of concurrent physiological and performance measurements. Availability of written audio and video recording is indicated in Appendix E-1 at the end of this section.

COMPRESSION EFFECTS EXPERIENCED BY SUBJECTS IN PHASE I (0-800-1200-800 fsw) AND PHASE II (0-800-1200-1600-1200 fsw)

SUMMARY OBSERVATIONS

The symptoms and overt manifestations observed in Phases I and II are shown in Table I and in Figs. 1 and 2. Table I summarizes for both phases the incidence and relative severity of symptoms and observable effects. The time course and pressure of occurrence for specific effects are shown in Figs. 1 and 2 on plots of the pressurization profiles, with descriptions of each manifestation and appropriate subject identification below the profiles. These effects will be discussed separately in detail for each phase of this program.

Essentially all of the manifestations reported by others, as cited previously, were also experienced by two or more of the subjects in this study at some time during the two exposures. The manifestations most frequently observed during compression included fatigue, nausea, lightheadedness, tremor of limbs and muscle fasciculations. They were most severe in the more rapid pressurization of Phase II, in the several hours following initial compression from the surface to 1200 fsw. Upon reaching this pressure two subjects missed portions of the programmed periods of physiological measurement, due to prominent symptoms generated by the initial compression, and fully used a rest period prior to continuing with measurements in the planned sequence. Given the opportunity they promptly fell asleep in the chairs provided, were easily awakened, and responded cooperatively and succinctly to questions. When allowed to rest further, they quickly dozed off again. After the first hour at 1200 fsw they were able to continue working effectively throughout the remaining series of experiment modules at that pressure.

Subject Responses

In judging the general state of the subjects and their self-appraisal of symptoms it is important to point out

TABLE I. Summary of Symptoms and Objective Changes During Compression and Exposure to Depths of 800, 1200 and 1600 fsw[a]

Obvious Effect	0-800 fsw Subj.	Severity	Phase	800 fsw Subj.	Severity	Phase	800-1200 fsw Subj.	Severity	Phase	1200 fsw Subj.	Severity	Phase	1200-1600 fsw Subj.	Severity	Phase	1600 fsw Subj.	Severity	Phase
Nausea	GM	+	II	GM	+++	II	CC GM	+ ++	I II	CC LJ GM FS	++ + ++ +	I I II II				GM	+	II
Vomiting				GM	+	II				GM	++	II						
Headache				CC	++	I	FS	++	I	FS FS	++ ++	I II						
Joint pain	WS CC	+ +	I II	CC WS CC MP GM	+++ + + + +	I I II II II	CC WS	++ +	I I	CC WS GM	++ + ++	I I II						
Coarse tremor (visible trembling)				CC	++	II	CC CC MP GM	++ ++ +++ +++	I II II II	CC FS CC MP FS	+ + ++ ++ +	I I II II II	MP	+	II	CC MP	++ ++	II II
Mental slowness	GM	+	II	MP CC GM	+ ++ ++	II II II	CC CC GM	+ + +	I II II	CC	+	II	MP	+	II	MP CC GM	+ + +	II II II
Dizziness and lightheadedness				MP FS GM	+ + +++	II II II	CC WS FS MP GM	+ + + ++ +++	I I I II II	LJ MP CC FS GM	+ + + + ++	I II II II II	GM	+	II	MP CC GM	+ + +	II II II
Postural imbalance										MP FS CC	+ + +	II II II	MP FS	+ +	II II	MP CC FS	+ + +	II II II
Incoordination							CC	+	I	MP	+	II				FS CC	+ +	II II
Ratchety movements										MP	++	II	MP	++	II	MP CC FS	++ + +	II II II
Fasciculations							MP	++	II	MP CC FS GM	++ + + +	II II II II	MP FS	+ +	II II	MP FS	+ +	II II

[a]Blank indicates "did not occur" in any subject; +, mild; ++, moderate; +++, prominent, ++++ severe.

that they had the exceptionally high morale and dedication which stemmed from the combination of positive attitudes, strong personal character, their extensive technical training, their close identification with and interest in the purposes of the study, and their clear recognition that the program aimed to induce derangements in order to investigate them. It was their intent and their function to succeed in spite of personal discomfort rather than to fail because of it. This approach of subject-divers to investigation is necessary to avoid the confusions generated by withdrawal from inadequately defined effects.

It is also necessary in judging the subjects' condition and responses to recognize the fatiguing effects of the long and strenous time course of the representative experiment day of initial compression. This involved extensive technical preparation and instrumentation checks prior to compression, performance of timed measurement modules during compression, and dismantling of experimental systems, followed by a late night meal, bathing and rearrangement of the chambers as habitats. Preparation, self-instrumentation and baseline measurement periods began at 6:00 a.m. and required about four hours. Compression and repeated performance of modular measurements required about eight hours of essentially uninterrupted and intensive activity. Habitation arrangement, evening meal and personal hygiene required about four hours. For these reasons general symptoms of fatigue and drowsiness occurring late in the pressurization sequence are considered in part to be normal and inevitable. Specific effects of compression should represent an overlay upon this normal fatigue.

Differences in occurrence and degree of effects induced must be related to individual subjects, to the same subject in different pressurizations, and to the intentional increases in rates of compression utilized to induce prominent compression effects for study. Therefore, even within this coordinated single investigative program the two subjects (CC and FS) who participated in both of the primary compression phases (I and II) did not respond identically during their first and second exposures.

Differences in Initial Compression

Exact comparison between all subjects and specific compressions throughout the entire program cannot be made for two reasons: The first is that Phase I, while a full-scale effort, was used as an exploratory exposure to examine selected compression rates for expected prominent symptoms on first arrival at the 1200 fsw pressure. Since only moderate symptomatic effects and essentially no objective changes, which could then be tracked toward resolution, were found, the compression rates for the four subjects in Phase II were greater than those selected for Phase I.

The second difficulty in exact comparison was not anticipated. The original intent to study four subjects (as opposed to the more usual investigation of a single pair) was followed. However, the two subjects of Phase I who developed neurological and vestibular decompression sickness (Section G-4) were intentionally not used as subjects for the second phase of the program. Therefore, while two subjects (CC and FS) did experience the entire pattern of compressions in both phases, GM and MP experienced only the more severe compression conditions of the 0-800-1200-1600-1200 fsw excursion and saturation of Phase II.

The intended primary comparisons between Phases I and II involve the initial compressions to 1200 fsw. Since it was anticipated that any effects generated during compression might increase rather than decrease in severity with time, even without further increase in pressure, the exploratory Phase I plan included withdrawal back to 800 fsw following transient (15-minute, 30-minute and 45-minute) exposures to the 1200-fsw pressure. In the second phase the subjects remained at 1200 fsw to use this pressure as the base for still deeper excursions.

The incidence and severity of symptoms and other effects were, with some exceptions, generally greater in Phase II than they were in Phase I (Figs. 1 and 2). This was the intended result of the faster compression from sea level to 800 fsw, and the shorter stop at 800 fsw before proceeding to 1200 fsw in Phase II (see Section D). In addition, following compression from sea level, the subjects of Phase II remained for many days at the 1200 fsw

depth (Fig. 2) while those in Phase I decompressed back to 800 fsw after an initial excursion of 15 minutes at 1200 fsw (Fig. 1).

SPECIFIC EFFECTS OF COMPRESSION IN PHASE I (0-800-1200-800 fsw)

Initial Compression to 800 and 1200 fsw

Of the four subjects compressed in Phase I, none experienced important symptomatic or objective neural effects on arrival at the 800 fsw pressure. Two subjects (WS and CC) did have moderate joint pain on compression to 800 fsw (Fig. 1A and C).

The only further symptoms developed in any subject during the two-hour stable exposure to 800 fsw, prior to the transient compression to 1200 fsw, were headache and joint pain (right hip, elbow and ankle) in CC (Fig. 1C). The joint pain was severe enough at 800 fsw to make it necessary for him to stop pedaling the ergometer bicycle temporarily. It was the only limiting effect upon performance observed in the first step of compression (0-400-800 fsw). Tremor was not sufficiently gross to be visible and it presented no evident obstacles to detailed technical procedures involved in performance of the experiments.

Compression to 1200 fsw did result in distinct symptoms in three of the four subjects (Fig. 1A and C). One (subject LJ, Fig. 1C) had no appreciable discomfort or other evidence of compression effect. Symptoms and general effects included headache (subject FS, Fig. 1A; subject CC, Fig. 1C), a dizziness on standing that was not vertigo (subjects FS,WS, Fig. 1A), "shakiness" and trouble concentrating, slight nausea, and some feeling of incoordination (subject CC, Fig. 1C).

Return from 1200 to 800 fsw led to prompt relief of symptoms cited, to a degree that the diver-subjects, while tired from the 14-hour day, considered work in water practical.

Excursion-Compressions on Days 2,3 and 5

On the days following initial pressurization, excursion-compression from 800 to 1200 fsw produced no gross effects in any of the four subjects. On the second day of exposure subjects WS and CC (Fig. 1B, D) felt some joint discomfort and subject CC "a little tremor" (Fig. 1D). No other or subsequent effects of pressure or excursion were considered important and the subjects appeared highly competent.

SPECIFIC EFFECTS OF COMPRESSION IN PHASE II (0-800-1200-1600-1200 fsw)

The more rapid rate of compression to 800 fsw in this phase was successful as planned, in inducing more prominent symptomatic effects in the four subjects at the higher pressure of 1200 fsw.

Initial Compression to 800 and 1200 fsw

At 800 fsw, effects were slight, and included minor joint discomfort in three subjects (GM, Fig. 2A; CC, MP, Fig. 2C). While all subjects felt and were generally competent, each had recognizable symptoms which included slight "light-headedness" (subjects FS, GM, Fig. 2A, nervous energy (subject FS), slight decrease in mental sharpness (subject CC, Fig. 2C) and slight dizziness without vertigo (subject MP, Fig. 2C). Three of the four subjects were hungry and had lunch. One (GM) became nauseous and vomited on drinking orange juice. No subject had grossly evident tremor. All felt "well" at the beginning of compression to 1200 fsw except for slight residual nausea in GM, slight shakiness (MP), and slight effect upon thinking (CC).

Each subject was noticeably affected by the compression to 1200 fsw. Effects were of such varied character and degree that they are best represented for individual subjects by the extracted summaries of Fig. 2A and C. Of special importance is that both members of one subject pair (FS,GM) had nausea and prominent fatigue with vomiting on arrival at 1200 fsw, and subject GM did not finish Module 10 at 1110-1180 fsw (Section D). For these subjects it was necessary to provide rest and time to adapt, resulting in their missing the initial experiment modules scheduled for 1200 fsw.

Members of the other subject pair (CC,MP) sensed fatigue with shakiness, tightness and nervous tension not evidently related to anxiety. Slight dizziness occurred in three subjects (GM,FS,MP) with a minor spinning component in one (MP). General mental responsiveness was prompt throughout in all subjects.

All subjects used the scheduled rest period between initial experiment modules at 1200 fsw. Two subjects (GM and FS) were allowed to skip experiment Modules 11 (MP) and 12 (FS) to allow time for partial recovery from the prominent nausea, weakness and fatigue generated by the initial rapid compression. Following about one hour of rest, they continued fully with the experimental procedures.

After about two hours of stable exposure to 1200 fsw all subjects were self-sufficient but exhausted. All participated in normal procedures of converting the chamber system from laboratory to residence uses.

Excursion-Compressions from 1200 to 1600 fsw

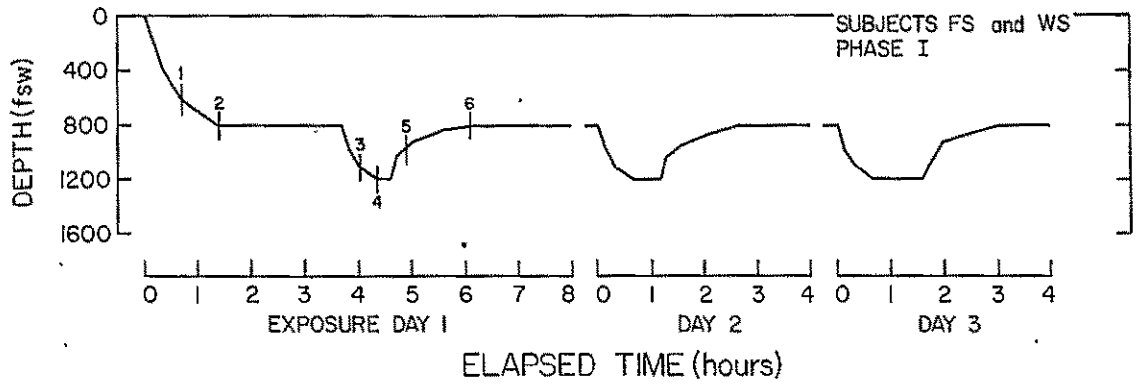
Excursion to 1600 fsw on the day following initial compression to 1200 fsw produced no severe or limiting overt effects. The pattern of individual subjective responses and reactions are elaborated in Fig. 2B and D. Two subjects indicated slight decrease in mental clearness (Fig. 2B) with feelings of "jumpiness" or "shakiness," without prominent visible tremor or twitching.

A day later (day 3 of compression) occasional muscle fasciculations were evident in several subjects, with symptoms of slight dizziness on rapid head movement (subject GM, Fig. 2B), lack of "fine muscle control" (subject FS, Fig. 2B; and subject CC, Fig. 2D), and some visible coarse tremor of extremities (FS). Mental and overall competence permitted active and effective performance of experimental and general functions without important discomfort.

All subjects felt well and appeared asymptomatic on return to 1200 fsw for saturation exposure and subsequent practical underwater work excursions to 1600 fsw.

FIG. 1

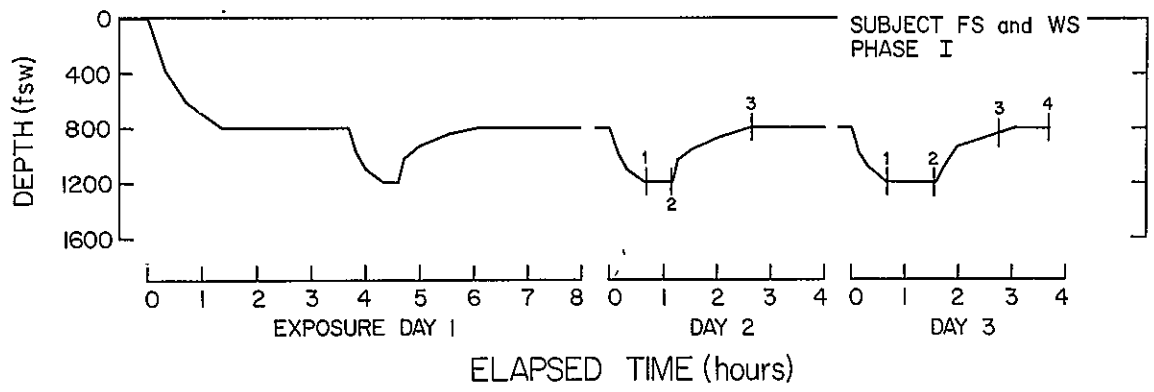
(A)



DAY 1

Depth (fsw)	Elapsed Time	Symptoms and Signs
600	0:41	1. WS had left wrist pain, only during movement; joint felt "dry."
800	1:23	2. Both subjects felt fine on reaching 800 fsw except for left wrist pain on movement in WS. Continued to feel well throughout "hold" of 2 hours 18 minutes at 800 fsw.
1100	4:01	3. WS slightly dizzy upon standing; lightheaded, not vertigo. FS had "just a bit of a headache" ... "right behind my eyes." "Otherwise I'm all right." Headache was worse while watching flashing light for visual evoked response.
1200	4:21	4. FS: "I have sort of a dizziness when I get up and down but I think that's just part of the headache. I pretty much feel okay." Dizzy feeling on standing started soon after leaving 800 fsw. WS had "just a little" pain in left wrist. Dizziness "went away before we got to the bottom." No other problems.
950	4:54	5. FS still had headache, no dizziness. WS had minor soreness in left wrist but not acutely painful.
800	6:05	6. Both divers felt fine. Both were tired during excursion but thought they could have done underwater work safely.

(B)



DAY 2

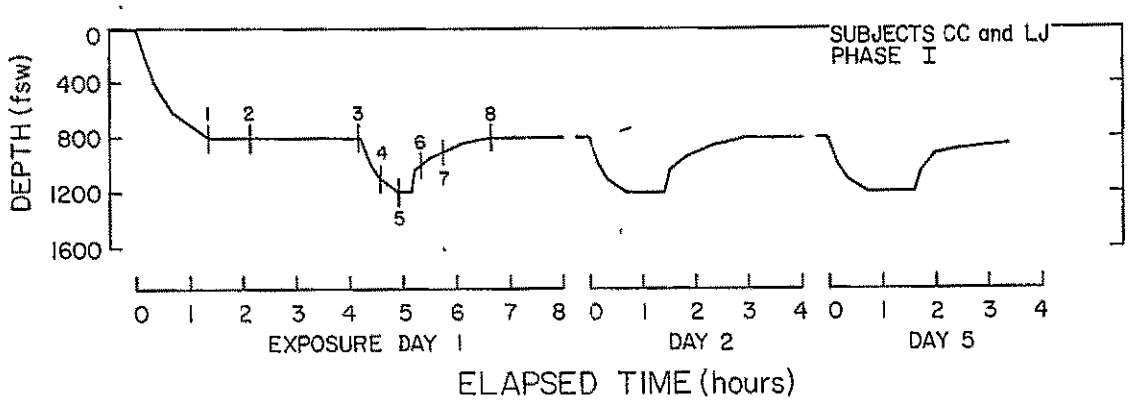
Depth (fsw)	Elapsed Time	Symptoms and Signs
1200	0:40	1. Both subjects felt fine on reaching 1200 fsw. FS: "it really feels smooth." "I just feel 100% better [than yesterday]." "I just feel the same as I felt at 800 fsw." WS: "I still have the same pain in my left wrist, and a few more joints have snapped, cracked and popped, but I feel great."
1200	1:10	2. No problems during stay at 1200 fsw.
800	2:39	3. Both divers felt fine on return to 800 fsw.

DAY 3

Depth (fsw)	Elapsed Time	Symptoms and Signs
1200	0:40	1. No subject experienced effects on compression from 800 fsw.
1200	1:35	2. No symptoms at 1200 fsw.
835	2:44	3. Decompression: WS developed progressive numbness and paresthesias of right leg, back and right side. Tracked for diagnosis of decompression sickness.
800	3:40	4. Recompressed to 1050 fsw to treat decompression sickness.

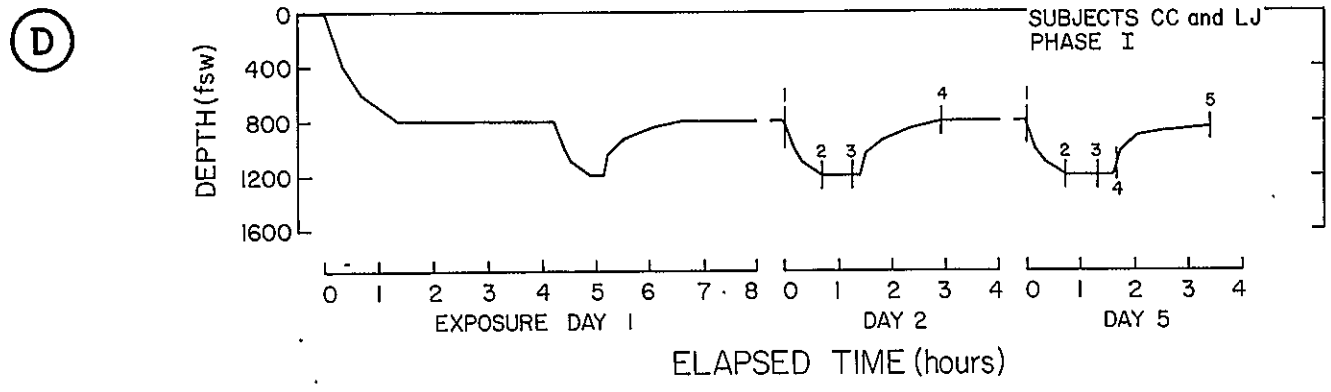
FIG. 1 (cont'd)

(C)



DAY 1

Depth (fsw)	Elapsed Time	Symptoms and Signs
800	1:20	1. Both subjects felt fine on arrival at 800 fsw. At about 500 fsw LJ had temporary sensation of clockwise rotation during electrical vestibular stimulation of right and left mastoid regions.
800	2:10	2. CC had headache and joint pain (right hip, elbow and ankle) which caused temporary cessation of exercise.
800	4:10	3. LJ had strong sensation of counterclockwise rotation during stimulation of left mastoid; right mastoid stimulation produced weak sensation. Both subjects felt fine at start of compression to 1200 fsw.
1100	4:33	4. Starting at about 1000 fsw, CC felt "a little slow," "thought they were increasing the tension on the exercise bicycle," and "had a rocking motion." His leg was shaking. Described feelings as: "a little bit shaky," "achy," "seasick and queasy." He felt tired and fell asleep sitting on the ergometer bicycle during some of the mental function tests, temporarily stopped breathing through mouthpiece because "the air is getting hot and heavy." LJ had no apparent problems.
1200	4:53	5. At 1200 fsw CC felt shaky; sitting on exercise bicycle he performed his roles but had trouble concentrating on tasks; seemed inattentive and poorly communicative in conversation; slightly nauseous; "the air is so hot and thick in here"; headache and dizziness were gradually subsiding; had joint pains in right hip and ankle and also elbow; felt uncoordinated but not weak. LJ was all right at 1200 fsw.
1000	5:20	6. CC had rapid decrease of joint pain and increased alertness by 1000 fsw. LJ had no problems.
905	5:45	7. CC was no longer dizzy but was still "just a little bit sick to my stomach."
800	6:37	8. Both divers felt fine, ready for full meal. CC: "just fine." LJ: "I feel terrific."



DAY 2

Depth (fsw)	Elapsed Time	Symptoms and Signs
800	0	1. CC somewhat slow in preparation for experiments but felt well with exception that "my joints are a little bit dry." Both subjects stated they were "ready to go."
1200	0:40	2. CC had grinding sensation for few seconds in right ankle between 800-900 fsw. At 1200 fsw, CC: "I feel pretty good." LJ: "I'm fine."
1200	1:16	3. CC: "I feel like I've got a little tremor, but I have no pains and no queasy feelings." Both felt well. LJ had no apparent problems. Left 1200 fsw at 1:25.
800	2:54	4. CC: "just fine." LJ: "no problem."

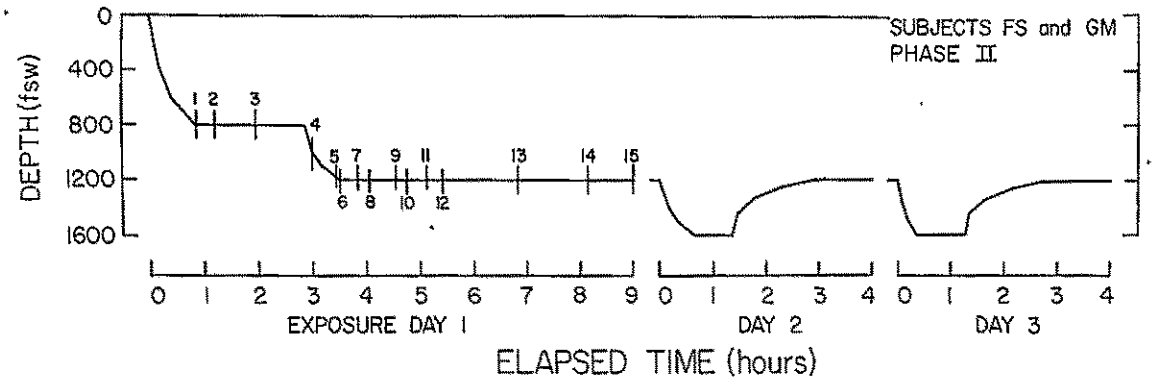
DAY 5

Depth (fsw)	Elapsed Time	Symptoms and Signs
800	0	1. CC had diarrhea in a.m. Still had headache and queasiness on beginning compression.
1200	0:40	2. Both subjects had no apparent symptoms on compression.
1200	1:19	3. LJ felt nauseated and thought the vestibular electrical stimulation gave unusually strong responses; dizzy on standing. CC: "I'm fine." Left 1200 fsw at 1:35.
1100	1:38	4. LJ not nauseated now. Both subjects felt okay.
845	3:23	5. Decompression sickness in LJ. Spinning sensation (felt room was moving counterclockwise); nystagmus (fast component right); recompression to 1050 fsw to treat vestibular decompression sickness.

FIG. 1. Symptomatic and objective manifestations: compression to pressures equivalent to depths of 400 and 800 fsw in Phase I, with further transient excursions to 1200 fsw pressure equivalent. Upper section in each part of figure shows profile of compression. Numbered points in profile indicate depth and time of cited effects. Quotations are descriptions of observed symptoms and signs in the subjects' own terminology. A,B, Subjects FS and WS; C,D, subjects CC and LJ.

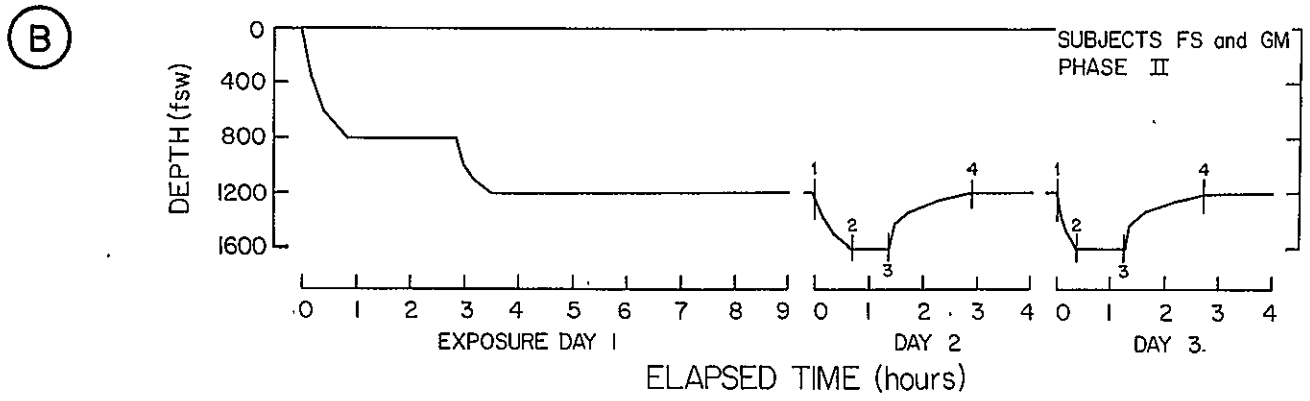
FIG. 2

(A)



DAY 1

Depth (fsw)	Elapsed Time	Symptoms and Signs
800	0:52	1. Subjects felt "very good" (FS) and "pretty good" (GM). GM's knees and left hip ached slightly during ergometer exercise only.
800	1:12	2. FS a "little bit" lightheaded with much "nervous energy." GM a "tiny bit" lightheaded.
800	1:57	3. FS felt "real good." GM suddenly became nauseous and vomited on drinking orange juice following about five glasses of water during compression, after 58 minutes at 800 fsw. Sweating, felt "hot" and "dizzy" on vomiting. Felt fine "almost 100%," immediately after vomiting, but with periodic recurrence of slight nausea. GM felt better every time temperature went down. Both subjects felt "very good" at point of beginning compression to 1200 fsw.
1000	3:00	4. GM felt disoriented and "little bit dizzy" on ocular fixation at 950 fsw. Required conscious effort not to fall from bicycle at about 1050 fsw.
1180	3:26	5. GM: involuntary coarse tremor of leg, persisted when weight put on leg at 1100 fsw. "Dizzy," felt as though "turning to right." Dizziness was severe. At 1180 fsw mentally clear but felt "as though rocking back and forth," with increasing nausea.
1200	3:30	6. On arrival at 1200 fsw: FS felt "slightly lightheaded," only discomfort was "slight headache"; doesn't feel right up to par. GM still "a bit dizzy" but this was slowly subsiding. Ten minutes after reaching 1200 fsw dizziness "suddenly getting much better."
1200	3:50	7. FS began "rest" module 20 minutes after arrival at 1200 fsw. GM excused from starting on "exercise" module due to extreme feeling of fatigue. FS became nauseous while standing on balance board, 13 minutes into module, prior to vestibular electric shock. Subjects allowed to sit and rest.
1200	4:03	8. GM felt "a tiny bit weak," as though he could sleep for several hours, but much less disoriented; gradually recovering.
1200	4:32	9. FS resting. GM vomited small amount, asked permission to remove esophageal tubes, still slightly nauseated, still slight headache. Willing to begin "exercise" module.
1200	4:45	10. FS felt better, slept briefly.
1200	5:08	11. GM performed experiment "exercise module." He felt no worse while exercising. FS showed occasional isolated coarse tremors in extremities and facial twitches while sleeping. FS still nauseated but ready for next module.
1200	5:25	12. GM and FS performed modules. Three minutes into module FS showed some postural incoordination, intermittent coarse tremor of muscles of neck, trunk and limbs; none interfered with technical performance. Following the module both subjects were allowed to sleep.
1200	6:50	13. GM and FS performed module pair. GM felt pretty good, no nausea, a bit tired. FS had headache again, eyes felt as though he must strain to see test numbers; not blurred.
1200	8:09	14. GM and FS performed final module pair, both very tired. FS still had headache, no other problems. GM felt "okay" except for ache in some joints on standing.
1200	9:00	15. Subjects prepared chamber for supper and for night. Tired but active, coordinated, in good spirits; clearly competent. Had essentially no supper; said they were not hungry. FS had slight nausea; headache not prominent; slept well.



DAY 2

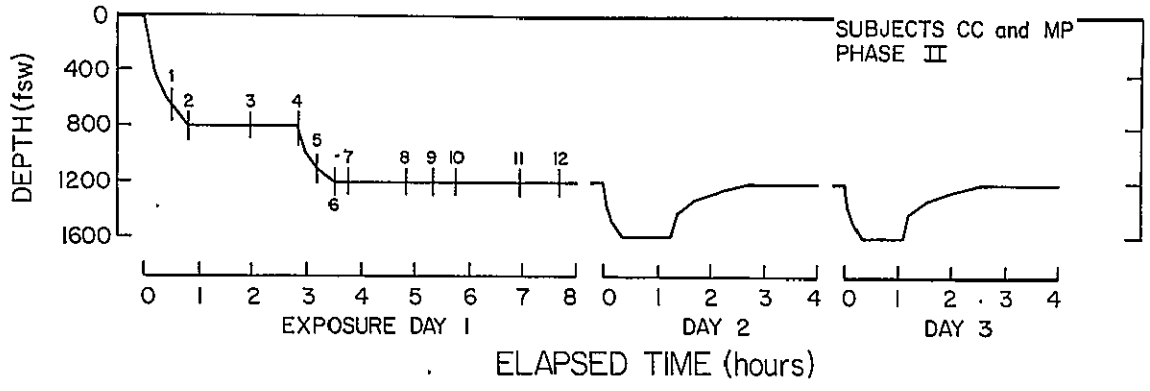
Depth (fsw)	Elapsed Time	Symptoms and Signs
1200	0	1. Both subjects were aroused promptly and were effective immediately. GM said that he had not been thinking as clearly as he would like, and he had some residual pain in knee and hip on bending. FS felt fine now--had slight headache and "squeamishness" before breakfast.
1600	0:40	2. Compression from 1200 to 1600 fsw produced no joint pain, headache, spontaneous nausea or spontaneous dizziness in either subject. GM "once or twice" had "a tremor or two"; had slight dizziness and slight nausea on intentional rapid head movement; slight lightheadedness and "slight" problem of concentration (said "mind" was 99%). FS lost headache during compression; showed occasional coarse twitch of hand or leg "as though I have a lot of nervous energy"; felt "a little bit slow mentally"; unsteadiness on balance board at 1480 and 1580 fsw, increased when eyes were closed; hungry.
1600	1:22	3. Both subjects felt fine prior to decompressing from 1600 fsw; no twitching or symptoms.
1200	2:51	4. Post-decompression both felt fine. GM stated sudden improvement in mental alertness on passing 1270 fsw--best since leaving surface. Slight hip pain, occasional catch in right shoulder.

DAY 3

Depth (fsw)	Elapsed Time	Symptoms and Signs
1200	0	1. Both subjects awakened easily from good sleep, without symptoms. Prior to compression to 1600 fsw FS showed slightly increased sway on balance board.
1600	0:20	2. On excursion to 1600 fsw, both subjects showed mild tremor and generally ratchety movements. FS had bursts of fasciculations in leg muscle at about 1550 fsw; less postural sway on balance board than during previous excursion. On arrival at 1600 fsw, FS was mentally clear and had no headache, joint pain, dizziness or nausea; felt slightly uncoordinated and does "not have that fine muscle control"; leg movements are "kind of choppy." GM had no joint pain; mentally "not quite 100%" but felt "pretty good"; "a little bit" of dizziness on very rapid head movements and slight nausea. Both subjects felt much better than on previous excursion.
1600	1:15	3. Felt as strong as normal; little change in condition of subjects during 55-minute stay at 1600 fsw. FS had perceptible decrease in ratchety movements; still had coarse tremor in all extremities, some unsteadiness in standing to get on balance board; short bursts of nystagmus in FS.
1200	2:44	4. Both subjects asymptomatic on reaching 1200 fsw.

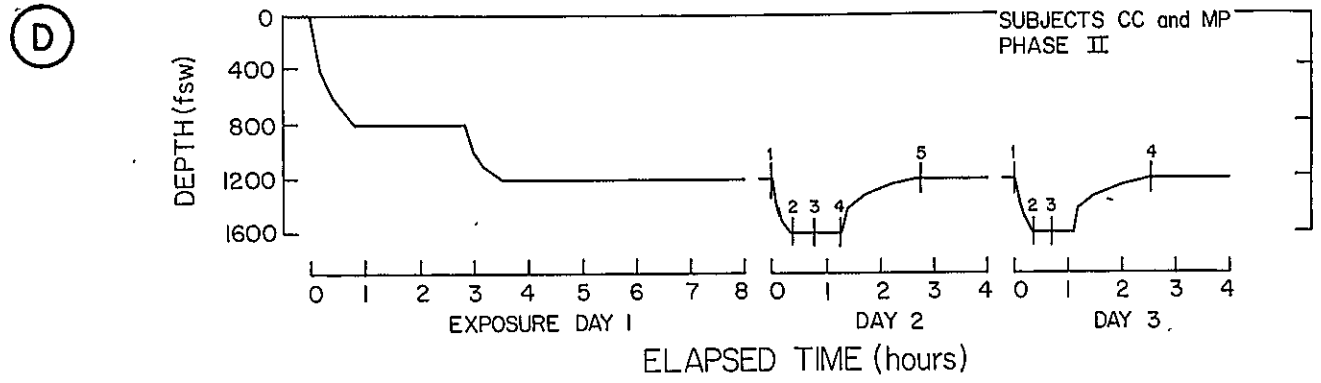
FIG. 2 (cont'd)

(C)



DAY 1

Depth (fsw)	Elapsed Time	Symptoms and Signs
640	0:30	1. CC had mild joint pain in right ankle beginning at 500 fsw, and right shoulder starting at 560 fsw, but only on motion.
800	0:50	2. Both arrived at 800 fsw and said they felt "all right," MP was "a little dizzy." CC still had a "little joint pain (right ankle, right shoulder) on motion,""little bit trembly," "not quite as sharp as normal," "about 95%."
800	1:56	3. CC no joint pains, no dizziness, "95% mentally." MP some "dizziness" on standing, no vertigo, no nausea, things "jump" a little bit. A little joint pain in right hip. Appetites for both normal, hungry, had lunch.
800	2:50	4. Prior to compression to 1200 fsw: MP felt well, believed he was clear mentally, with no symptoms except for "little pain in both hips," use of different lines on visual acuity test, "little shaky." CC: "thinking is a little bit distant, like 90 or 95%, like about 4 o'clock in the morning." Generally weak, no dizziness, headache, nausea or joint pain. "Kind of faint when doing pulmonary functions."
1100	3:12	5. MP felt "pretty good" but "very shaky."
1200	3:30	6. On arrival at 1200 fsw: MP felt mentally clear, "just a bit shaky," no headache or nausea, "spinning a little" but "if I turn 5 or 6 degrees it reverses direction." "Can feel muscle jerks" (upper thighs and abdomen), "not terribly uncomfortable." CC "mentally okay, about 90 to 95%." "Just a little bit edgy and jumpy"--"as if I'm all wound up tight." "No quivering in legs"--"have all kinds of energy going through my legs and I can't get rid of it."
1200	3:45	7. CC had increased intention tremor of hands; both legs also showed coarse tremor. Right leg moved rhythmically in manner of foot tapping; motion could be stopped voluntarily but occurred periodically even while supporting body weight. Said he felt "tight in all my muscles," "all strung out," "energy to burn." No nausea or dizziness.
1200	4:50	8. MP a little dizzy during electrical vestibular test but "otherwise feel fine."
1200	5:20	9. Subjects stated they felt tired; looked exhausted. Completed module.
1200	5:45	10. Subjects allowed to rest and sleep for "over" an hour.
1200	6:56	11. Awakened for final modules. MP "feels good," no symptoms except for a little pain in upper left thigh on certain movements. CC okay,"feel some tiredness but otherwise I am sharp," legs were shaking "a little bit" during module.
1200	7:41	12. Completed final module. Subjects looked very tired, exhausted but were otherwise without grossly observable effects. MP had headache he believed was related to EEG needles. Subjects effectively removed experiment leads, stowed equipment, prepared chamber living systems, had full dinner and uneventful night.



DAY 2

Depth (fsw)	Elapsed Time	Symptoms and Signs
1200	0	1. Good breakfast and lunch prior to excursion. Competent rigging of electrodes and apparatus. MP: "a little bit slow and little bit tired," a little dizzy "if I move really fast." Movements seemed slightly ratchety. "Still have a little bit of pain in [right] hip if I step wrong." Working dive in the sea "wouldn't bother me." CC "wasn't fed enough." Had slight pain in right knee which went away. Mental capacity "just about" normal. "Not really" dizzy but "balance is a little bit off." No vertigo. (All tremor effects appeared more intense early in a.m., then wore off).
1600	0:20	2. On arrival at 1600 fsw: CC mentally clear, had no dizziness or nausea, "occasionally I feel a little bit off-balance," "a little bit jumpy," "a little bit higher strung" than normal, "100% better" than previous night at 1200 fsw. MP: "mentally I'm about the same [as previous night at 1200 fsw]." "I don't feel slow." "Kind of shaky." No nausea. Unsteady ratchety movements when standing to get on balance board, "not really dizzy." "Wouldn't want to try standing up without holding on to something," "spinning a little bit" only with eyes closed on balance board.
1600	0:45	3. MP: "feel better," "a lot steadier," "think I'm faster all the way around right now." Dizzy only "when they hit me with the shocks on the right mastoid." CC slight increase in hand tremor, no leg tremor during module, "a little off-balance," "not really dizzy."
1600	1:15	4. Prior to decompression: MP "noticed my eyes are focusing farther away" in vision test. Both subjects appeared all right, no large problems.
1200	2:44	5. Arrival at 1200 fsw: both subjects felt all right, appeared fine. Had full dinner.

DAY 3

Depth (fsw)	Elapsed Time	Symptoms and Signs
1200	0	1. Prior to 1600 fsw excursion: good appetite. Competent rigging of experiment electrodes and apparatus. No symptoms except CC's earache.
1600	0:20	2. Arrival at 1600 fsw: MP felt "a lot better than yesterday [at 1600 fsw]," "more alert," better balance and coordination, no nausea. CC felt "about the same as yesterday," mentally "maybe a little bit better," "noticed that my small movements are kind of jerky." Joints felt okay, "just a little bit of pain."
1600	0:42	3. At 1600 fsw: occasional isolated muscle fasciculations in arm (CC), back (MP).
1200	2:31	4. Return to 1200 fsw: Both subjects were asymptomatic.

FIG. 2. Symptomatic and objective manifestations of compression to pressure equivalents of 400, 800 and 1200 fsw in Phase II with further transient excursions to 1600 fsw pressure equivalent. A,B, Subjects FS and GM; C,D, subjects CC and MP.

GENERAL FUNCTIONAL COMPETENCE IN COMPRESSION

The subjects experienced numerous manifestations of compression effects and showed overt signs of adverse influences. Direct observations of the subjects clearly cannot, alone, explain the fundamental bases of the derangements caused by increased hydrostatic pressure, neither can measurements of the induced tremor, electroencephalographic changes, or impaired coordination and balance. These observations and measurements are descriptive and are not directly pertinent to causes of the observed effects. Nevertheless, systematic and precise observations of such effects may at least provide clues concerning the affected structures and physiological mechanisms.

It must also be recognized that hydrostatic pressure acts upon many sites and functions as a composite of interacting influences which together cause the effects that may be monitored directly or by physiological measurements. The arbitrary selection of one easily recordable measure for a particular study neither prevents the selected measure from being influenced by other, unmonitored responses nor does it necessarily provide an appropriate index of general functional competence. This frequent oversight in the design of many pressure studies in man or other animals is mentioned here because it has special pertinence to investigation of the limits of human tolerance.

In the presence of symptoms and overt manifestations of compression and pressure effects, the subjects' ability to effectively perform a variety of tasks reflected the overall adequacy of the many physiological and psychological compensations that are normally available to man. Mental and sensory competence can evidently coexist with physical limitations such as mild tremor. During exposure to rapid compression and extreme hydrostatic pressure--just as during exposure to other environmental stresses such as nitrogen narcosis, altitude hypoxia, and motion sickness at sea or in space--an individual's tolerance and ability to think and work are determined by his initial reserve capacity as well as by the severity of stress to which he is exposed. Thus, an experienced individual could be essentially fully competent in the presence of mild stress and could perform many familiar tasks even while affected by severe stress.

Upon evaluation of the subject-divers' reactions in the light of such considerations, the following summary interpretations and judgments are made:

1. On slow, multiday compression and stable exposures to pressures of 1200 fsw, as in Predictive Studies III (14,15,26), no important physiological or performance derangements were detectable.

2. On the rapid, approximately one-hour, compression to 800 fsw, symptoms were induced in some subjects, but not in others. The reactions were transient and did not persist at 800 fsw following same-day excursion to 1200 fsw. It is likely that the deeper excursion accelerated adaptation to the 800 fsw exposure.

3. First-day rapid excursion to 1200 fsw produced the prominent symptoms which were sought in the study. Symptoms were temporarily prominent on arrival at 1200 fsw, to a degree seriously limiting practical function and security. Evident partial recovery occurred during continued exposure to 1200 fsw on the first day, but over a period of hours. Effects of hydrostatic pressure were still present in some subjects on the second morning at 1200 fsw but were of minor significance and not limiting.

4. Second-day excursion to 1600 fsw produced clear symptoms and signs of a pressure syndrome, which was less prominent than on the initial day of compression to 1200 fsw. Adaptation had begun and was sufficient in one day to allow effective technical performance at the 1600 fsw pressure.

5. Successive excursions showed still less effect, indicating continued, if slow, adaptation to at least some effects of compression, with capability for fine and vigorous mental and physical activity.

6. Compression from 1200 to 1600 fsw after five to nine days for working dives did elicit mild but visible tremor, indicating that some aspects of adaptation were still incomplete.

7. Effects generated by excursion to 1600 fsw disappeared on return to 1200 fsw.

8. No detectable residual effects of the entire composite of saturation-multiple excursion exposures occurred in any of the six subjects.

9. No evident obstacles to performance of the intricate, precisely timed and skillful technical work by the subjects existed following the initial compression day.

10. Subject-divers who had adapted after a several-day exposure to 1200 fsw were evidently as close to normal competence in the rapidly paced underwater work experiments at 1600 fsw as when performing the same tasks near sea level. Work rates appropriate to the pressure, depth and gas density circumstances would be still more practical.

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Dr. Ronald E. Hammond edited and condensed the various written and audio logs which provided the information discussed in this section and prepared initial drafts of the table and figures describing the time course of the observed symptoms.

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APPENDIX E-1

Documentation of Symptoms and Overt Manifestations

The following detailed records of symptomatic and objective effects of compressions will remain part of the Experiment Data Bank for Predictive Studies IV:

Audio monitoring log

Video monitoring log

Investigative log

Decompression Data Bank log

Medical log

Subject-investigator voice tapes

Video tapes of subject and underwater performance

Motion picture films of experiment and underwater performance

E-2. ELECTROENCEPHALOGRAPHIC CHANGES

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Rapid compression of animals (2,4-6,9) or men (1,3,7,9, 10,12,14) while breathing $He-O_2$ is associated with electroencephalographic (EEG) changes characterized by consistently increased theta (4-8 Hz) activity, less frequent augmentation of delta (2-4 Hz) activity, and decreased alpha (8-13 Hz) and beta (13-30 Hz) activity. The most severe conditions of compression and high pressure transform the waking EEG into patterns resembling those found in stage I or stage II sleep (1,3,9,12,14). (Patterns in sleep are described in Section E-11.) The EEG changes first appear at pressures equivalent to 400 to 1000 fsw and are accentuated by increasing the rate and/or degree of compression. Theta activity tends to occur initially in the temporo-occipital areas of the cortex and then spreads to involve middle and anterior regions of both hemispheres.

Although alterations of normal EEG activity have been found in all subjects under sufficiently severe conditions, the nature and intensity of effect varies considerably between individuals. Typically the EEG changes stabilize or ameliorate somewhat while remaining at a constant high pressure, but they may sometimes continue to progress under stable, saturation conditions. Reversion of the EEG to normal does not usually occur until well into the decompression phase of a pressure exposure (1,3,7,9,10,12,14).

In contrast with repeated associations of prominent EEG changes with rapid compression to increased hydrostatic pressures, similar alterations did not occur during the multi-day stepwise compression of Predictive Studies III

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to a simulated depth of 1200 fsw and helium saturation exposure at that pressure (15). One possible interpretation of these results is that the effects of some degree of hydrostatic pressure on brain electrical activity can be avoided if the rate of compression is sufficiently slow either to prevent their occurrence entirely or to allow concurrent adaptation.

Predictive Studies IV was designed to intentionally impose significant stress of compression rate and degree upon normal men, relating electroencephalographic to other functions. In particular the intent was to monitor onset and any adaptation to more rapid compression than had previously been investigated in sustained high pressure exposure. The plan and design of the overall study incorporating electroencephalographic measures is described in Sections C and D.

As in the overall Predictive Study, it was considered important to accomplish the electroencephalographic measurements during helium-oxygen breathing, without the presence of narcotic inert gases which might mask important changes being sought. It was further planned to investigate effects of central nervous system activity in exercise upon the compression-induced changes in brain electrical activity.

The electroencephalographic records were supplemented systematically in the experiment modules with measurements of visual and somatosensory evoked cortical responses. The combined monitoring allowed extensive tracking of improvements or deteriorations in brain electrical activity over multi-day exposures to the increased pressures. It has also permitted correlation of changes in brain electrical activity with measures of sensory and neuromuscular function, along with concurrent evaluation of mental and psychomotor performance.

METHODS

The recording of electroencephalographic activity was accomplished in both phases of Predictive Studies IV, using the pre-planned modular recording periods described in Section D. Only the major findings in the Phase II

records are described in this report. The less prominent effects observed in the lower pressure studies of Phase I will be presented in a subsequent report. Complete EEG recordings obtained in each module throughout the Predictive Study have been preserved.

GENERAL PROCEDURES

Electroencephalographic activity was recorded continuously in rest and exercise subjects throughout each initial compression to a 1200-fsw pressure equivalent and during the first two excursions to 1600 fsw. Recording from the rest subject was done on a 16-channel Grass model 6 electroencephalograph; an 8-channel Grass model 6 electroencephalograph was used for the exercise subject. Amplification of both recorders was 7 μV/mm and frequencies were filtered above and below a range of 1 to 70 Hz.

Platinum alloy, subdermal needle electrodes (Grass Instrument Co. model E2B) were positioned in accord with the universal 10-20 electrode placement system, with slight modifications to facilitate recording of somatosensory evoked cortical potentials. Electrode placements for the rest and exercise subjects are shown in Fig. 1A and B. Several electrode placements were included as backups against the possible loss of one or more electrodes during a recording period. Accurate placement of electrodes was assured by removing small (1 cm^2) patches of scalp hair at the appropriate locations.

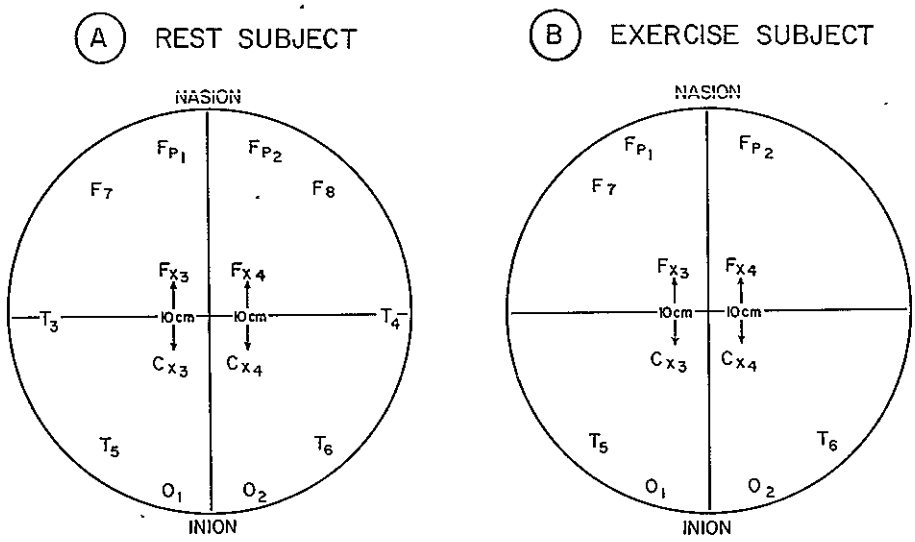

FIG. 1. Positions of EEG electrodes.

DAILY PREPARATION FOR EEG RECORDING

All four subjects were thoroughly trained in the techniques of electrode application and performed these procedures on each other throughout the pressure exposure when they were isolated from the investigators. After the scalp area for placement was cleaned of oils, the needle was inserted and the insertion site was covered with a circular patch of ventilated adhesive. The entire scalp electrode assembly was cushioned with cheesecloth padding and secured by a specially designed, snug-fitting, well-ventilated cap sewn from nylon mesh fabric.

The electrode wires were gathered into a bundle at the nape of the neck and fastened to the cap for stress relief. Each pin-tip connector was placed in the proper receptacle of a miniature Grass EEG junction box (model MEB-INT25) attached to the back of the subject's shirt. The cable and connector from this junction box were replaced by a length of flexible, flat cable(component of umbilical cord--see Section D) and a flat connector adapted from a printed circuit board. Insertion of the flat connector into a ceiling-mounted receptacle produced electrical continuity (via 2 ma fuses in each lead) with the electrode selection panel of the electroencephalograph located outside the chamber. The bilateral EEG montages used for the two subjects are shown in Table I.

Impedances of all bipolar electrode connections were tested immediately prior to the start of each recording session and were checked intermittently throughout each session. Measured values for the subdermal needle electrodes never exceeded three thousand ohms.

EEG MONITORING AND ANALYSIS

The EEG records obtained during the initial compression from sea level to 1200 fsw and during the first two excursions to 1600 fsw were monitored continuously as they were produced, by an EEG specialist member (JOD) of the investigator group. The extensive and detailed visual analysis of all records was performed subsequently by another EEG investigator (JDB). The frequency and prominence of observed EEG responses to different exposure conditions were determined subjectively by comparison with control

records obtained during training and immediately prior to compression from one ata. At the present phase of record analysis, the primary emphasis is on accurate description of the observed electroencephalographic responses.

TABLE I. Bilateral Montages for Electroencephalographic Study in Rest and Exercise Subjects

Channel Number	Electrodes	
	Rest Subject	Exercise Subject
1	$Fp_2 - F_8$	$Fp_2 - Fx_4$
2	$F_8 - T_4$	$Fx_4 - Cx_4$
3	$T_4 - T_6$	$Cx_4 - T_6$
4	$T_6 - O_2$	$T_6 - O_2$
5	$Fp_1 - F_7$	$F_7 - Fx_3$
6	$F_7 - T_3$	$Fx_3 - Cx_3$
7	$T_3 - T_5$	$Cx_3 - T_5$
8	$T_5 - O_1$	$T_5 - O_1$
9	$Fp_2 - Fx_4$	
10	$Fx_4 - Cx_4$	
11	$Cx_4 - O_2$	
12	$Fp_1 - Fx_3$	
13	$Fx_3 - Cx_3$	
14	$Cx_3 - O_1$	
15	$O_2 - C_z$	
16	$O_1 - C_z$	

RESULTS

All measurements of brain electrical activity obtained at sea level and during pressure exposure were studied intensively and repeatedly compared with each other visually. Characteristically, the observed changes were intermittent with varying degrees of frequency and prominence. Quantitative computer analytical methods have not yet been used in this examination because a complete survey of all channels recorded is technically

impractical and results would, therefore, be heavily influenced by the required selection of specific segments for analysis. Since the functional significance of the specific changes observed in brain electrical activity in compression is not known at the present time, such selected results would still not be interpretable.

CONTROL MEASUREMENTS

Control measurements of brain electrical activity at atmospheric pressure before the initial compression were obtained on two occasions in the electroencephalographic laboratory and a third record was obtained several weeks later in the environmental chamber immediately prior to compression. It is important with regard to interpretation of compression effects that noteworthy variations in these control records were found in three of the four Phase II subjects. Although the observed variability was limited to mild asymmetry during sleep in subject FS, it was more prominent in subjects MP and CC. Marked differences found in subject MP on one occasion in the EEG laboratory several weeks before compression could be explained at least partially by alcohol ingestion on the previous night. Some deviations in this control record were more prominent than those found later at increased ambient pressures. However, several control records for subject CC in the absence of prior day alcohol ingestion also showed variations which resembled those later found at pressure. These variations included asymmetry of posterior sleep activity and an increased incidence of lower frequency forms over several areas of brain cortex.

ELECTROENCEPHALOGRAPHIC RESPONSES TO COMPRESSION AND INCREASED HYDROSTATIC PRESSURES

The EEG responses of all four subjects to the initial compression from sea level to a 1200-fsw pressure equivalent are summarized in Table II. The observations are grouped into four recording periods: compression to 800 fsw; the two-hour stable hold at 800 fsw; compression from 800 fsw to 1200 fsw; and the first five hours of the stable saturation exposure at 1200 fsw.

Tables III and IV summarize the EEG changes observed before, during and after the first and second excursions from 1200 to 1600 fsw. The observations in both tables are grouped into five recording periods: a pre-excursion period at 1200 fsw; compression from 1200 fsw to 1600 fsw; the 42-55 minute hold at 1600 fsw; decompression from 1600 fsw to 1200 fsw; and a post-excursion period at 1200 fsw.

Specific changes in the EEG appearance that were observed during compression and exposure to increased hydrostatic pressures are indicated in Table V, and the subjects in whom these changes occurred are indicated. Examples of the various EEG alterations are shown in Figs. 2-7. The times at which the illustrated recordings were made are shown in relation to the exposure profile in Fig. 8.

The EEG changes listed in Table V were progressive with increasing pressure of exposure in all four subjects. Another consistent finding in every subject, however, was that the EEG changes associated with the first excursion to 1600 fsw were significantly diminished or absent during the second excursion to the same pressure (Tables III and IV). This apparent adaptation was most obvious in subject GM whose EEG record during and after the second excursion to 1600 fsw was essentially the same as that observed at 1200 fsw prior to the excursion (Table IV).

In contrast with the marked adaptation of EEG responses to the 1200-1600 fsw excursions, the EEG appearance at 1200 fsw was not altered consistently throughout the multi-day saturation exposure. Over the first three days at 1200 fsw, the EEG of subject FS showed progressive accentuation of the observed changes with increases in both their frequency and prominence. On day 8 of the saturation exposure, his EEG appearance at 1200 fsw resembled that recorded on day 2 (i.e., changes more marked than on day 1 and less than on day 3). Although the other three subjects showed day-to-day variability in EEG appearance over the first three days at 1200 fsw, the incidence and prominence of changes were neither increased nor decreased consistently. On exposure day 8, the EEG patterns of subjects CC and GM were not appreciably different from those observed during the first three days at 1200 fsw, while the EEG of MP showed increased lability with intermittent shifting to accentuation or diminution of the previously observed changes.

TABLE II. Electroencephalographic Changes During Rapid Compression to a Pressure Equivalent to 1200 fsw.

SUBJECT	CHANGES IN ELECTROENCEPHALOGRAM
	Compression from One ata to 800 fsw (50 min)
CC	Minor, intermittent, asymmetric 4-7 Hz activity over frontal and central areas.
GM	No truly significant change. On two occasions mild disorganization of activity and lower frequency forms (5-7 Hz) observed over frontal and central areas bilaterally but more prominent on right.
MP	Compression beyond 680 fsw associated with increased proportion of lower frequency forms (8 Hz) intermingled with background activity over central, temporal, posterior and occipital regions.
FS	No significant change.
	Period of Stable Pressure at 800 fsw (120 min)
CC	Intermittent mild increase in relative amount of 5-12 Hz activity at low to moderate amplitude over pre-frontal, frontal and inferior frontal regions. Mild degree of asymmetry with greater prominence independently on either side. Additional occurrence of scattered 4-7 Hz activity.
GM	Paroxysmal lower frequency (range 1-11 Hz) and sharp forms at amplitudes higher than background occurred in brief bilateral bursts over frontal and central regions with varying asymmetry.
MP	Increased prominence of intermittent poorer organization of background activity with relatively lower frequency forms at higher amplitude.
FS	Definite mild, intermittent, lower frequency forms (5-7 Hz) with some low amplitude, relatively amorphous, irregular activity as low in frequency as 2 Hz occurring asymmetrically on either side with slightly greater frequency on right.
	Compression from 800 to 1200 fsw (40 min)
CC	Much of record obscured by artifact. Slightly increased incidence of the same changes as noted during initial 0-800 fsw compression.
GM	Continued sporadic, higher amplitude activity over frontal and central areas. Progression of paroxysmal, lower frequency and sharp forms. More obvious, 5-7 Hz frontal and central activity bilaterally and asymmetrically.
MP	No additional changes noted from 800 fsw.
FS	Progression of changes observed at 800 fsw with especially noticeable increases in 4-9 Hz activity near vertex and 5-7 Hz activity over frontal and central areas bilaterally.

TABLE II (cont'd).

SUBJECT	CHANGES IN ELECTROENCEPHALOGRAM
	First 5 Hours of Exposure at 1200 fsw
CC	No additional changes from 800 fsw initially. Marked, transient (2-min) changes occurred after about 3.5 hrs at 1200 fsw--characterized by disorganization of anterior activity and appearance of moderate amplitude, very irregular forms as low in frequency as 1 Hz in the anterior areas (Fig. 6).
GM	Initial increases in proportion of 6-7 Hz activity over frontal areas at low amplitude and 5-7 Hz activity at slightly higher amplitude over central regions. Subsequently, 4-6 Hz activity over frontal and central areas became less apparent and 7-11 Hz activity predominated. Other changes included intermittent disorganization of frontal and central background activity and intermittent trains of 4-7 Hz rhythmic activity over anterior areas (Figs. 4 and 5).
MP	Trains of rhythmic, higher amplitude, 7-8 Hz activity continued to appear intermittently over temporal and central regions. Intermittent disorganization and lower frequency components of background activity also continued. Later when subject activity increased at 1200 fsw, lower frequency forms in general were not as apparent, though still greater than at sea level.
FS	Increased 5-7 Hz activity over frontal, central and sometimes temporal areas. One definite larval, paroxysmal burst of spike and lower frequency activity more prominent on right. Lower frequency components of background activity were apparent during sleep (Fig. 7).

This table summarizes the qualitative characteristics of EEG alterations observed in all four divers at various stages of the initial compression from one atmosphere through a holding period at 800 fsw to the first day arrival at the pressure equivalent of 1200 fsw. Duration of each stage is indicated in the table.

The EEG changes described are those which were judged by an experienced, clinical electroencephalographer to represent noteworthy deviations from control records obtained at one atmosphere just before compression. In this form of analysis, emphasis has been placed on relative actual characteristics of the EEG records, with no attempt to quantitate the absolute frequency or prominence of the observed changes.

This laborious direct approach was used to avoid missing changes when, on initial evaluation, it was learned that the majority of EEG alterations which did occur were intermittent, brief and not prominent in degree. Large portions of the records from each subject contained only minimal modifications or were essentially normal in appearance. The examples selected for illustration in Figs. 2 to 7 represent the most obvious changes.

TABLE III. Electroencephalographic Changes Before, During and After the First Excursion From 1200 to 1600 fsw. (Duration of each excursion stage is indicated in the table. See legend of Table II for additional details relevant to interpretation of the tabulated information.)

SUBJECT	CHANGES IN ELECTROENCEPHALOGRAM
	Pre-Excursion Period at 1200 fsw (60 min)
CC	Normal patterns delineated less readily than on previous day at 1200 fsw with disorganization of background activity. Greater tendency toward 7-8 Hz forms over posterior regions. Intermittent occurrence of 1.5-7 Hz forms over frontal, central and temporal areas.
GM	Continuation but with decreased prominence of changes noted previously at 1200 fsw. Disorganization of background activity especially over central regions, and lower frequency components of background activity over frontal and central areas. Intermittent and scattered paroxysmal occurrence of lower frequency forms (as low as 1.5 Hz) asymmetrically on either side as well as bilaterally.
MP	Greater proportion of low frequency activity than at 1200 fsw on the previous evening. Background activity in alpha frequency range intermittently at 9-13 Hz but with frequent preponderance of 7-8 Hz components. Although central activity occurred in rhythmic trains with predominant frequency of 10-11 Hz, there were frequent, generally rhythmic trains of higher amplitude activity with a varying degree of disorganization and a predominant frequency of 7-9 Hz. Low amplitude, irregular forms (4-6 Hz) were intermingled with background activity (especially that at 7-8 Hz) and even lower frequency forms (as low as 1 Hz) occurred on scattered occasions (Figs. 2 and 3).
FS	Appearance essentially the same as at 1200 fsw on the previous evening. Continued intermittent, lower frequency activity most prominent at vertex but also over frontal, central and posterior temporal areas. Type delineation remained definite.
	Compression from 1200 to 1600 fsw (40 min--GM, FS) (20 min--CC,MP)
CC	Increased incidence of lower frequency forms over anterior areas with intermittent predominance of somewhat rhythmic 3-5 Hz activity. Posterior activity contained prominent 5-7 Hz forms. Disorganization of activity also accentuated.
GM	Mild, transient increase in disorganization of background activity with no real change in EEG appearance during compression.
MP	Lability of background activity continued during compression. During periods of decreased alpha activity, occasional trains of moderate amplitude, 6-7 Hz activity appeared with intermingled activity as low in frequency as 3 Hz over central areas and 5-7 Hz over temporal areas. During periods of alpha abundance, intermittent appearances of increased incidence of low frequency activity and greater degree of background disorganization.
FS	Increased amount of intermittent, lower frequency activity near vertex, over temporal and posterior temporal areas, and especially over frontal and central areas.

TABLE III (cont'd).

SUBJECT	CHANGES IN ELECTROENCEPHALOGRAM
	Period of Stable Pressure at 1600 fsw (42 min--GM, FS) (55 min--CC, MP)
CC	Continued occurrence of changes noted during compression with further accentuation of disorganized activity and lower frequency forms over anterior and superior regions.
GM	Increased disorganization of anterior activity with accentuation of lower frequency background components, lower frequency forms in general, and sometimes rhythmic activity over anterior areas. However, changes were less paroxysmal and lower frequency forms less in amplitude than at 1200 fsw on previous evening.
MP	EEG appearance similar to that at 1200 fsw for much of recording period. However, the very low frequency forms most obvious at the vertex occurred more frequently, and background forms contained a slightly greater proportion of relatively low frequency components.
FS	EEG appearance essentially the same as that noted during compression to 1600 fsw.
	Decompression from 1600 to 1200 fsw (89 min)
CC	Transient increase in degree of disorganization and lower frequency activity generally over anterior areas occurred immediately after start of decompression. Intermittent disorganization and lower frequency forms (but higher than at 1600 fsw) occurred throughout decompression with progressively decreasing incidence at lower ambient pressures.
GM	Continued occurrence of changes noted previously.
MP	Intermittent changes continued as before with occasional, more prominent disorganization of background activity and greater content of lower frequency components. Also occasional, brief bursts of high amplitude activity at 2 Hz over right frontal area with some reflection on left.
FS	Increased incidence of intermittent, lower frequency forms, which began during compression to 1600 fsw, continued during decompression to 1200 fsw.
	Post-Excursion Period at 1200 fsw (60 min)
CC	Return to EEG appearance observed at 1200 fsw before compression to 1600 fsw. Possibly a slight increase of rhythmic 5-8 Hz activity remained over anterior areas.
GM	EEG appearance generally similar to that observed at 1200 fsw before compression. Continued disorganization of activity and lower frequency forms over frontal and central regions.
MP	Initially following return to 1200 fsw, continued intermittent accentuation of background disorganization, low frequency background activity, and low frequency forms in general. For a time these changes were more prominent than those noted before compression to 1600 fsw and even exceeded those observed at 1600 fsw. About 20 min after decompression to 1200 fsw, EEG appearance gradually returned to that observed at 1200 fsw on the previous evening.
FS	EEG appearance similar to that noted at 1200 fsw before compression.

TABLE IV. Electroencephalographic Changes Before, During and After the Second Excursion From 1200 to 1600 fsw. (Duration of each excursion stage is indicated in the table. See legend of Table II for additional details of procedure.)

SUBJECT	CHANGES IN ELECTROENCEPHALOGRAM
	Pre-Excursion Period at 1200 fsw (60 min)
CC	EEG appearance similar to that observed at 1200 fsw on previous day. Lower frequency forms over anterior areas less prominent in some EEG patterns and more prominent in others.
GM	No significant change in EEG appearance from that observed at 1200 fsw on previous day.
MP	EEG appearance similar to that noted on previous day at 1200 fsw. Slightly decreased incidence of disorganization, lower frequency background components, and lower frequency forms.
FS	Increased incidence of intermittent 4-7 Hz activity over frontal and central areas as compared with activity at 1200 fsw on previous day.
	Compression from 1200 to 1600 fsw (20 min)
CC	Mild increase in lower frequency activity and disorganization of activity over anterior and posterior areas. Changes not as prominent as during compression on previous day.
GM	No significant change in EEG appearance during compression.
MP	Slight accentuation of background disorganization, lower frequency components of background activity, and lower frequency forms. All of these changes occurred less frequently and less prominently than during compression on previous day.
FS	No significant change in EEG appearance during compression.
	Period of Stable Pressure at 1600 fsw (55 min--GM, FS) (44 min--CC, MP)
CC	No further progression of changes noted during compression with suggestion of decreased incidence and prominence of lower frequency forms.
GM	No significant change in EEG appearance.
MP	No further accentuation of changes observed during compression. Alterations less prominent than on previous day at 1600 fsw.
FS	Slightly greater proportion of low amplitude activity at lower frequencies over temporal areas and variable, intermittently increased, low frequency activity over frontal and central areas.

TABLE IV (cont'd).

SUBJECT	CHANGES IN ELECTROENCEPHALOGRAM
	Decompression from 1600 to 1200 fsw (89 min)
CC	Accenuation of lower frequency forms over anterior regions and lower frequency components of posterior background activity continued during early decompression but diminished with ascent to 1200 fsw.
GM	No significant change in EEG appearance.
MP	No significant additional change. Brief bursts of high amplitude, low frequency activity which appeared over right frontal area during decompression on previous day did not recur.
FS	Reversal of changes noted at 1600 fsw.
	Post-Excursion Period at 1200 fsw (60 min)
CC	Background disorganization and incidence of lower frequency forms were decreased to a degree less than that noted at 1200 fsw before compression. Further accentuation in prominence of rhythmic activity at 5-7 Hz over anterior areas.
GM	EEG appearance the same as that observed at 1200 fsw before compression to 1600 fsw.
MP	EEG appearance similar to that noted before compression. Observed changes less prominent than those seen after decompression to 1200 fsw on previous day.
FS	EEG appearance returned to that observed before compression to 1600 fsw.

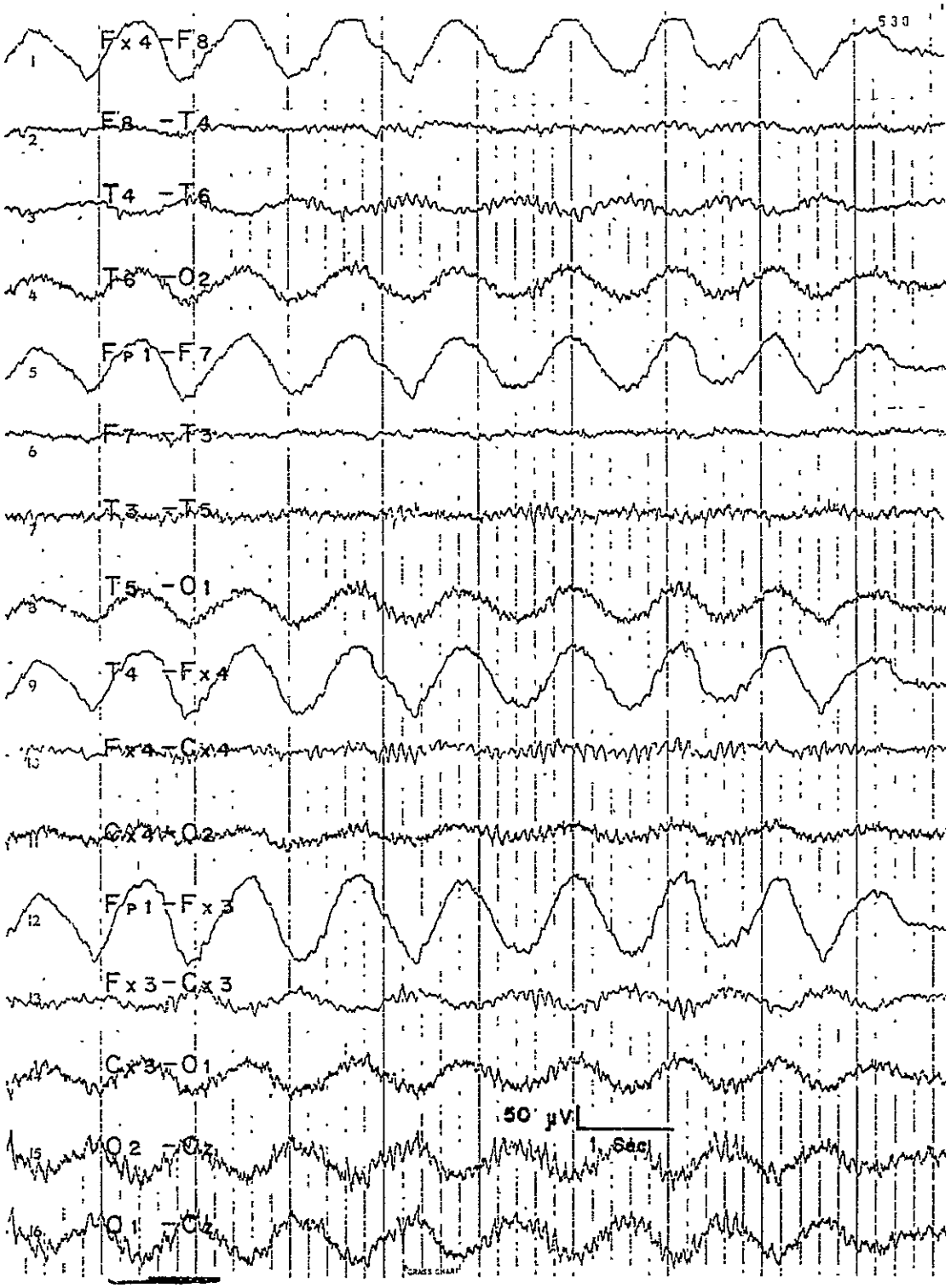

FIG. 2. Left, Subject MP, 1 ata control.

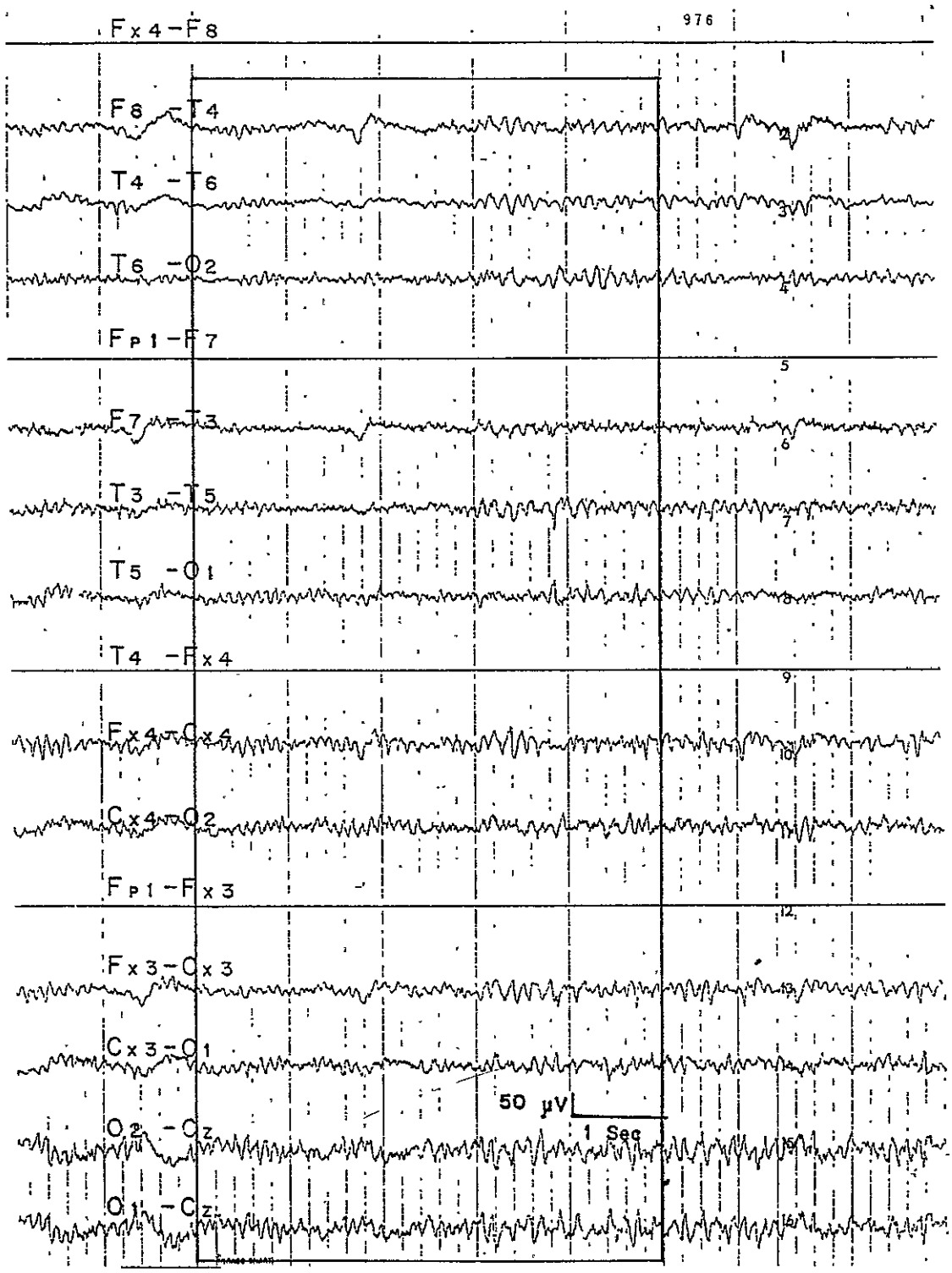

FIG. 2. Right, Same subject, day 2 at 1200 fsw, pre-excursion to 1600 fsw. This record is an example of background lability and lower frequency components of background activity. Changes are most obvious within the blocked area.

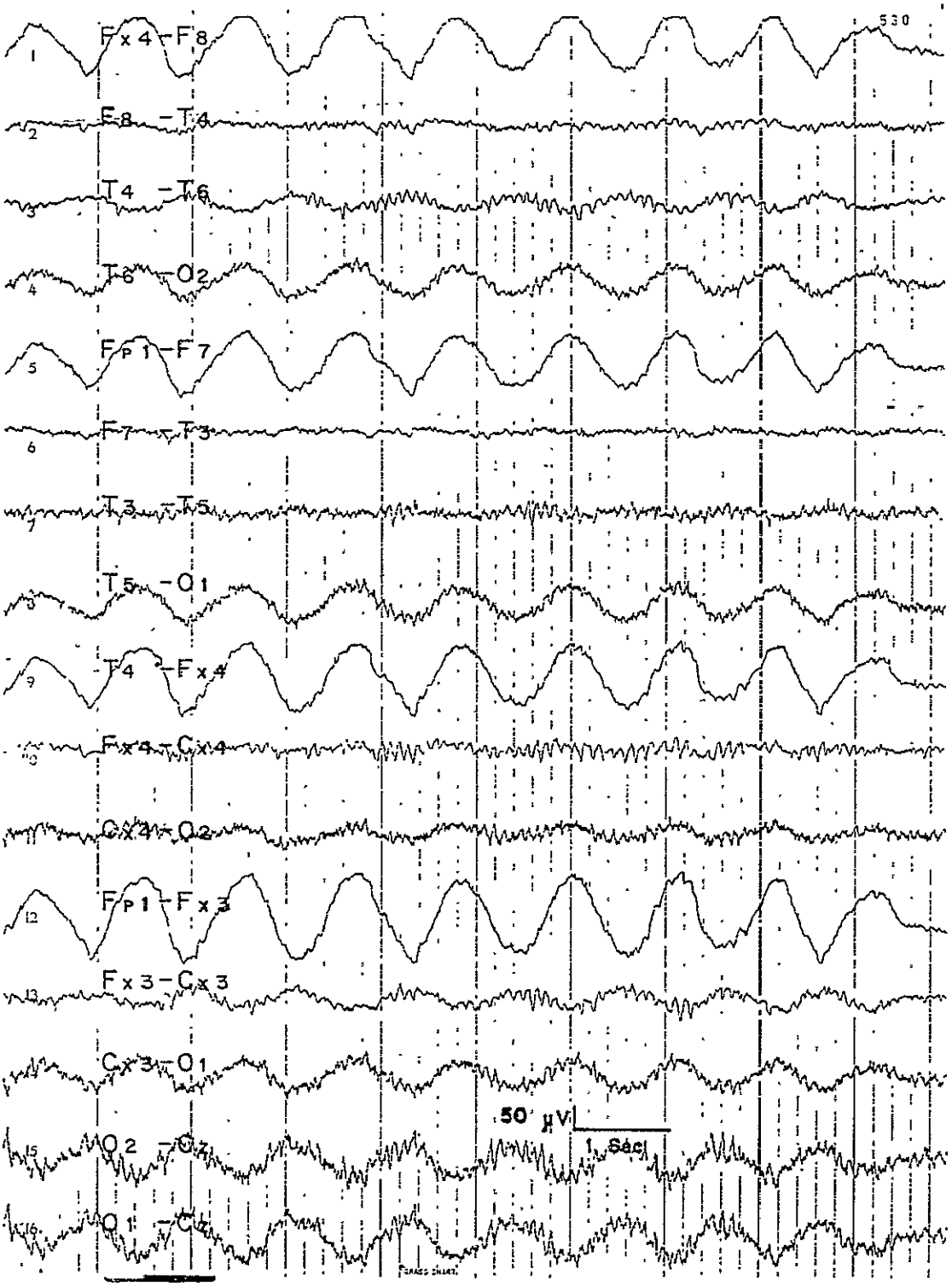

FIG. 3. Left, Subject MP,1 ata control (same as in Fig. 2).

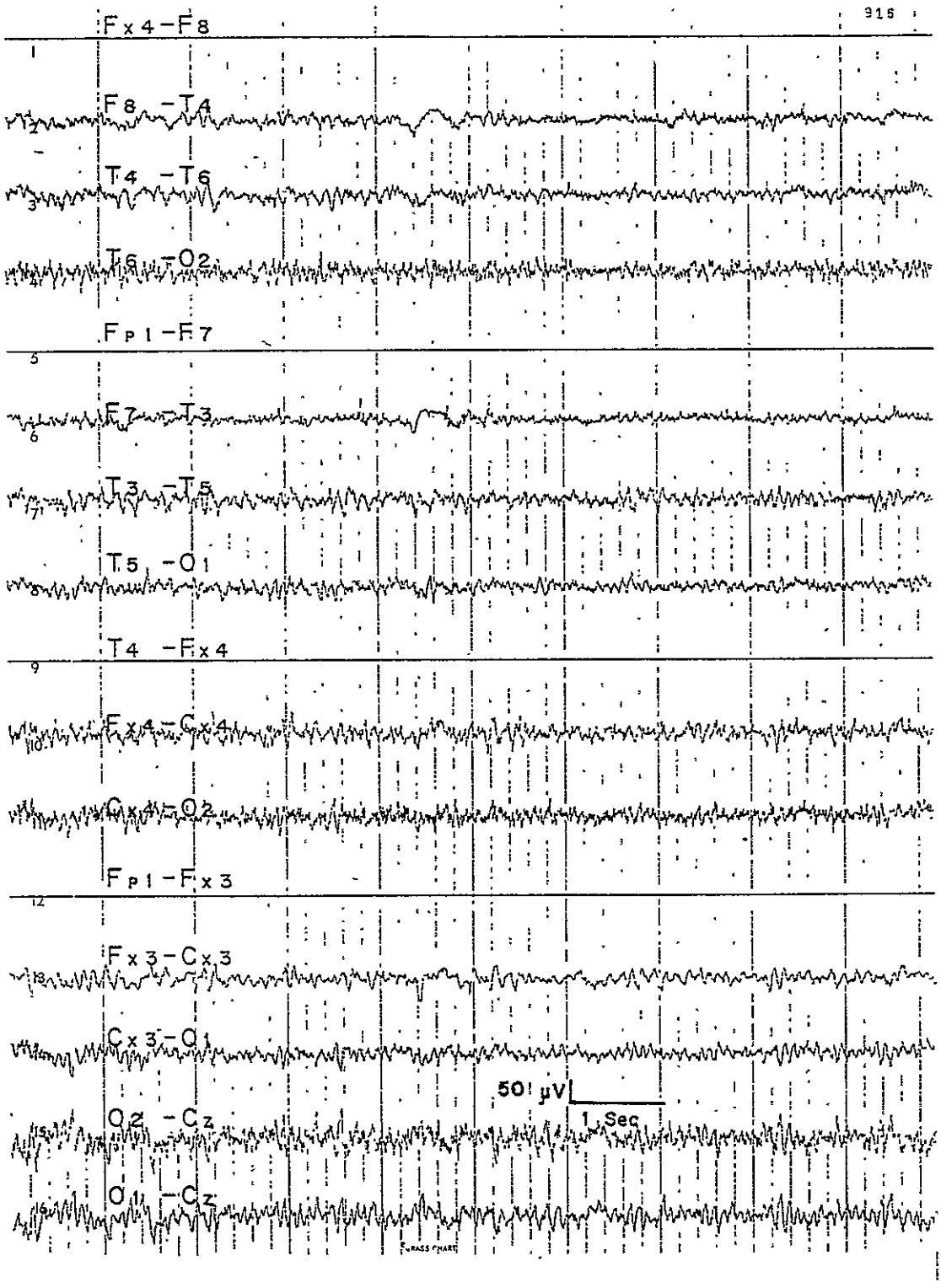

FIG. 3. Right, Same subject, day 2 at 1200 fsw, pre-excursion to 1600 fsw. Intermittent, irregular, lower frequency forms are shown. Lability and disorganization of background with lower frequency background components are also shown on this record.

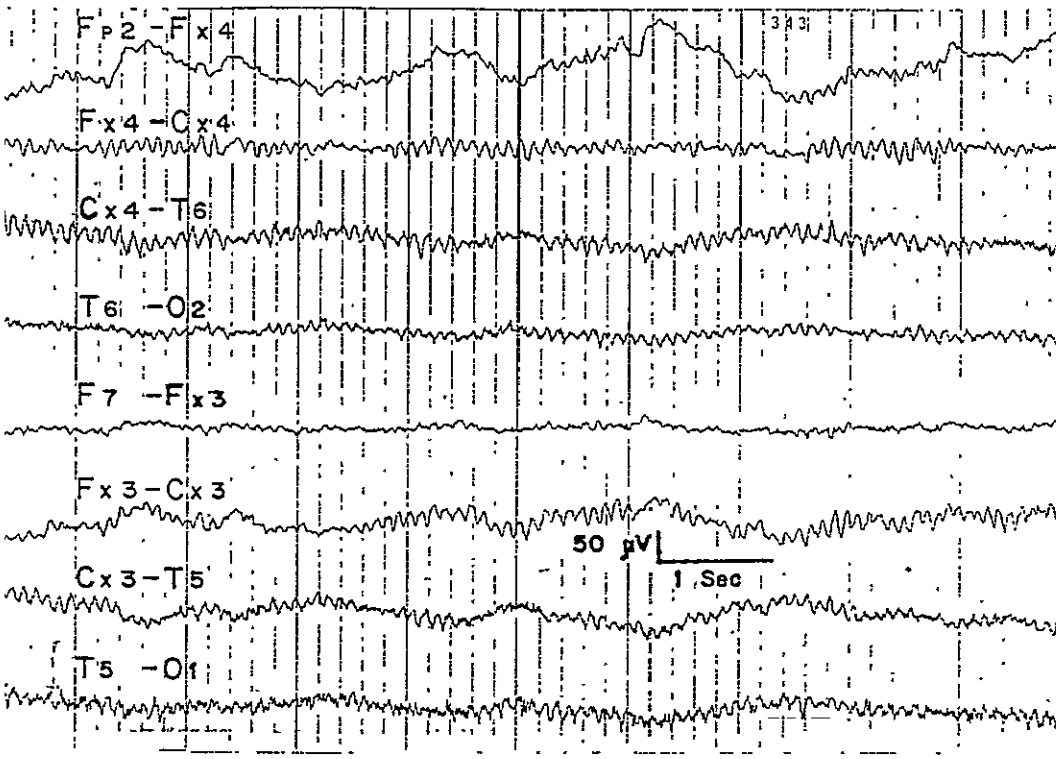

FIG. 4. Left, Subject GM, 1 ata control.

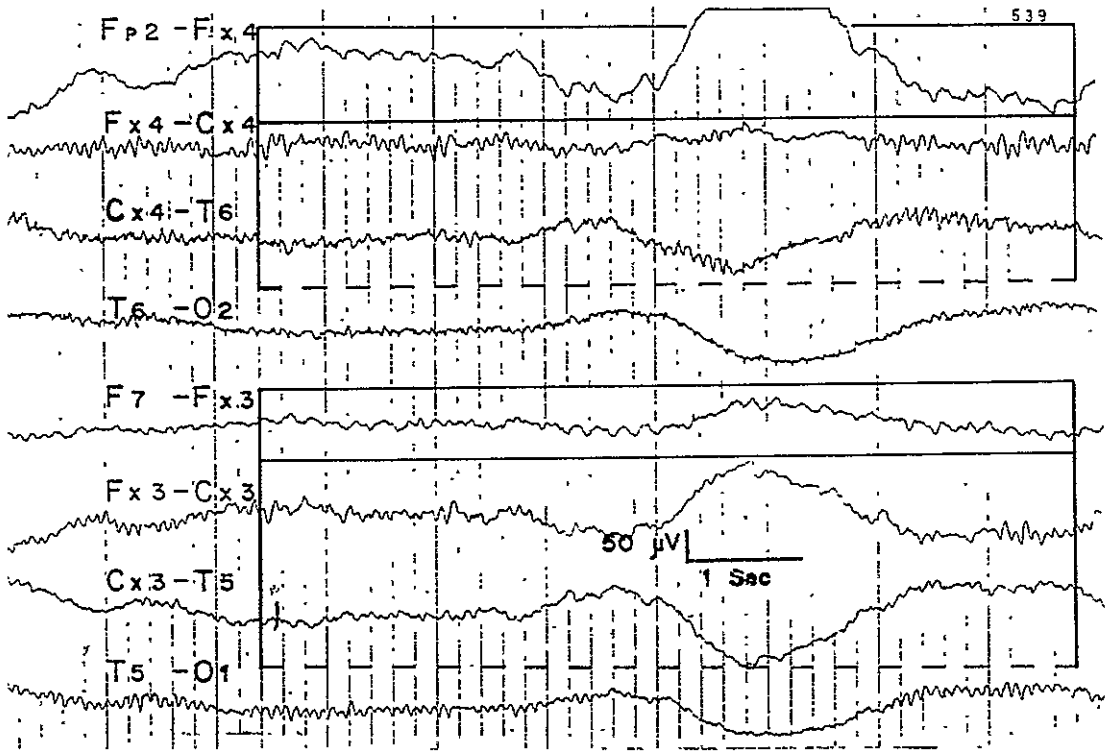

FIG. 4. Right, Same subject, first day exposure to 1200 fsw. This record is an example of rhythmic lower frequency (4 Hz) forms over frontal areas. The most obvious changes are blocked in solid lines. Areas within broken lines show more subtle changes.

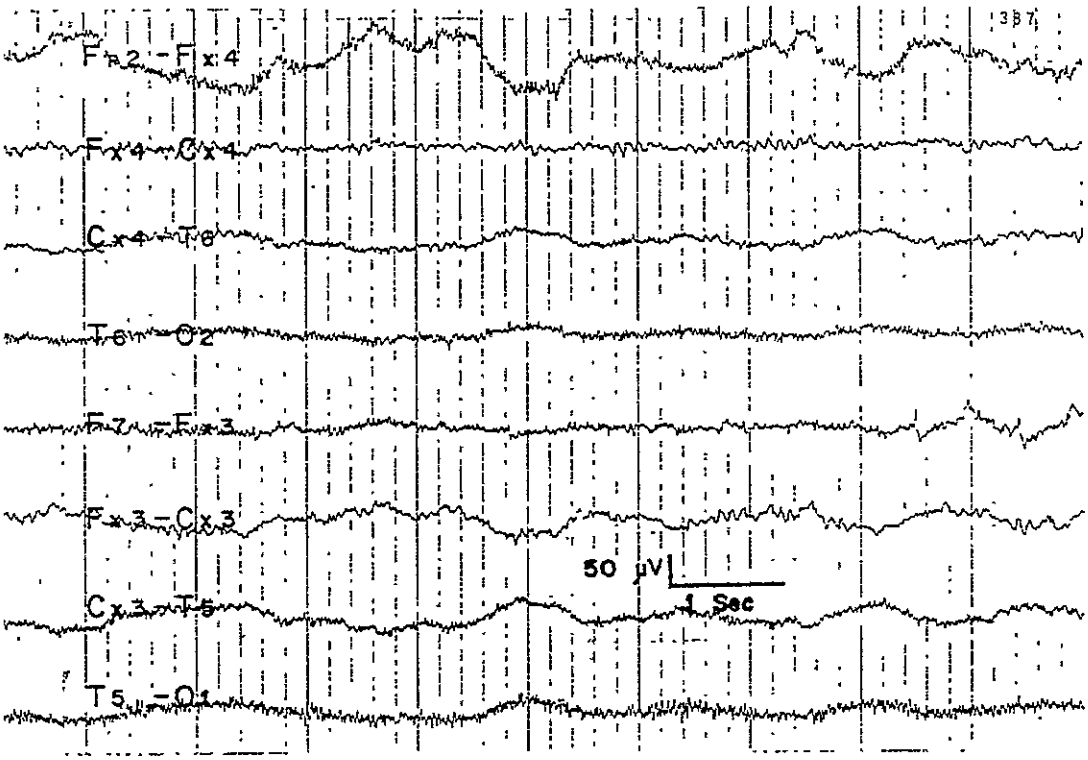

FIG. 5. Left, Subject GM, 1 ata control (same as in Fig. 4).

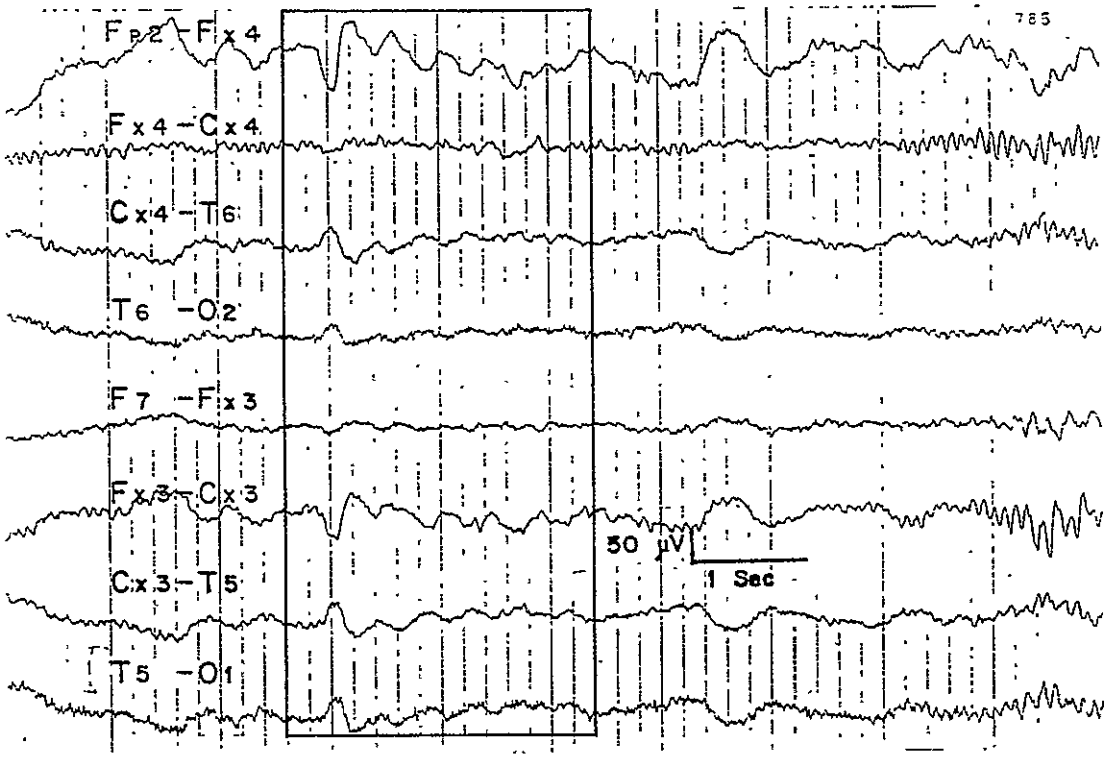

FIG. 5. Right, Same subject, first day exposure to 1200 fsw. Paroxysmal, very low frequency forms are shown. The blocked area shows bilateral brief trains of forms as low as 2 Hz in frequency.

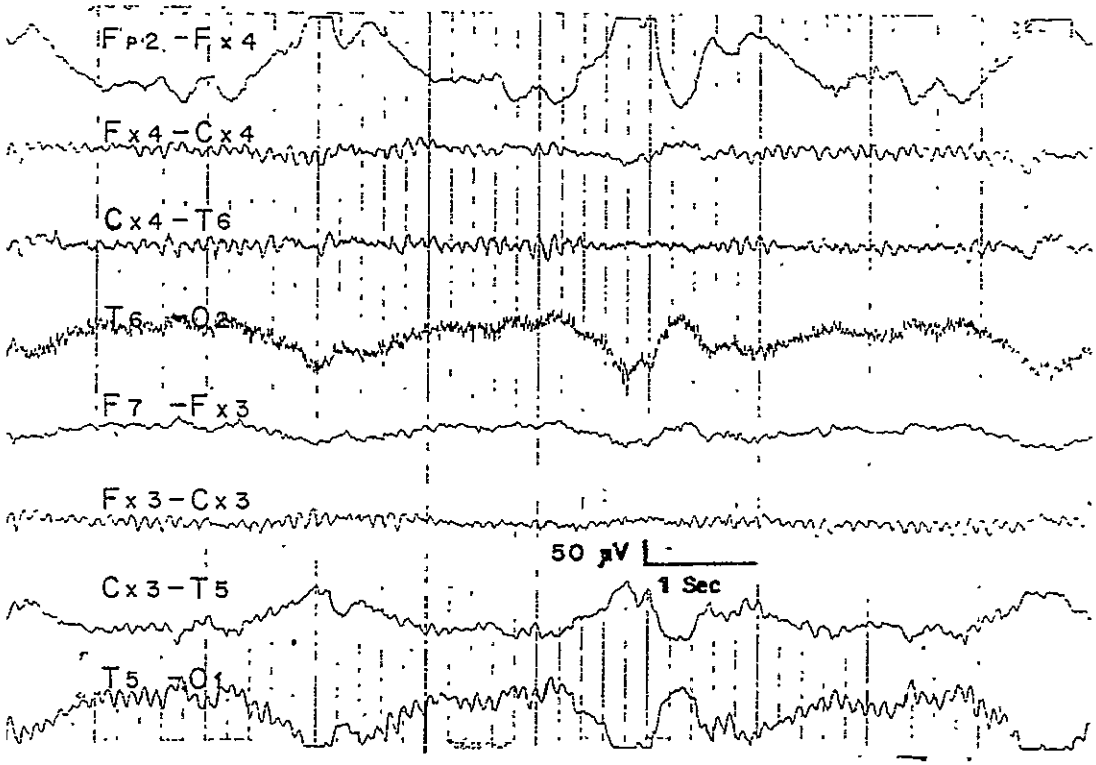

FIG. 6. Left, Subject CC, 1 ata control.

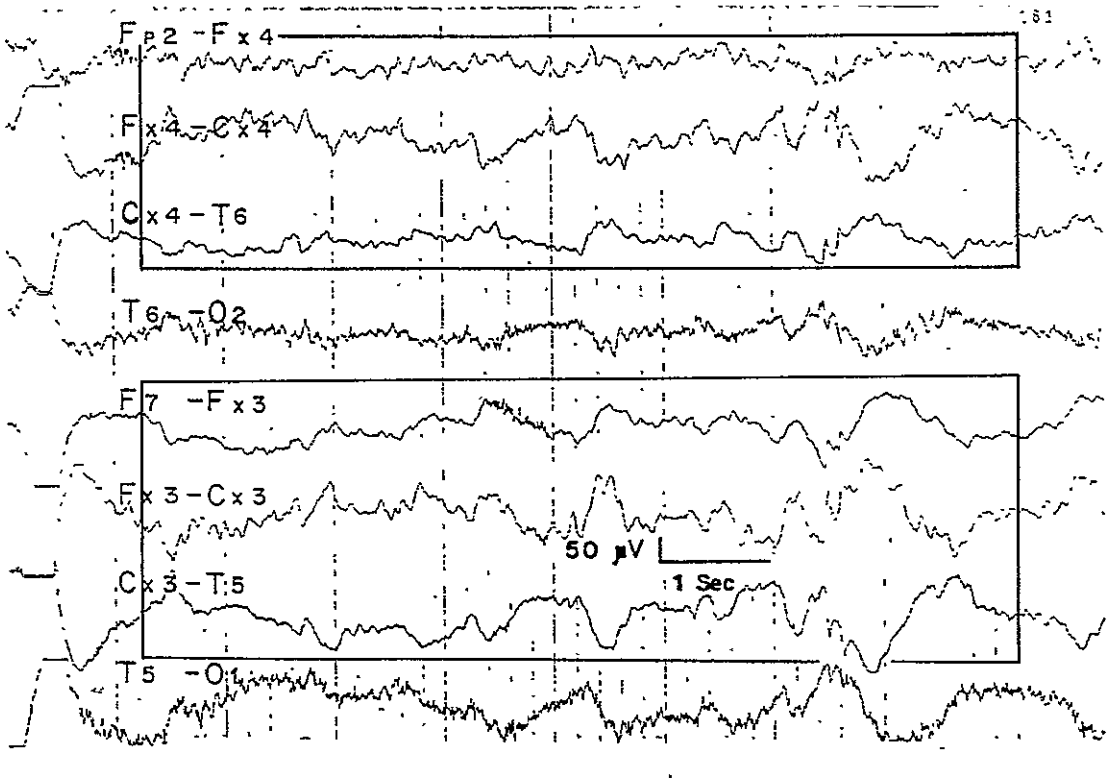

FIG. 6. Right, Same subject, first day exposure to 1200 fsw. This record is an example of paroxysmal, long trains of lower frequency forms. In contrast with the usual intermittent, brief appearance of lower frequency forms, these occurred over an interval of about 2 minutes.

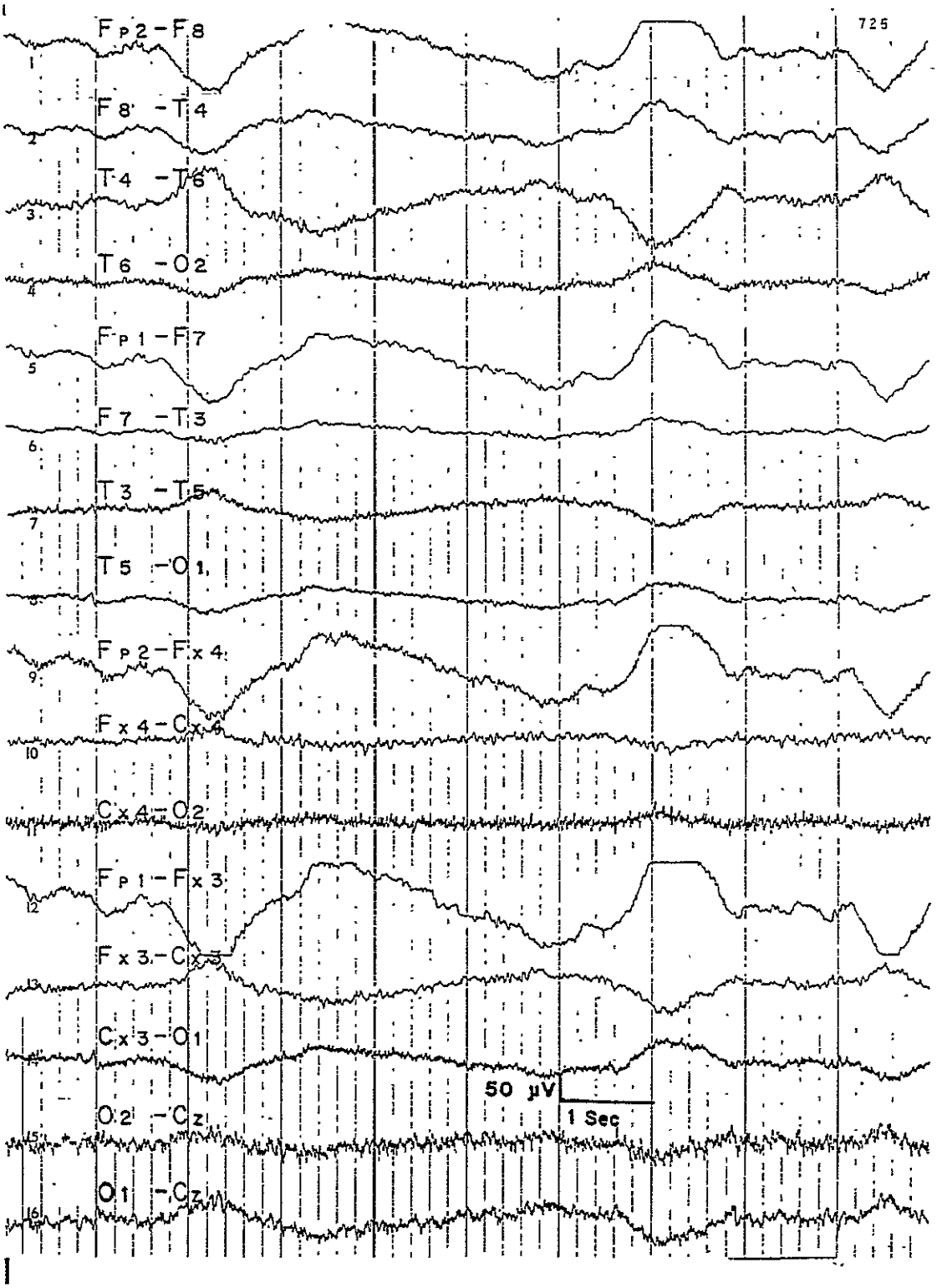

FIG. 7. Left, Subject FS, 1 ata control.

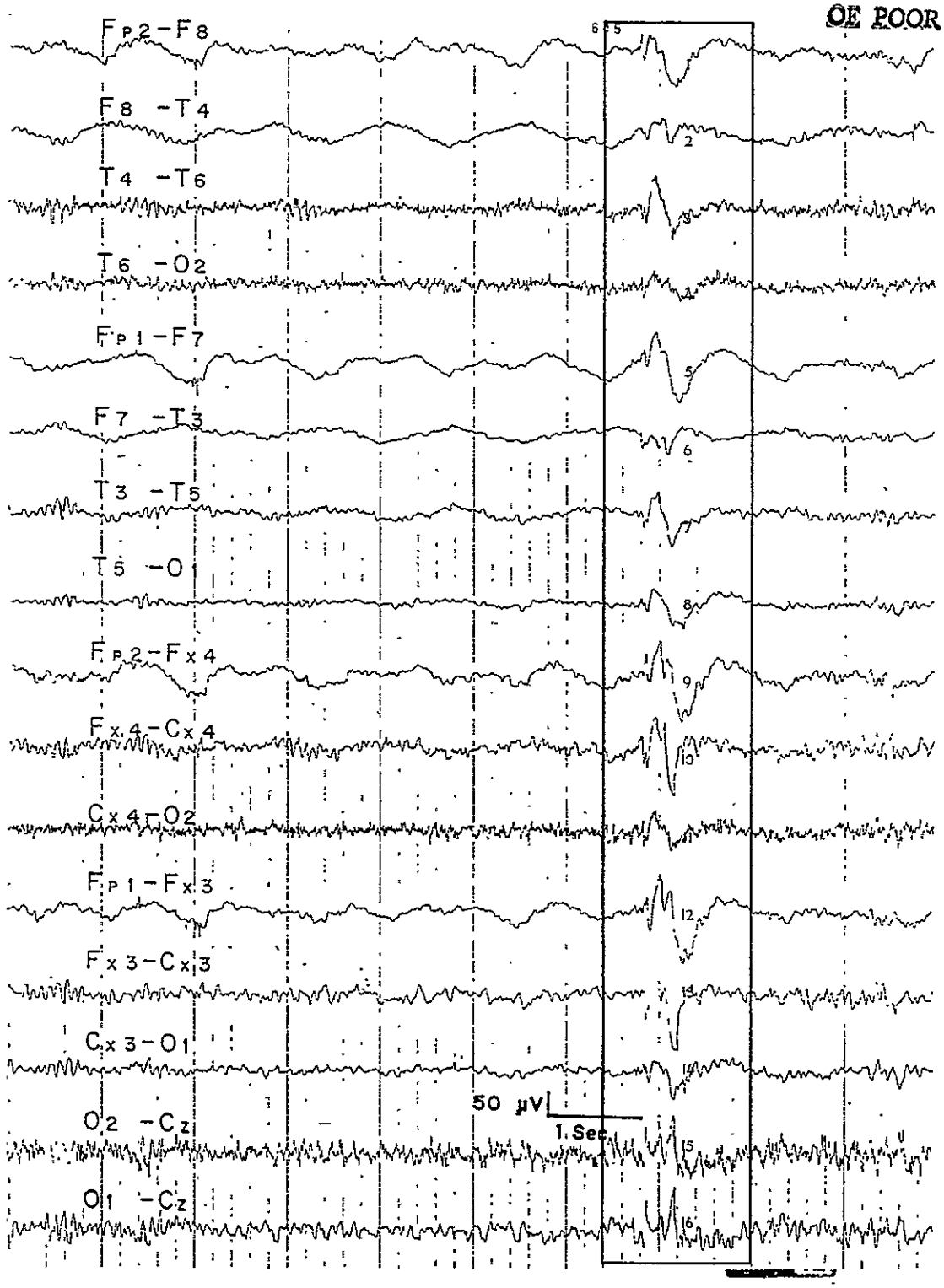

FIG. 7. Right, Same subject, first day exposure to 1200 fsw. Paroxysmal spike wave is shown in the blocked area. This did not reoccur throughout the remainder of the saturation-excursion exposure.

TABLE V. Summary of Electroencephalographic Alterations Observed During Rapid Compression and Exposure to Increased Hydrostatic Pressures

EEG Alteration	Illustration (Figure No.)	Subject			
		Exercise		Rest	
		CC	GM	MP	FS
Background lability	2,3	+	+	+	+
Lower frequency background activity	2,3	+	+	+	+
Intermittent lower frequency forms	3	+	+	+	+
Rhythmic slow activity	4	+	+	0	0
Paroxysmal slow activity	5,6	+	+	+	0
Paroxysmal spike wave activity	7	0	0	0	+
Changes progressive with increased pressure		+	+	+	+
Adaptation to compression effects		+	+	+	+

+ Observed
0 Not observed

Different types of alterations in EEG activity are listed and the subjects in whom they appeared are indicated without specification of the frequency and prominence of the observed changes. Most of the changes were intermittent and brief in duration. For example, the episode of paroxysmal spike wave activity was observed only once, in one subject, during the entire exposure. The most obvious examples of each type of altered EEG activity are shown in Figs. 2 to 7.

POST-EXPOSURE MEASUREMENTS

In all four subjects the EEG recorded at sea level approximately 12 to 24 hours after the end of the saturation-decompression was not strictly identical to that obtained in the environmental chamber immediately before the initial compression. The specific nature of the observed differences varied somewhat from subject to subject.

The EEG appearances of subjects CC and GM post-decompression were similar to those recorded during their saturation exposure at 1200 fsw but the alterations were less prominent and less frequent in occurrence. In addition, in subject GM the central rhythms at sea level were higher in frequency and intermittently better organized than those observed at 1200 fsw

The EEG of subject MP manifested continued frequent occurrence of lower frequency components of background activity, disorganization of background activity, and occurrence of activity as low in frequency as 1.5 Hz over temporal, occipital, and central regions with some asymmetry. These changes were less prominent and frequent than those observed in saturation at 1200 fsw. They were not present in the recording obtained immediately prior to initial compression but resembled alterations found earlier at sea level in the EEG laboratory.

Persistent changes in the EEG appearance of subject FS were not as prominent as those during saturation at 1200 fsw. There was continued intermittent occurrence of increased lower frequency activity with associated disorganization of background activity more prominent on the right.

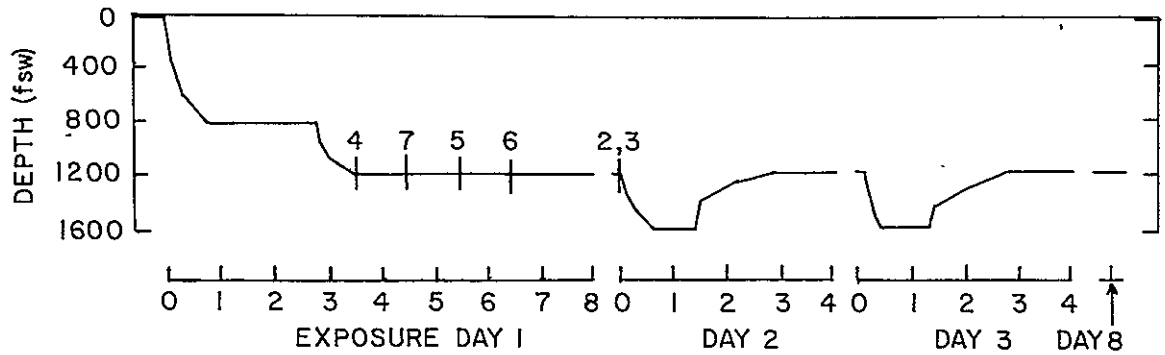

FIG. 8. Numbers on profile for first three days of Phase II represent times of selected EEG record examples and refer to figure numbers in this section (e.g., 2 = Fig. 2).

DISCUSSION

The use of a nearly complete clinical montage for EEG recording in Predictive Studies IV permitted the monitoring of electrical activity over a more extensive area of brain cortex than that surveyed in previous studies of EEG responses to compression and increased hydrostatic pressures (3,12,14). With respect to clinical electroencephalography in which a comparable extensive brain cortical area is normally monitored, little information is available regarding EEG variation with time in a normal subject population, except for a single study in one individual in whom repeated EEG recording showed a 1.5 Hz variation in mean alpha rhythm (8). Both the less extensive EEG monitoring in previous studies and the lack of information regarding normal variability have had to be considered in evaluation and interpretation of the EEG recordings obtained in Predictive Studies IV.

EEG VARIATION AT NORMAL AMBIENT PRESSURE, PRIOR TO COMPRESSION

The serial control measurements which were obtained at atmospheric pressure on three different occasions prior to exposure to increased hydrostatic pressures showed significant differences from time to time in three of the four subjects. Many of these differences represented normal variants that could be related to changes in mental activity, or to the cited probable effect of alcohol. However, some of the observed alterations would be classified in a routine clinical reading of EEG as "abnormal" (i.e., at an extreme of a normal distribution of average individuals without recognized clinical disturbance). In the absence of normal standards relating EEG variations to time in the same individuals, it is not possible to evaluate the significance of the variations found in control records of the subjects in the present study. For this reason the observed EEG responses to compression and increased pressures were compared with sea level control measurements in the same subject without reference to routine clinical standards and terminology concerning "normality." The EEG recording obtained in the environmental chamber immediately prior to the initial compression was therefore used as the primary control standard.

EFFECTS OF COMPRESSION AND INCREASED HYDROSTATIC PRESSURES

Distinct EEG changes observed in all four subjects during the initial rapid compression to 1200 fsw included background lability, lower frequency background activity and lower frequency forms (Table V). Other, less consistently observed EEG manifestations of rapid compression were paroxysmal slow activity in three subjects, rhythmic slow activity in two, and paroxysmal spike waves on one occasion in one subject. All EEG alterations appeared intermittently with varying degrees of prominence. The EEG records in Figs. 2-7 were selected to show the most prominent changes. They do not represent "average" EEG appearances over long periods of time because large proportions of the records from each subject contained only minimal changes.

Deviation of the EEG appearance from the sea level control recording occurred progressively with exposure to increasing pressures in all four subjects. Typically this progression was manifested by increments in both frequency and prominence of the observed alterations. However, despite generally progressive modification of the EEG in parallel with increasing symptoms during the initial compression to 1200 fsw, there was no definite correlation between these events. Severe symptoms and prominent EEG changes appeared to occur independently in individual subjects.

In addition, EEG alterations were not closely correlated with measured decrements in cognitive, perceptual and psychomotor functions (Section E-10). This apparent lack of correlation may be related to the fact that the intermittent occurrence of EEG alterations and the programmed intermittent testing of mental function resulted in only infrequent temporal overlapping of these two events. In a previous exposure to high ambient pressures, Rostain and Charpy (12) also found a general relation of increased EEG theta activity and decreased sensorimotor efficiency but without close correlation of the two phenomena.

Other measures of brain electrical activity which changed in parallel with the EEG alterations were an increased latency and decreased amplitude of visual evoked cortical potentials (Section E-3). Both of these changes were consistently progressive with increasing depth

of exposure and they reflect pressure effects on discrete areas of brain cortex. The changes in alpha frequency (Tables II-IV) were not evident in the localized electroencephalographic recordings of the visual evoked response component of this study. In contrast with the specific nature of the visual evoked response, the EEG modifications described in Table V were generalized, bilateral and often asymmetrical in occurrence. Definite relationships between these specific and general alterations in brain electrical activity cannot now be defined.

The EEG responses to rapid compression and exposure to increased pressures found in Predictive Studies IV are generally similar to previously reported observations in compression (1,3,9,12,14). However, earlier workers have not emphasized the intermittent nature of the EEG changes and some have done frequency analyses of results with computer techniques (3,12,14,15). The use of such methods alone to describe intermittent effects could mask or distort results unless great care is employed in the selection of EEG record segments for analysis.

The EEG responses to rapid compression observed in Predictive Studies IV as well as in previous studies (1,3,9, 12,14) are in marked contrast to the absence of such changes in a slow, stepwise compression over nine days to the simulated depth of 1200 fsw with multi-day stages at 400, 700 and 900 fsw in Predictive Studies III (15). In the latter study, some decrements in mean frequency of EEG activity were observed in drowsy subjects but alpha activity, when present, was unchanged in frequency. However, there was a significant decrease in the amplitude of alpha activity that was progressive with depth, irrespective of the inert gas breathed (He, Ne or N_2).

ADAPTATION TO COMPRESSION EFFECTS

The marked attenuation of EEG responses to the second 1200-1600 fsw excursion-compression, as compared with the changes induced during the first excursion, indicates that definite adaptation to the effects of compression on brain electrical activity occurred between the second and third days of saturation exposure at 1200 fsw. This adaptation was particularly obvious in subject GM whose EEG was

essentially unaltered throughout the second 1200-1600 fsw excursion. The lack of response by subjects GM and FS to the second excursion is even more remarkable in light of the fact that its compression rate was twice that of the first (Section D, Table II). The degree of adaptation to compression effects which occurred between the first and second days of saturation exposure cannot be determined accurately because it was not sensible to effect excursion to 1600 fsw on day 1. However, the probability that significant adaptation did occur during the first 24 hours is supported by the observation that many of the EEG alterations induced by the initial 1200-1600 fsw excursion-compression (Table III) were no more prominent than those found after compression to 1200 fsw on the previous day (Table II).

The apparent absence of consistent EEG adaptation to a stable pressure equivalent to 1200 fsw over the multi-day saturation exposure in this study contrasts with the amelioration of excursion-compression effects to 1600 fsw. Although some of the EEG alterations appeared to diminish with time at stable pressure, others did not change significantly, and still others actually became more prominent. During the same period of time, the increased latency and decreased amplitude of visual evoked cortical potentials partially reversed but did not reach pre-exposure control values (Section E-3).

Bennett and Towse (3) reported that during a staged compression to 1500 fsw, compression-induced increments in delta and theta EEG activity continued at stable pressures of 700, 1000, 1300 and 1500 fsw for about 6 hours and then gradually reverted to near control levels over the next 12 hours. In the slower staged compression to 1200 fsw in Predictive Studies III cited previously (15), delta activity and theta activity were not increased even initially. The persistence of EEG alterations at 1200 fsw following the rapid compressions of Predictive Studies IV is consistent with the observation of Rostain and Charpy(12) that EEG responses to compression persist for prolonged periods at various increased pressures or even progress at stable elevated pressure. Since earlier investigations have not included repetitive excursions over the same range of pressures used in the present study, no other information is available relating to the marked attenuation of EEG responses to such excursions.

POST-EXPOSURE EEG STATUS

The presence of the variations observed before pressure exposure and the absence of extensive control EEG monitoring under similar conditions in a normal (average) population complicate interpretation of the persistent EEG modifications in all four subjects following the end of the saturation-decompression. Rostain (11) and Rostain and Naquet (13) have observed similar, persistent, post-exposure EEG changes for one to two days at normal atmospheric pressure in approximately 30% of the subjects studied, for one to two weeks in approximately 10%, and for as long as three weeks in a single subject. Post-exposure, residual EEG alterations appeared to be more persistent in those individuals who had more prominent changes during exposure. However, it was not possible to correlate functional or behavioral effects with EEG modification either during or after exposure to increased ambient pressures.

Because of the apparent temporary persistence of post-decompression EEG alterations, additional studies of EEG activity at normal as well as at increased pressures will be required to determine the significance of these variations.

Acknowledgment

The authors are grateful for the advice and suggestions provided by Dr. J.D. Frost, Jr. of the Methodist Hospital, Texas Medical Center, Houston, Texas.

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E-3. VISUAL EVOKED CORTICAL RESPONSES

J.A. Kinney[1], R.E. Hammond[2], R. Gelfand[2]
and J.M. Clark[2]

The visual evoked response (VER) in man has been studied during saturation exposures to very high pressures of He-O_2 (14, 19, 20) and to relatively low pressures of N_2-O_2 (11, 18). Reduction of the VER amplitude and increased latency of VER components have been observed in both situations, but without clear correlation with either exposure pressure or degree of inert gas narcosis. The physiological origins or consequences of alterations in VER are not certain since the entire neural mechanisms and pathways involved in generation of its various components are not known. However, with systematic study over an extremely large range of hydrostatic and gas pressures, the patterns of changes should become evident.

The design of Predictive Studies IV (Section D) provided opportunities both to confirm and to extend the previous findings and to ascertain if they were associated with concurrent changes in visual function and eye-hand coordination (Sections E-8 and E-10). VER was measured during rapid compression to pressure equivalents of 400-800-1200-1600 fsw and at stable high hydrostatic pressures, using both slow and rapid stimulation rates to examine different aspects of the visual-evoked response (15, 16).

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[2]Institute for Environmental Medicine, University of Pennsylvania.

METHODS

RECORDING TECHNIQUE AND PROCEDURES

VERs were recorded from a bipolar configuration of the electrode montage worn by each subject for EEG monitoring (Section E-2). The active electrode was O_2, the reference C_Z, and a common ground linked the forehead and neck. The signal taken from the EEG polygraph was recorded onto magnetic tape for subsequent signal averaging and analysis.

The stimulus for the evoked responses was produced by a striped pattern placed over an 8-inch diameter viewport and backlighted by a photostimulator (Grass Instruments, model PS-2) set at maximum intensity. The chamber was darkened during measurement periods. At the subject's viewing distance of 75 cm, the stripes subtended an angle of 50 minutes, and the total pattern, 10 degrees. Two different flash rates were employed, one per second and sixteen per second, with a flash duration of 10 microseconds (μsec). One minute of VER stimulation was recorded at each flash rate. As an additional measure for comparison with EEG monitoring during visual stimulation, the unstimulated EEG was recorded for one minute from the same electrode configuration while the subject stood quietly with his eyes closed. Since the same recording procedures for VER and EEG have been used in a variety of other simulated dives, there is ample experience on the effects of various exposure conditions on these responses (11-14, 18).

Two of the four subjects were designated for electrophysiological study in Phase I (subjects LJ and FS) and Phase II (subjects FS and MP). VERs and EEGs were recorded on these men at prescribed intervals at 1 ata, during compression from 1 ata, at stable pressures and during excursions. In addition, extensive control measurements were made on subjects LJ and FS during preliminary exposures to 60 fsw prior to the start of Phase I. One post-dive measurement was made on each subject.

DATA ANALYSIS

Average Evoked Response

Evoked response averaging was performed with a computer of average transients. Figure 1 shows sample responses to both 1 flash/second (VER 1) and 16 flashes/second (VER 16) for subject FS. The VER 1, averaged for N = 60, has two major components: a positive one at about 100 msec (A) and a negative one at approximately 160 msec (B). These responses were analyzed for the latency of both the A and B components and for the peak-to-trough amplitude between them. This amplitude is the numerical equivalent of the amplitude of A minus the amplitude of B, both measured from the recording baseline. For the VER 16 the amplitude of each of the 16 individual measurements obtained in the one-second interval was determined and the mean and standard deviation calculated, the latter indicating the variability about the mean of the evoked responses.

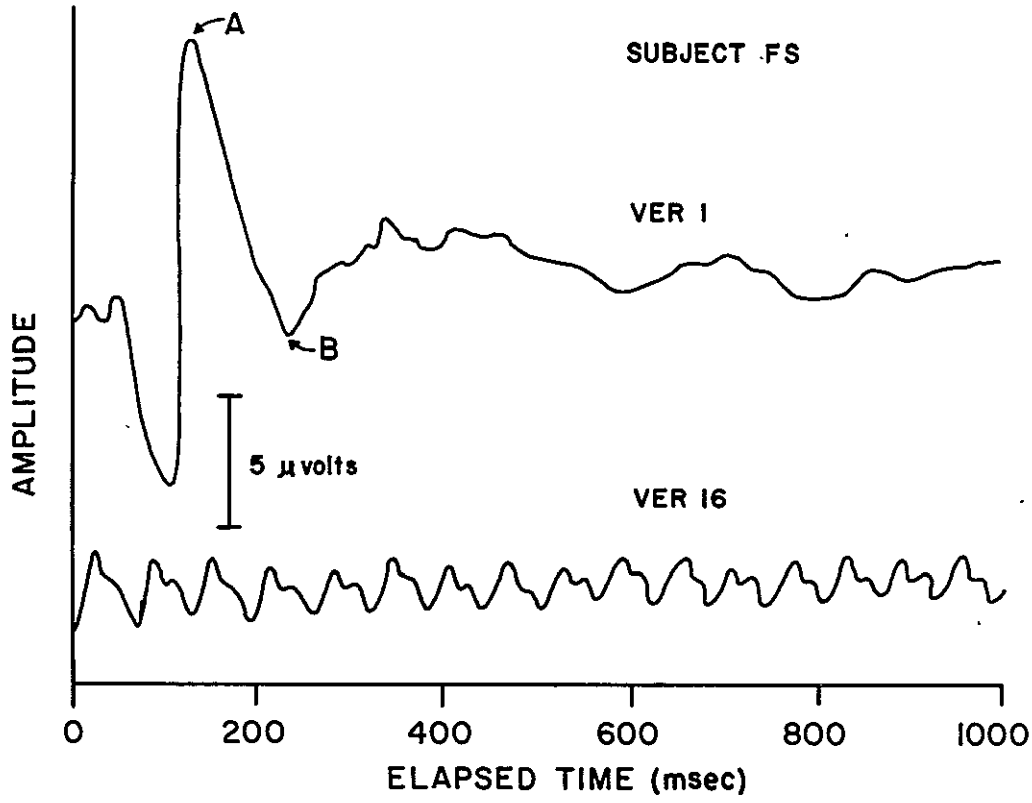

FIG. 1. Example of evoked response to patterned light flashing 1/second (VER 1) and 16/second (VER 16) (subject FS).

Frequency Analysis

Frequency analysis (real-time spectrum analysis with fast-Fourier transform algorithm) of a single channel of raw EEG from leads O_2-C_Z was also performed for all conditions: that is, while the subject watched the light flash at the slow and at the rapid rates, and while standing with his eyes closed. This analysis gave the amplitude at each frequency analyzed in 1/4 Hz steps. Sample data are given in Fig. 2 for subject LJ. The frequency spectrum of EEG recorded while the subject watched the light flashing 16 times/second showed a peak response at 16 Hz. A harmonic peak at 32 Hz was also seen in the analysis for some subjects. The analysis for the flash rate of one/second for subject LJ showed only minor peaks in the theta and alpha regions. Some subjects, however, produced harmonics to the slow rate of stimulation. The eyes-closed condition produced a large response at approximately 10 Hz in the alpha region. The following measures were made from these records: the amplitude at 16 Hz and 32 Hz for the rapid flash rate; the amplitude and frequency of peaks in the theta region (4-7 Hz) and in the alpha region (8-12 Hz) for all conditions. In addition, all records were examined for unusual phenomena.

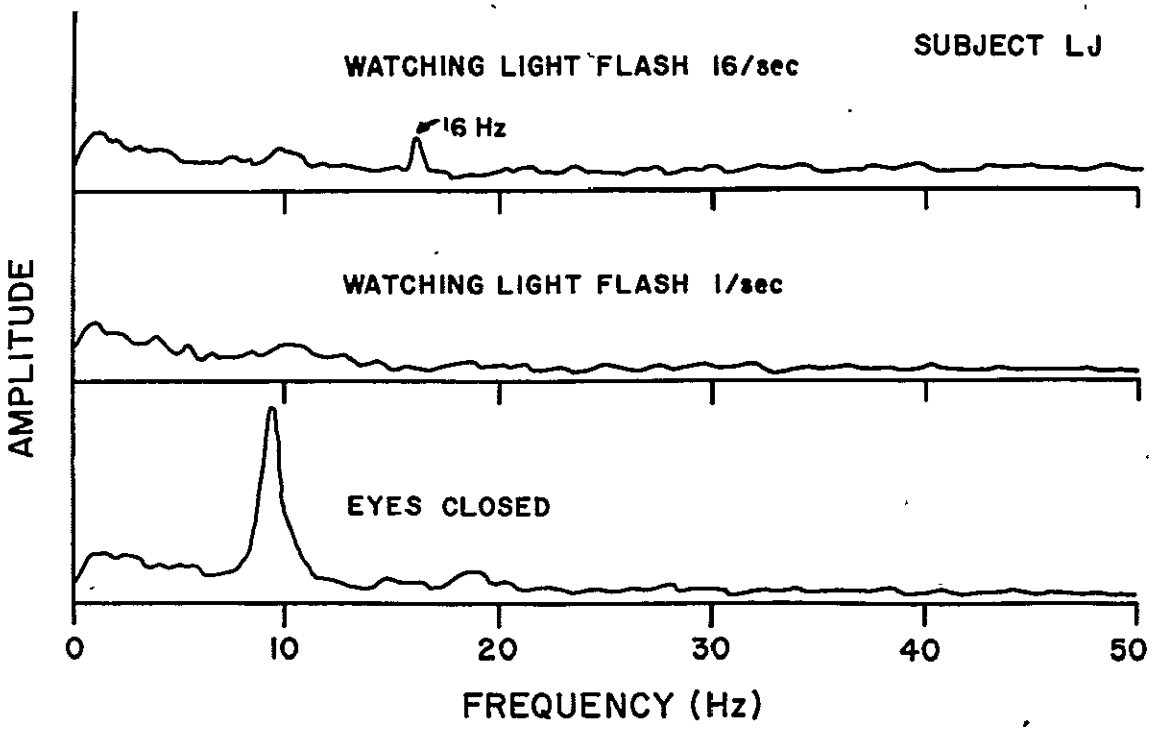

FIG. 2. Example of frequency analysis of the EEG recorded under various conditions (subject LJ).

The types of analyses are summarized in Table I. Since all measurements were made in time segments of one minute this represents a large quantity of data. Only those results which appeared meaningful and significantly different from control data are presented in this report.[3]

TABLE I. Summary of data analysis

Analysis	Light Flashing (1/sec)	Light Flashing (16/sec)	No Light Eyes Closed
VER 1			
Latency of components	X		
Amplitude	X		
VER 16			
Mean amplitude		X	
Standard deviation		X	
Frequency analysis from EEG:			
Theta amplitude	X	X	X
Theta frequency	X	X	X
Alpha amplitude	X	X	X
Alpha frequency	X	X	X
Response at flash rate	X	X	
Harmonics of response at flash rate	X	X	

[3]Additional data are available in tabular form from the authors or from the Institute for Environmental Medicine.

Statistical Analysis

For the statistical analyses, the data were combined in several ways to provide numbers sufficiently large to assess. The analyses were performed largely by *t*-test, since the outcome of the various combinations rarely resulted in the equal or proportional numbers in each group required for straightforward analysis of variance.

Data collected in training exposures at low pressures (to 60 fsw) were used as control values for subjects LJ and FS; experimental protocols and conditions were the same as in the extended exposures, with the exception of pressure and the breathing mixture, which was air.[4] Records made at 1 ata, during low pressure training exposures, and during decompression from these shallow trials were remarkably similar to one another; therefore, it was decided to use these compiled data as an index of normal functioning. Since similar data were not available for subject MP, mean values from the lowest pressures (usually 0-800 fsw) were used in the comparisons and are indicated as "controls" in the tables.

RESULTS

VISUAL EVOKED RESPONSE AT THE SLOW FLASH RATE (VER 1)

Latencies of the second major component of the VER 1 in Phase II are plotted in Fig. 3 for subjects FS and MP as a function of the pressure profile over time. The peak-to-trough amplitudes are similarly plotted in Fig. 4. Mean values of control data (Table II) are shown as solid horizontal lines.

There were increases in the latency of the component for both subjects during the exposures (Fig. 3). Despite these increases in latency during the exposure, the data

[4]Extensive previous experience with this technique of recording the VER has shown there are no differences attributable to depth for values less than 150 fsw while breathing either air or helium (11-14).

points varied irregularly and there were no obvious systematic relationships to pressure or time in the excursion profiles.

A similar picture is seen when the amplitude of the VER 1 is plotted as a function of time. Reduced amplitudes of response are found during the course of the exposure but these are not obviously related to the pressure profile; subject MP (Fig. 4) has reduced responses widespread throughout the greater pressures while subject FS consistently shows marked reductions only on exposure day 2.

In order to determine the factors underlying these changes in the VER 1, two different analyses were performed. In one, the latency and amplitude values were averaged by depth, in 400-foot increments, to determine the effect of pressure. In the other, the latency and amplitude values in single excursions were averaged, in order to determine the effects of single-excursion profiles or exposure duration. The measures included in this analysis started with the first module during compression and ended with the first module upon return to saturation depth. The time period covered is approximately two to three hours depending upon the excursion profile.

Table II shows the average data for all VER 1 measures by depth for subjects FS and MP.[5] Regular changes occurred: the greater the pressure, the longer the latencies of both components and the less the amplitudes of the responses. In both subjects comparison of the average values at the different depths with their respective controls indicated that all of the average latencies and the average amplitudes at 1201-1600 fsw were significantly different. Clearly pressure is one factor in the changes evidenced in the VER 1.

[5]The data for subject FS in Phases I and II have been combined for each depth since this subject maintained an identical evoked response waveform over the three-month period in which measurements on him were made. No data are reported for subject LJ in Phase I, since the waveform varied irregularly, precluding meaningful measurement of components.

TABLE II. VER 1 Measurements Compared by Pressure and by Excursion

Condition	N	Latency of Component A (msec) Mean ± SEM	Latency of Component B (msec) Mean ± SEM	Amplitude Peak to Valley (μ volts) Mean ± SEM
SUBJECT FS (Phases I + II combined)[a]				
Controls (0-60 fsw)	20	105.8 ± 0.8	166.8 ± 1.1	18.4 ± 0.4
Combined by Pressure[b]				
400-800 fsw	12	109.2 ± 0.8*	171.9 ± 1.2**	17.6 ± 0.5
801-1200 fsw	19	110.2 ± 1.0**	174.6 ± 1.0**	17.2 ± 0.8
1201-1600 fsw	9	111.8 ± 2.1*	176.8 ± 1.1**	13.8 ± 1.1**
Combined by excursion[c]				
First to 1200 fsw	5	110.8 ± 1.3	172.4 ± 2.3	16.5 ± 0.7
Second to 1200 fsw	6	109.2 ± 0.9	174.5 ± 1.9	17.6 ± 0.8
First to 1600 fsw	6	114.7 ± 2.2	176.8 ± 1.5	11.1 ± 0.7
Second to 1600 fsw	5	107.6 ± 1.2*	177.4 ± 1.0	17.2 ± 0.2**
SUBJECT MP (Phase II)				
"Control" (0-800 fsw)	5	113.8 ± 1.1	148.6 ± 1.2	8.0 ± 0.5
Combined by pressure[b]				
801-1200 fsw	8	121.0 ± 1.2**	155.0 ± 1.1**	7.7 ± 0.5
1201-1600 fsw	8	121.1 ± 0.8**	152.1 ± 0.8*	6.2 ± 0.4*
Combined by excursion[c]				
First to 1600 fsw	5	121.4 ± 0.4	152.4 ± 1.3	6.4 ± 0.7
Second to 1600 fsw	5	120.8 ± 1.2	153.6 ± 1.2	6.6 ± 0.4

[a] The data for subject FS in Phases I and II have been combined for each depth since this subject maintained an identical evoked response waveform over the three-month period in which measurements were made.

[b] For comparisons of data combined by depth, asterisks indicate mean value at pressure significantly different from mean control value. * $p < 0.05$, ** $p < 0.01$

[c] For comparisons of data combined by excursion, asterisks indicate that average value during first excursion to any pressure is significantly different from second excursion to that same pressure. * $p < 0.05$, ** $p < 0.01$

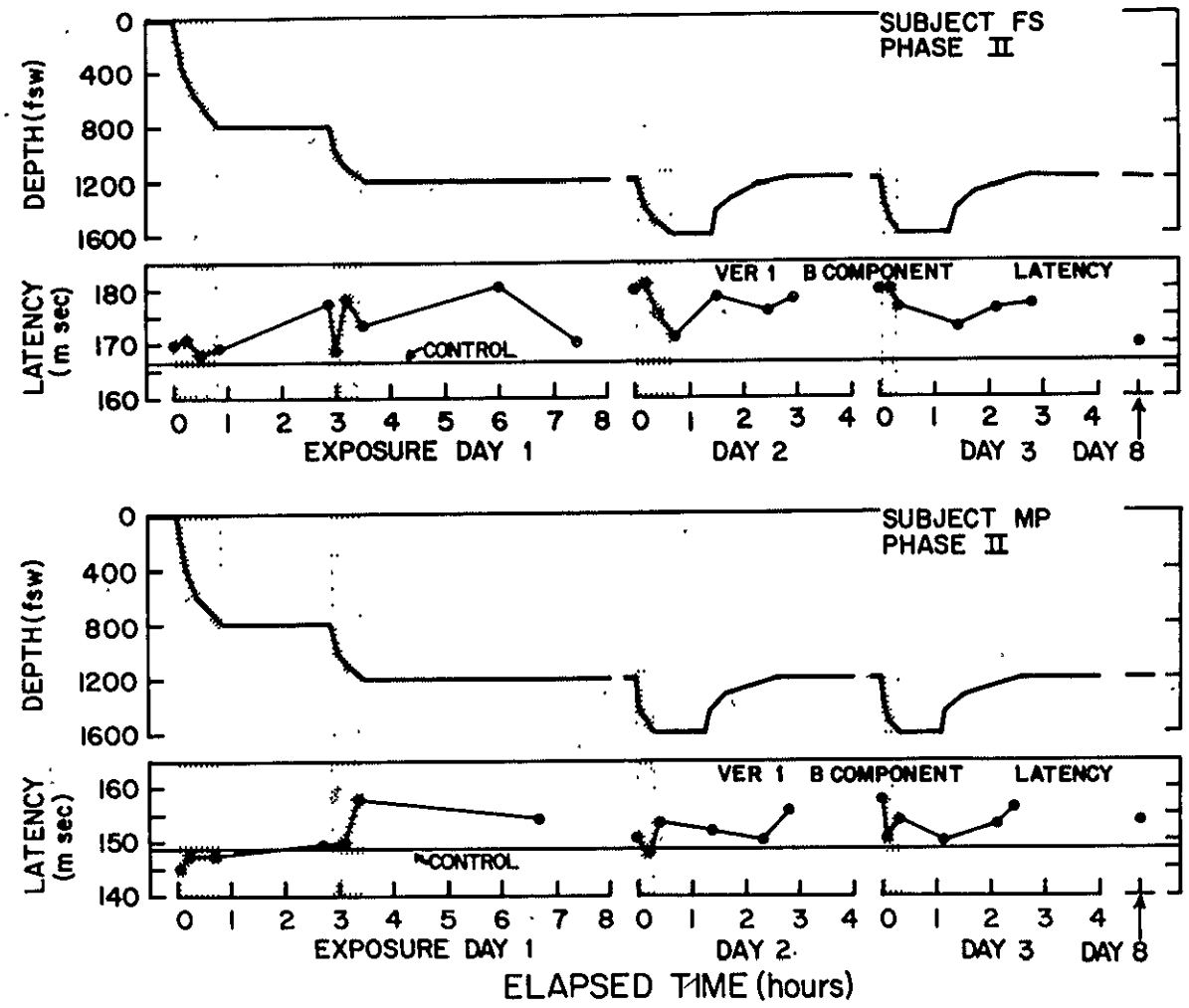

FIG. 3. The latency of the second (B) component of the VER 1 as a function of the pressure profile (subjects FS and MP).

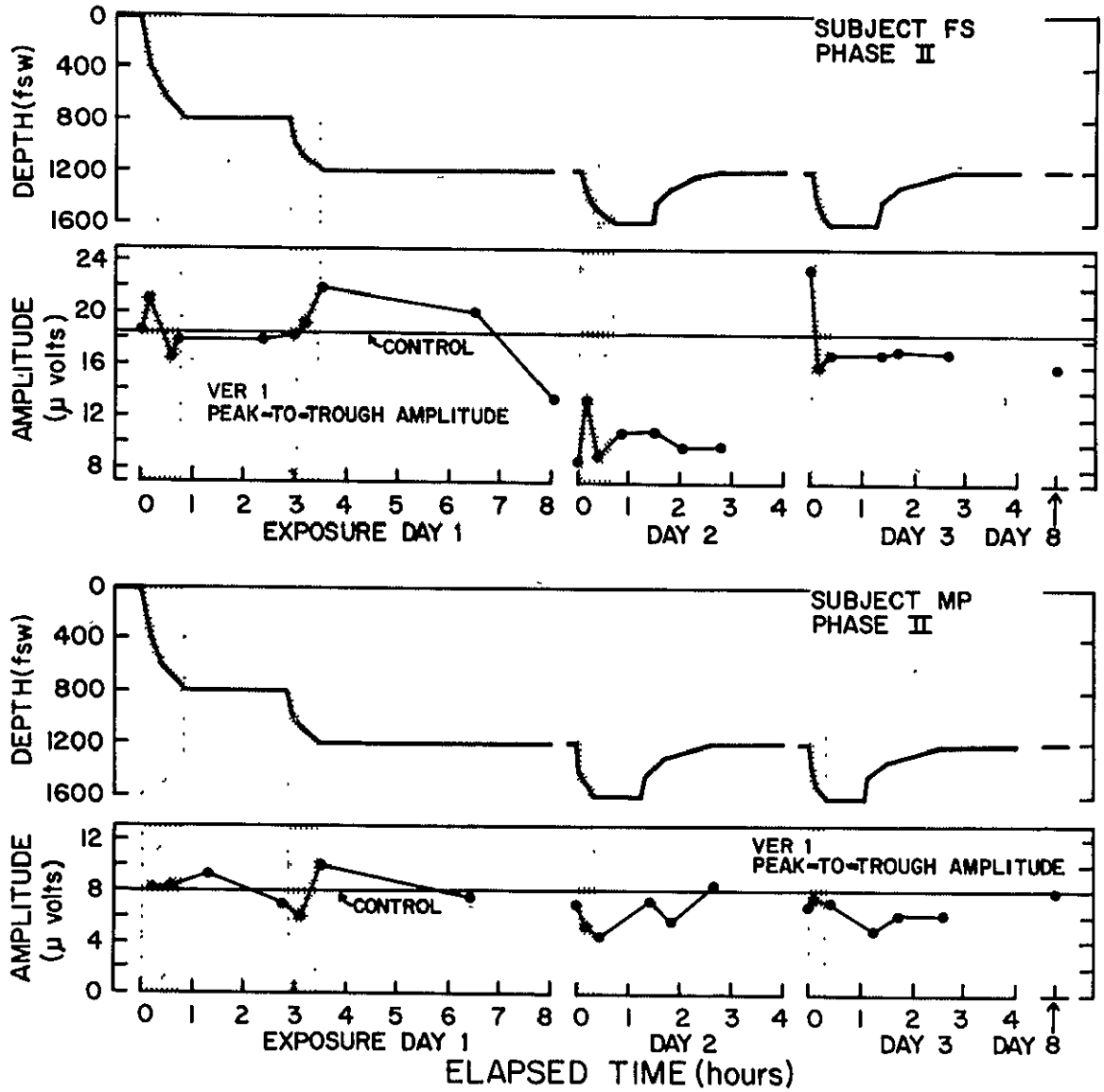

FIG. 4. The peak-to-trough amplitude of the two major components in VER 1 as a function of the pressure profile (subjects FS and MP).

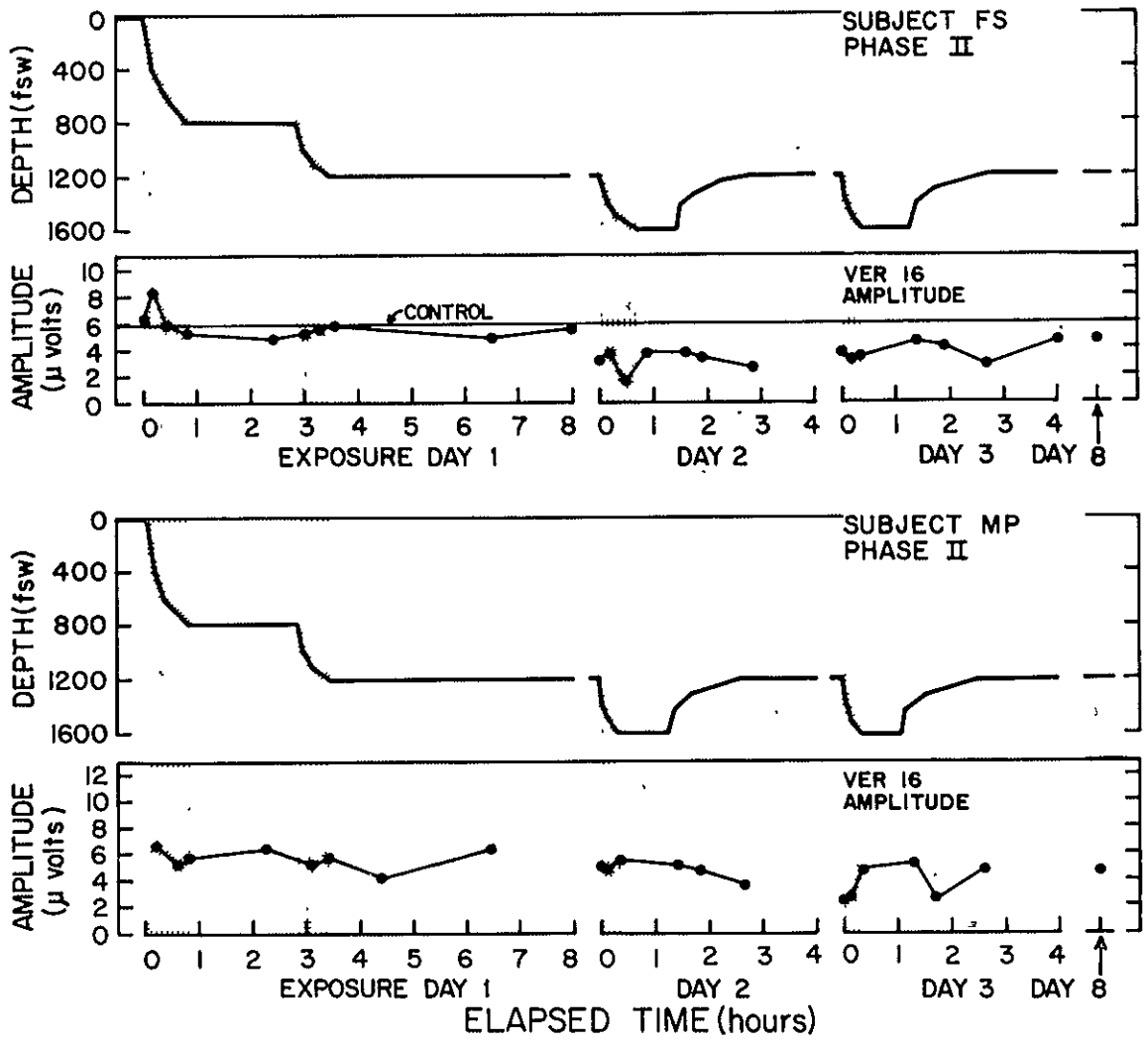

FIG. 5. The amplitude of the VER 16 as a function of the pressure profile (subjects FS and MP).

Table II also gives the average data by excursion for the VER 1. The effect of pressure is again apparent: the mean latencies during excursions were, without exception, longer than the mean control latencies, and the mean amplitudes of the responses were less than control. However, differences between the two excursions were generally absent. There were no statistically significant differences between excursions in the responses for subject MP. For subject FS the mean latency of component A and the mean amplitude measured during the second excursion to 1600 fsw were significantly different from those measured during the first excursion. The averages of the measurements during the second excursion are more like the control data than those measured during the first excursion. These differences are in the opposite direction to a possible effect of compression rate, since the second excursion had a more rapid compression; they are consistent with adaptation to the excursion conditions.

Evoked Response at the Rapid Flash Rate (VER 16)

The mean amplitudes of the 16 responses/second are plotted in Fig. 5 for each of the subjects (FS and MP) in Phase II. There was a general decrease of amplitude at the greatest pressures for both men, but no systematic relationship between the decrease and the pressure profile.

The absolute amplitudes for subject FS at 1 ata in Phase II differ considerably from his control amplitudes in Phase I (Table III). This resulted from a change in his waveform from a double-peaked response in Phase I to a higher amplitude, single-peaked response in Phase II. It is in contrast with his responses to VER 1, which remained completely consistent over the three-month testing interval, and it precludes comparison of absolute amplitudes between the two phases. A relative comparison, however, shows the decrements to be comparable below 800 fsw; 68% to 75% of control or surface values for the 800-1200 fsw depths and 62% below 1200 fsw. The mean control amplitude plotted in Fig. 5 is therefore the average only of the mean amplitudes measured immediately prior to Phase II.

These data were averaged by pressure and by excursion order in Table III in the same manner as the results for the slow flash rate. At pressures greater than 800 fsw,

TABLE III. VER 16 Mean Amplitudes Compared at Various Pressures and During Different Excursions

Condition	Phase I N	Phase I Mean Amplitude (μvolts) Mean ± SEM	Phase II N	Phase II Mean Amplitude (μvolts) Mean ± SEM
		SUBJECT FS		
Control (0-60 fsw)	13	3.67 ± 0.44	4	5.91 ± 0.28
Combined by pressure[a]				
400-800 fsw	6	2.71 ± 0.28	4	6.07 ± 0.80
801-1200 fsw	9	2.51 ± 0.20*	10	4.46 ± 0.37*
1201-1600 fsw		—	9	3.68 ± 0.23**
Combined by excursion[b]				
First	5	2.36 ± 0.31	6	3.31 ± 0.28
Second	6	2.56 ± 0.19	5	3.76 ± 0.35
		SUBJECT LJ		SUBJECT MP[c]
Control (0-60 fsw)	13	1.30 ± 0.12		—
Combined by pressure[a]				
400-800 fsw	9	1.29 ± 0.10	4	3.10 ± 0.16
801-1200 fsw	14	1.33 ± 0.13	9	2.37 ± 0.21*
1201-1600 fsw		—	8	2.27 ± 0.22*
Combined by excursion[b]				
First	5	1.02 ± 0.18	5	4.67 ± 0.33
Second	6	1.50 ± 0.13	5	3.80 ± 0.55
Third	5	1.61 ± 0.25		—

[a] For comparisons of data combined by pressure, asterisks indicate that mean value at pressure is significantly different from mean control. * $p < 0.05$, ** $p < 0.01$

[b] For comparisons of data combined by excursion, asterisks indicate that average value during first excursion to any pressure is significantly different from second excursion to that same pressure. * $p < 0.05$, ** $p < 0.01$

[c] Data for subject MP are compared to 400-800 fsw average value.

the average amplitudes of the VER 16 are significantly reduced for subjects FS and MP but not for subject LJ.

The comparison between excursions, also given in Table III, indicates that no significant differences in mean amplitudes occurred.

Frequency analysis of the raw EEG, recorded while the subject was watching the light flash 16 times/second gave the same results as the VER 16. The amplitude of the peak at 16 Hz decreased significantly for both subjects FS and MP at the higher pressures.

Spectral Analysis of VER-Related EEG Recording

Section E-2 presents the findings from the full study of electroencephalographic patterns at rest and in exercise, throughout compression and periods during residence at elevated pressure. To aid interpretation of changes in VER, frequency spectrum analysis of the O_2-C_z channel was carried out for each of the one-minute periods associated with the VER study. Findings prior to and during exposure to light flash are described.

Theta. The amplitude of theta for all conditions (Table IV) is plotted as a function of the exposure profile for subjects FS and LJ (Fig. 6) in Phase I and for subjects FS and MP (Fig. 7) in Phase II. These figures show the mean amplitude from the pre-exposure values. Since each point represents frequency analyses of only one minute of EEG, considerable variability should be expected. Nevertheless, definite increases in theta amplitude with depth occurred for all subjects and these increases were generally greater during Phase II than Phase I.

Despite this overall trend with depth, distinct individual differences existed in the pattern of the theta activity over the course of the excursions. For subject LJ (Fig. 6), the initial compression to 800 fsw and the excursion to 1200 fsw on day 1 were associated with normal theta amplitudes. Each subsequent excursion to 1200 fsw was accompanied by increasingly greater theta amplitudes. An

opposite pattern was found for subject FS in both phases (Figs. 6 and 7) and for subject MP in Phase II (Fig. 7); the first excursion to a given depth resulted in higher amplitudes of theta.

Similar individual differences were found in the pattern of the theta increases during a single excursion. Subject MP in Phase II (Fig. 7) showed very large increases immediately upon compression with a subsequent reduction over time. For the other two subjects, however, the highest values occurred one to two hours after compression. For these two subjects, the theta amplitudes prior to an excursion were higher than those of the previous day after several hours of stabilization at a given saturation depth (i.e., 800 or 1200 fsw).

In order to test the statistical significance of these trends, the data were grouped according to depth, order of excursion, and compression or decompression stages (Table IV). All theta values, for eyes open and closed, were included since there were no obvious differences between these states. Increases in theta amplitude with depth, particularly deeper than 800 fsw, are significant for all subjects. Prior to compression from one ata in Phase II, subject FS had a somewhat higher theta than in the original pre-exposure controls, but the difference was not significant.

In comparing the several excursions for subject LJ in Phase I, the average amplitudes for theta in the second and third excursions were significantly larger than in the first excursion (Fig. 6); in contrast, the average amplitudes in the second excursions for subjects FS and MP in Phase II were significantly smaller than in the first excursion (Fig. 7). There was no overall difference in theta amplitude between compression and decompression for any of the subjects. Analysis of variance performed on the data of subject MP, for whom there were equal numbers of measurements in each excursion, showed that there was not a statistically significant interaction between excursion and travel-direction, that the effects of excursion were significant, and that direction of travel was not.

Alpha. The amplitude and frequency of alpha (i.e., the peak in the range from 7 to 12 Hz) were analyzed for all subjects both while watching the flashing light and

with eyes closed without the flashing light. None of the subjects showed an appreciable amount of alpha while watching the light but all did with their eyes closed. Separate analyses, therefore, were carried out for the eyes-closed condition. The data for Phase II are plotted in Fig. 8 for subjects FS and MP; all data for both phases are analyzed by depth and by excursion in Table V. Subject MP had three distinct and sizeable analysis peaks in the alpha region at about 8, 10 and 12 Hz; the data for all three are shown in the bottom part of Table V.

The amplitude of alpha showed few significant changes during Phase I. Subject LJ had no changes in amplitude with depth, but the average amplitude during the third excursion was lower than that during the first excursion. This is the same excursion in which subject LJ showed significant increases in theta and in which he later sustained vestibular decompression sickness. For subject FS, alpha amplitude decreased significantly with depth. In the 400-800 fsw range, this was the result of two sessions with virtually no alpha (i.e., records similar to those with eyes open). At depths greater than 800 fsw, the amplitudes were generally somewhat below those of control measures.

In Phase II, subject FS again had significant reductions in alpha with depth (Fig. 8). A very large decrease occurred during the first excursion; during the second excursion, his amplitudes returned toward but did not reach the levels found at shallower depths. Subject MP, on the other hand, showed no changes in amplitude with depth for any of his three peaks. There was a significant difference between excursions in the mean amplitude of one peak that was in the direction opposite to that of subject FS.

The frequency of alpha was very stable in both phases for all subjects (Table V). There were only two significant differences among the numerous comparisons made, and these were in opposite directions.

Frequency Analysis of EEG Related to VER 1 (Subject FS)

The frequency analysis of EEG recorded with the subject watching the light flashing once a second is, for most subjects, not remarkable: there is little alpha activity,

an expected response at one Hz, and not much else except for theta peaks seen under some conditions (Fig. 2).

The frequency analysis of subject FS's data, however, consists of a regular set of harmonics from 4 to 8 Hz and sometimes even higher frequencies. A sample frequency analysis is shown in the top portion of Fig. 9. These responses are extremely consistent: the average frequency for 16 control measures is 4.02, 5.09, 6.15, 7.28 Hz, all with standard deviations less than ± 0.1 Hz. During Phase I, this regular response remained intact up to 1200 fsw; of 33 EEGs analyzed, all but one were of this pattern and the irregular one occurred at 1200 fsw. During the higher pressure exposures of Phase II, the regular pattern appeared without exception at depths less than 1200 fsw, but at 1200 fsw and beyond, there were distinct changes. If the harmonics appeared, their latency was slowed; frequently they did not appear at all. Examples are shown in the lower portion of Fig. 9. Of seven measurements made at 1200 fsw, three were regular and four were not; of nine measurements made at greater depths, only two displayed the regular harmonic pattern.

DISCUSSION

A number of statistically significant changes occurred in the visual evoked responses, as well as in associated electroencephalographic patterns, during both phases of this study. While these must be considered related to rapid compression and increased hydrostatic pressure, the changes were small but consistent alterations of normal responses. Actually, many might have gone undetected except for the opportunity generated by the extensive data accumulated, particularly in the pre-exposure control periods. Concurrent measurements of electroencephalographic changes (Section E-2), visual function (Section E-8) and eye-hand coordination (Section E-10) are available for comparison with these VER changes.

CHANGES IN THE VISUAL EVOKED CORTICAL RESPONSE

The VER changes observed included loss of amplitude of both the slow and rapid stimulus types of VER and an

TABLE IV. Amplitude of Theta Compared at Various Pressures, During Different Excursions and as a Function of Compression-Decompression (Theta Measures in VER 1, VER 16 and with Eyes Closed Combined)

Condition	Phase I N	Phase I Amplitude Theta (μ volts) Mean ± SEM	Phase II N	Phase II Amplitude Theta (μ volts) Mean ± SEM
		SUBJECT FS[a]		
Control (0-60 fsw)	23	3.03 ± 0.11	8	3.40 ± 0.15
Combined by pressure[b]				
400-800 fsw	16	3.55 ± 0.14**	8	3.57 ± 0.13
801-1200 fsw	18	3.98 ± 0.13**	20	4.10 ± 0.15**
1201-1600 fsw		—	18	5.05 ± 0.29**
Combined by excursion[c]				
First	10	3.83 ± 0.32	12	5.28 ± 0.42
Second	12	3.95 ± 0.14	10	4.28 ± 0.19*
Combined by direction of travel[d]				
Compression	8	3.72 ± 0.16	6	4.99 ± 0.21
Decompression	8	4.29 ± 0.34	8	4.86 ± 0.64
		SUBJECT LJ		SUBJECT MP
Control (0-60)	33	2.14 ± 0.08	7	3.39 ± 0.23
Combined by pressure[b]				
400-800 fsw	27	2.36 ± 0.08	12	4.34 ± 0.42
801-1200 fsw	41	2.89 ± 0.16**	24	4.46 ± 0.19**
1201-1600 fsw		—	24	5.00 ± 0.28**
Combined by excursion[c]				
First	15	2.09 ± 0.09	15	5.63 ± 0.35
Second	17	3.05 ± 0.17**	15	4.19 ± 0.13**
Third	15	3.33 ± 0.33**		—
Combined by direction of travel[d]				
Compression	18	2.57 ± 0.11	12	5.16 ± 0.49
Decompression	18	2.80 ± 0.25	12	4.85 ± 0.28

[a] No VER 1 data for subject FS included. See section on frequency analysis of EEG for VER 1.

[b] For comparisons of data combined by pressures, asterisks indicate that mean value at pressure significantly different from control. * $p < 0.05$, ** $p < 0.01$.

[c] For comparisons of data combined by excursion, asterisks indicate average value during first excursion to any pressure significantly different from second or third excursion to that same pressure. * $p < 0.05$, ** $p < 0.01$.

[d] For compressions of data combined by direction of travel, asterisks indicate that average of values measured during compression are significantly different from those measured during decompression. * $p < 0.05$, ** $p < 0.01$.

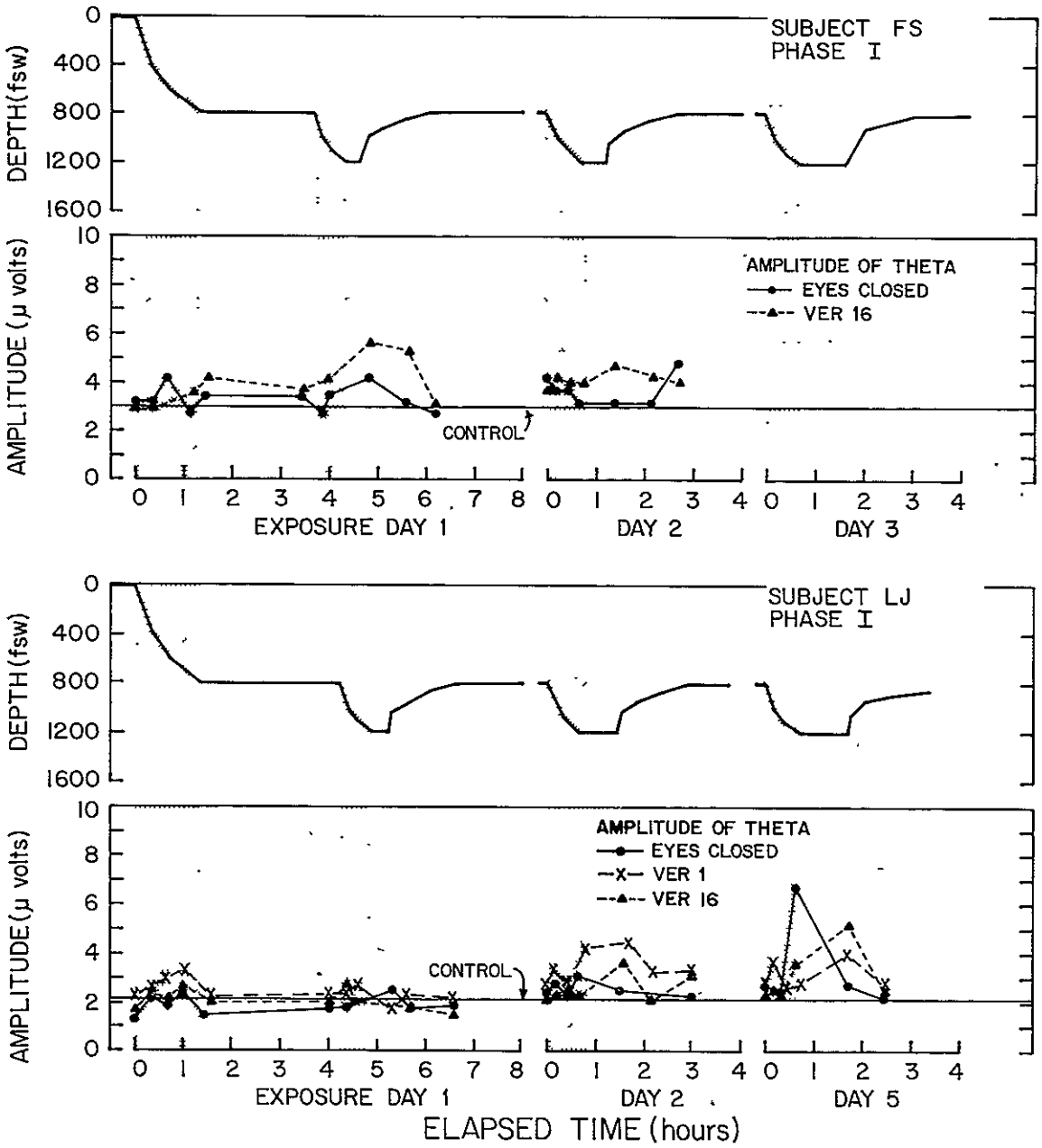

FIG. 6. The amplitude of theta recorded under various conditions as a function of the pressure profile (subjects FS and LJ).

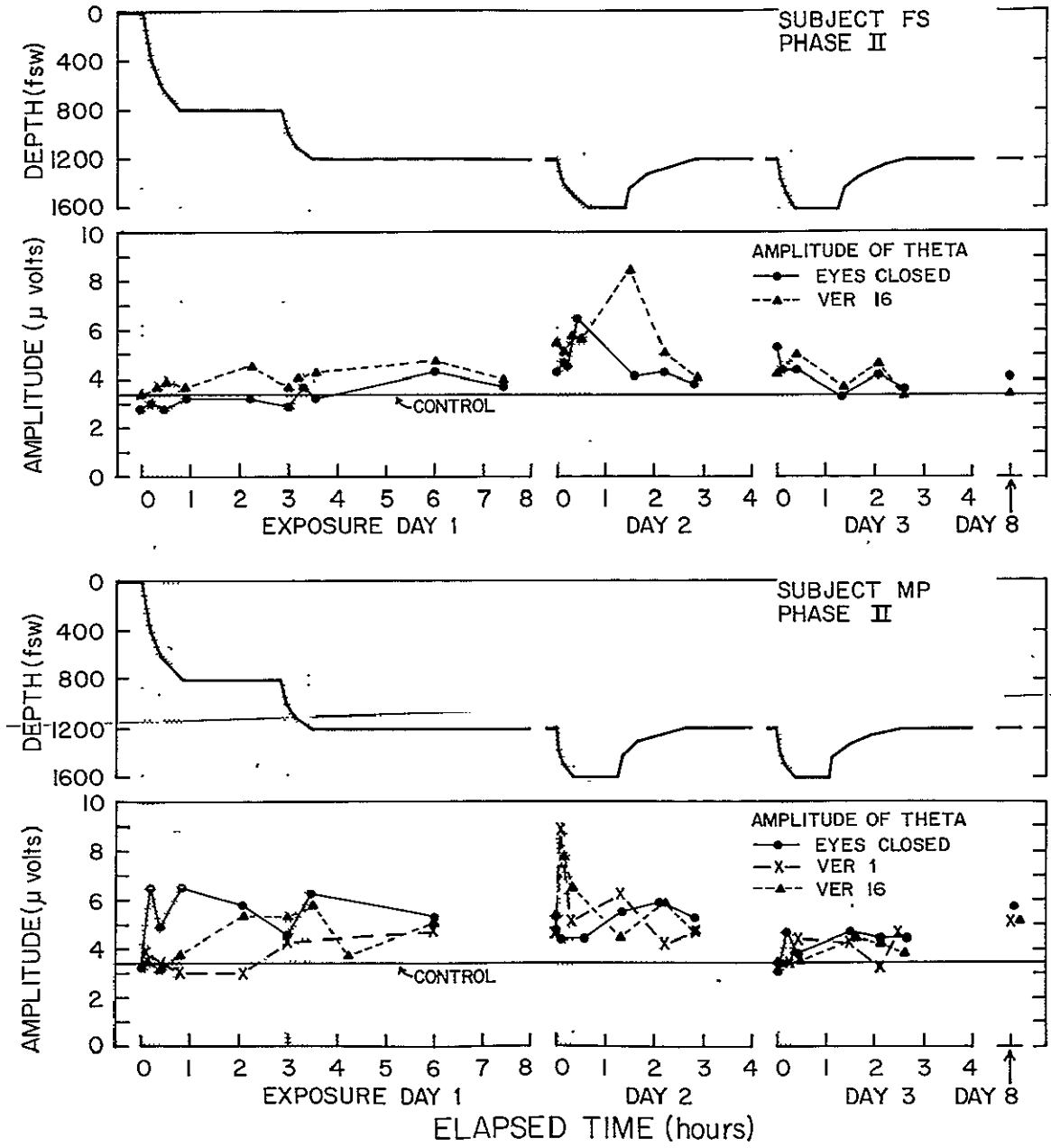

FIG. 7. The amplitude of theta recorded under various conditions as a function of the pressure profile (subjects FS and MP).

TABLE V. Measures of Peaks in the Alpha Region Compared at Various Depths and During Different Excursions (Eyes Closed)

Condition	Phase I N	Phase I Amplitude (μ volts) Mean ± SEM	Phase I Frequency (Hz) Mean ± SEM	Phase II N	Phase II Amplitude (μ volts) Mean ± SEM	Phase II Frequency (Hz) Mean ± SEM
SUBJECT FS						
Control[a]	8	23.01 ± 1.53	9.60 ± 0.10	7	15.02 ± 0.80	9.97 ± 0.22
Combined by pressure[b]						
400-800 fsw	9	15.27 ± 2.52*	10.08 ± 0 16			
801-1200 fsw	9	17.46 ± 0.95**	10.46 ± 0.23	10	10.56 ± 1.17*	10.30 ± 0.21
1201-1600 fsw		—	—	9	8.95 ± 0.94**	9.86 ± 0.08
Combined by excursion[c]						
First	5	16.37 ± 1.38	10.82 ± 0.31	6	6.71 ± 0.31	9.90 ± 0.09
Second	6	18.26 ± 0.75	9.90 ± 0.16*	5	11.13 ± 0.76**	9.66 ± 0.12
SUBJECT LJ				**SUBJECT MP**		
Control	7	9.53 ± 0.53	9.41 ± 0.15			
Combined by pressure[b]						
400-800 fsw	9	11.59 ± 0.72	9.70 ± 0.07		See below for data	
801-1200 fsw	13	10.41 ± 0.65	9.62 ± 0.07			
1201-1600 fsw		—				
Combined by excursion[c]						
First	5	11.31 ± 0 85	9.66 ± 0.10			
Second	5	11.92 ± 0.85	9.60 ± 0.08			
Third	5	8.05 ± 0.34	9.54 ± 0.13**			

	SUBJECT MP--Phase II[d]					
	N	Amplitude (μ volts) Mean ± SEM	Frequency (Hz) Mean ± SEM	Amplitude (μ volts) Mean ± SEM	Frequency (Hz) Mean ± SEM	Amplitude (μ volts) Mean ± SEM	Frequency (Hz) Mean ± SEM
Control (0-800 fsw)	6	6.05 ± 0.99	7.78 ± 0.09	8.36 ± 0.44	9.92 ± 0.09	5.06 ± 0.48	11.74 ± 0.09
Combined by pressure[b]							
801-1200 fsw	8	6.26 ± 0.78	7.89 ± 0.12	7.28 ± 0.69	9.81 ± 0 14	3.81 ± 0.84	11.99 ± 0.15
1201-1600 fsw	8	8.05 ± 1.00	7.86 ± 0.13	7.55 ± 0.52	10.36 ± 0.10**	5.52 ± 0.52	11.58 ± 0.15
Combined by excursion[c]							
First	5	9.28 ± 1.14	7.78 ± 0.08	7.70 ± 0.21	10.26 ± 0.20	5.02 ± 0.82	11.72 ± 0.17
Second	5	5.98 ± 0.77	8.10 ± 0.21*	7.26 ± 0.84	10.22 ± 0.14	4.88 ± 0.88	11.68 ± 0.26

[a] Control for subject FS for Phase I is average of measurements made at 0-60 fsw, for Phase II, 0-800 fsw.

[b] For comparisons of data combined by pressure, asterisks indicate mean value at pressure, significantly different from control. * $p < 0.05$, ** $p < 0.01$

[c] For comparisons of data combined by excursion, asterisks indicate average value during first excursion to any pressure, significantly different from second or third excursion to that same pressure. * $p < 0.05$, ** $p < 0.01$

[d] See text for explanation.

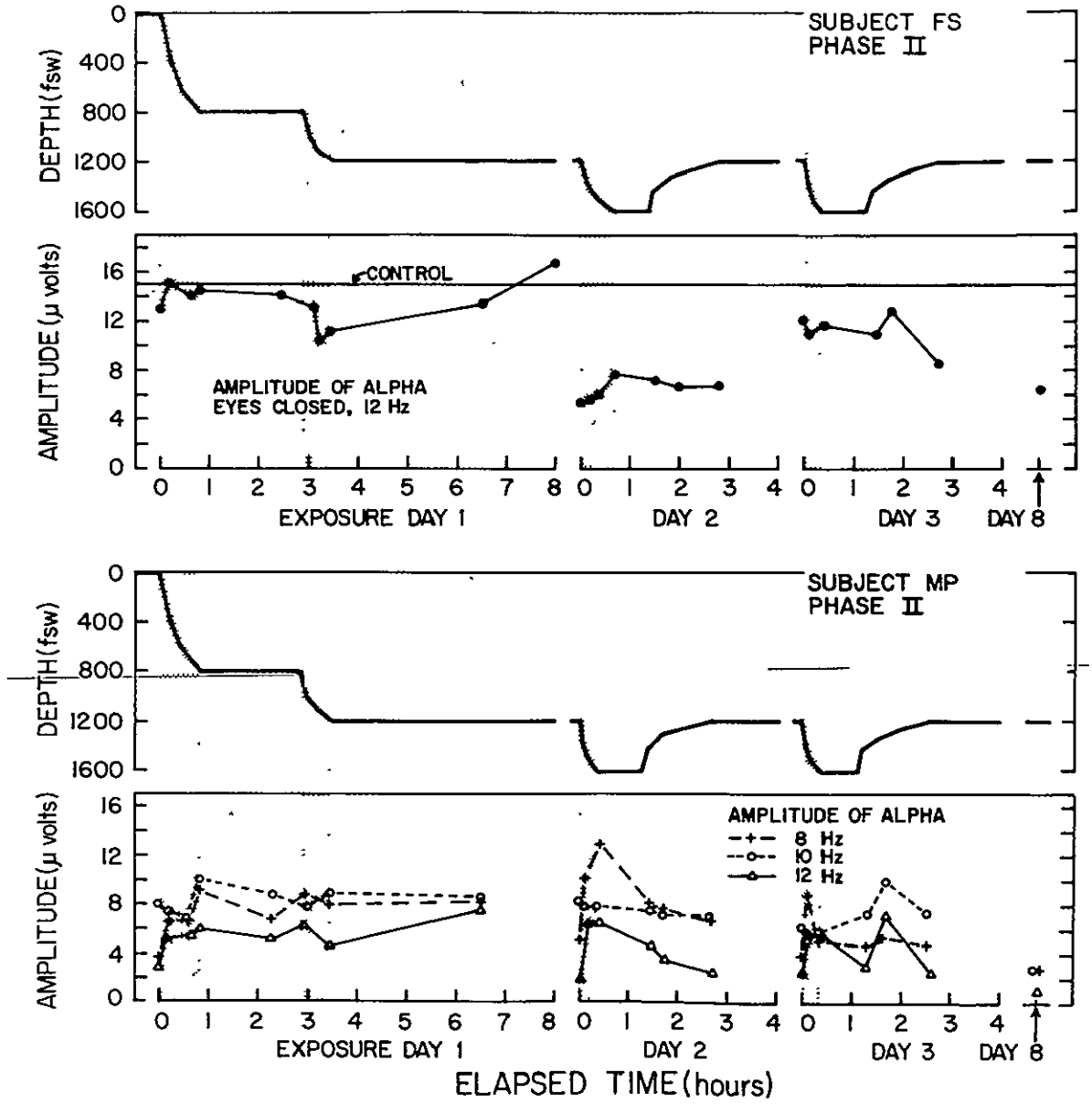

FIG. 8. The amplitude of alpha recorded with eyes closed as a function of the pressure profile (subjects FS and MP).

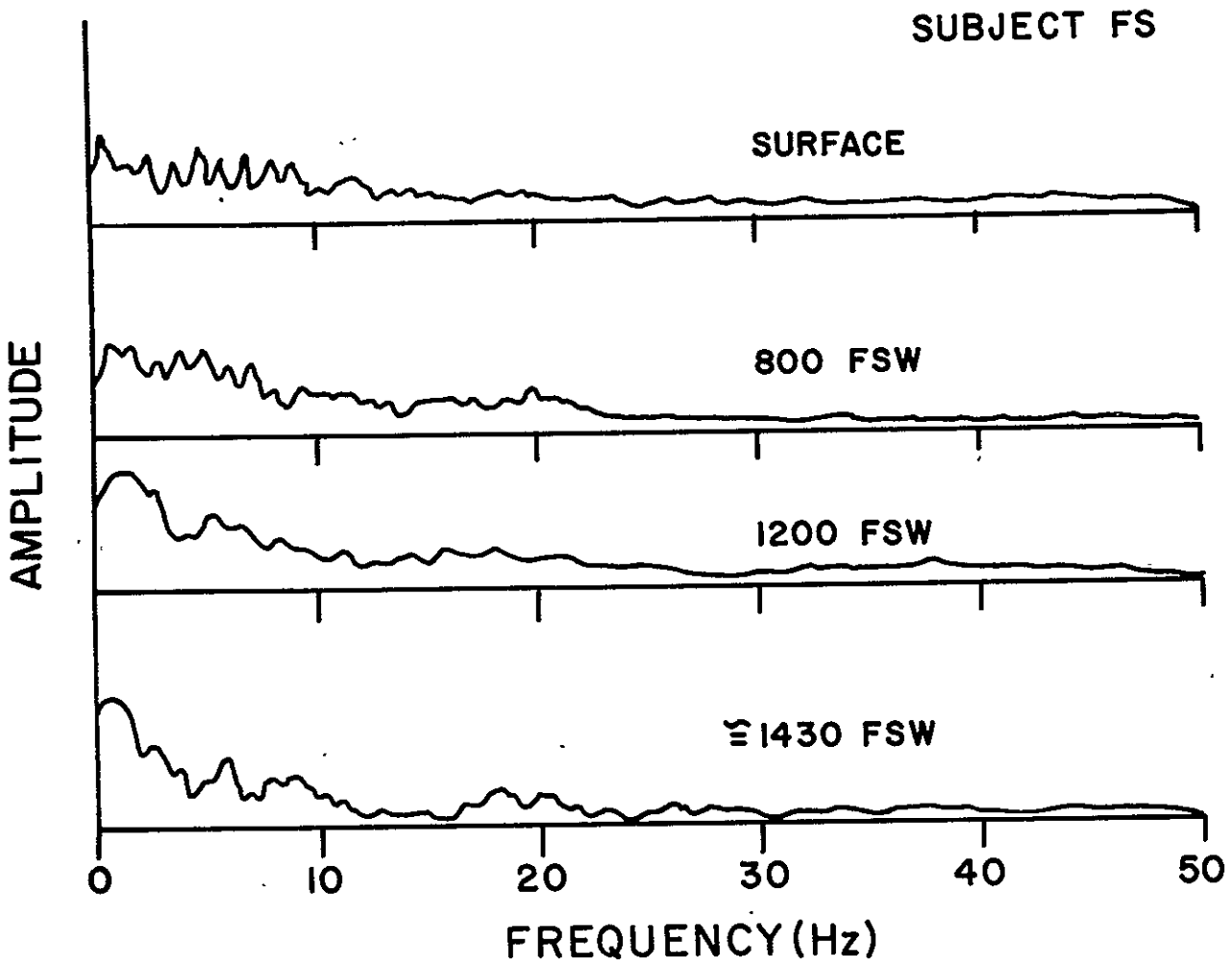

FIG. 9. Frequency analysis of the EEG at various pressures recorded while watching the patterned light flashing once a second (subject FS).

increased latency of the components in the slow type (VER 1). Losses of amplitude of evoked responses have been reported in a number of previous exposures to high ambient pressure (1-3,14,20) and have been considered to reflect a general depression of EEG activity by increased hydrostatic pressure itself. In the present study there was no clear indication that an effect of compression rate is responsible for reduced VER amplitude. When changes did occur they tended to persist. Partial reversal of changes toward pre-exposure values occurred during sustained exposure to high pressure, but none actually reached control mean values, even on exposure day 8. While most signs and symptoms of compressior gradually abated after a few hours at a constant pressure, some effects appeared to remain (Section E-1), even after days of saturation (4,8). The reduced amplitude of the VER was apparently one such sign; similar long-term effects have already been noted for the auditory evoked response (3).

The increased latency of the components of the VER 1 was another persistent effect. Apparently it was observed previously in two very different pressure studies, i.e. NISAT-I, a shallow saturation dive on nitrogen-oxygen (18), and Physalie VI, a helium-oxygen exposure (20). NISAT-I was a particularly stressful exposure and its subjects evidenced a number of behavioral and physiological signs, including increased theta and decreased alpha activity.

The etiologies of increased latencies may differ with the types and degrees of compression. The results of this study are comparable to those reported for Physalie VI (20) despite many differences in conditions and methods. For Predictive Studies IV the visual stimulus was a striped pattern in contrast to the flash of light through closed eyelids used in Physalie VI. The resulting evoked responses have different components. The compression rate was relatively rapid in Predictive Studies IV and slow in Physalie VI (20). Pressures and breathing gas mixtures, however, were comparable and it may be that the increased latency of the evoked response is a fundamental consequence of high pressure exposures.

In support of this concept, pressurization (with helium) increases synaptic delay in the squid giant synapse (7) at levels of pressure only slightly greater than half that at which man has been exposed. At 35 ata, equivalent to about

1100 fsw, synaptic delays were 50% longer than at 1 ata; they were increased fourfold at 200 ata. In addition, prolongation of the action potential, usually resulting in decreased conduction velocity, has been found during exposures of many isolated neural preparations to increased helium pressures (5-7,9,10,21,22). Thus, there is considerable evidence supporting a direct effect of increased hydrostatic pressure on neural activity. The absence of measurable effect of helium itself on discrete mental functions in man, even to pressures of 1200 fsw (17), has been offered as the basis for considering the neural changes to be evoked by pressure rather than by helium (16,17).

SUMMARY

Visual evoked cortical responses and related electroencephalographic activity were recorded from subjects during rapid compression and exposure to pressures equivalent to 400, 800, 1200 and 1600 fsw. Small but consistent alterations of normal responses to visual stimulation were found. Decreases in amplitude and increases in latency of the VER were found at the higher pressures, with incomplete return to pre-exposure values even after several days of continued exposure to increased pressure. Changes were not associated with compression rate or decompression. Electroencephalographic theta activity increased progressively with increasing pressure. While all subjects showed overall increases in theta activity, the pattern of increase varied with the individual.

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E-4. TREMOR AND SOMATOSENSORY EVOKED CORTICAL RESPONSE

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Development of visible tremor has been one of the effects cited as a prominent consequence of compression, collectively described as the "High Pressure Nervous Syndrome" (HPNS) (4). In efforts to identify its characteristics, quantitative measurement of finger microtremor has been performed in several simulations of deep diving using helium-oxygen breathing mixtures (1-4) and mixtures of oxygen with helium, neon and nitrogen (7). Typically, tremor amplitude is increased during periods of rapid compression with partial or complete return to normal during periods at stable pressure. However, methods of measurement, exposure conditions and even type of tremor measured have varied considerably in different laboratories. Individual variability in response to compression also appears to be great among the few subjects it has been possible to study under reasonably controlled conditions.

The present investigation was part of an integrated program which included measurements of neurological and related effects of compression and pressure. Compression rates were intentionally selected which would be rapid enough to produce prominent symptomatic and objective effects in the subjects. It was planned to record and analyze the forms, onset, severity and time course of change in associated tremor activity, providing information not only on effects induced, but on rates of adaptation.

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As quantitative measures of the tremor form of neurological response to compression and pressure, both intentional tremor and postural tremor were monitored in all subjects, in Phases I and II of Predictive Studies IV. Intentional tremor here refers to spontaneous finger movements occurring while a constant force is exerted by the finger; postural tremor indicates finger movements recorded while the arm is held outstretched against the force of gravity (1). It was considered important to monitor both types of tremor, since sensory and motor pathways are probably not identical for the two types. A comparison of responses to the same stresses may suggest possible bases for observed effects.

In the higher compression rate and pressures of Phase II, conduction times along both peripheral and central pathways were also measured. The latency of median nerve action potentials from wrist to elbow was measured as an index of peripheral conduction time. Latencies of primary and secondary components of somatosensory evoked cortical responses (SSER) to the median nerve stimulus were determined as a measure of combined peripheral and central conduction times. The concurrent study of latencies for components of the visual evoked cortical response (Section E-3) (5) was intended to permit comparison of compression and pressure effects on conduction times along two different central neural pathways.

METHODS

To study the rate and degree of adaptation to increased pressures, repeated measurements of tremor were made during compressions, during excursion-decompressions, and for the first few hours at stable increased pressures, in "rest" and "exercise" subjects. Possible residual influences of neuromuscular, circulatory and metabolic responses to exercise were evaluated in the exercise subjects by recording tremor prior to and immediately following six minutes of standard light work (see Section E-14). Concurrent measurements of skin, deep body and ambient temperatures were made in all subjects (Section E-15), permitting evaluation of possible effects of hyperthermia, which may increase tremor intensity (10).

Somatosensory evoked cortical responses were measured in all subjects at stable pressures before and after but not during periods of rapid compression. For the subjects who carried out physical exercise, these measurements were made during periods of rest, between work intervals. The measurement of peripheral nerve conduction velocity was not technically successful and will not be discussed.

TREMOR MEASUREMENTS

Intentional tremor was measured with a Statham strain-gauge transducer mechanically coupled to an aluminum bar-plastic finger cup assembly (8). Each rest and exercise subject had a separate tremor device mounted on a platform within his reach. The electrical output of the tremor transducer was recorded on magnetic tape for two-minute periods while the subject applied a downward force of 500 g exerted by the middle finger on the finger cup assembly. An electrical panel meter visually indicated to the subject deviation from this intended force. The forearm rested on the platform to stabilize the arm itself.

Accelerometers (Grass model SPA) attached to the middle finger by plastic clips measured postural tremor for two minutes immediately following measurement of intentional tremor. The arm was supported at the elbow and held parallel to the floor with the hand pronated, opened and the fingers extended. Accelerometer output was also recorded on magnetic tape.

An index of the magnitude of tremor activity was obtained by electrically integrating (Grass Instruments Co., Polygraph Integrator model 7P10B, 0-50 Hz) the amplitude of tremor signals which were filtered (Krohn-Hite model 3321) prior to integration to remove frequency components from 0 to 2.5 Hz and at 60 Hz or higher. To avoid saturation, the integrator was reset to zero every 10 seconds. This "tremor index" was expressed as the average of all 10-second intervals obtained during each measurement period.

Frequency analyses for both intentional and postural tremor were obtained from a spectrum analyzer (Federal Scientific model UA-10) and spectrum averager (Federal Scientific model 10-16) over a range of 0-50 Hz. Data

samples of four seconds' duration were analyzed and plotted on an x-y plotter (MFE Plotamatic model 715) and inspected for dominant frequency. Frequency spectra are described elsewhere (6).

In addition to integration of the tremor signals covering the 2.5-50 Hz band, integrated amplitudes of frequencies covering bandwidths of 3-7 Hz and 8-12 Hz were compared in the four subjects (CC, GM, MP, FS) of Phase II. This type of analysis was done because frequency of tremor is often used to describe clinically abnormal tremors such as those associated with Parkinson's disease. Tremor in each respective bandwidth probably involves different brain sites or pathways, and this analysis might help describe the involvement of the CNS in mediating tremor associated with compression and pressure.

ELECTROPHYSIOLOGICAL MEASUREMENTS

The somatosensory evoked responses were recorded after the median nerve was stimulated by a mild shock for 250 microseconds every 3 seconds, delivered via two electrodes (Grass Instruments Co., gold-cup) placed 2 cm apart at the wrist. The stimulus was a constant current, the levels of which were adjusted in intensity from 9 to 29 milliamperes to elicit a visible thumb twitch. It was delivered by the combination of a stimulator (Grass Instruments Co. model S-9) with a constant-current unit (Grass Instruments Co. model CCU1). A ground plate was attached to the arm between the stimulating and recording electrodes to reduce shock artifact. Subdermal pin electrodes (Phase II) or silver:silver chloride (Beckman Biopotential) electrodes (Phase I) for the SSER were placed over postrolandic areas 7 cm below and 4 cm lateral to the vertex (cz). Electrode impedance was less than 2000 ohms. All evoked potentials were amplified by a preamplifier in the electroencephalograph (Section E-2), recorded on magnetic tape, and analyzed by a computer of average transients with a write-out time of 500 msec.

STATISTICAL ANALYSIS

Analysis of variance for repeated measures (9) was performed on the more complete results obtained in Phase II for intentional and postural tremor data (2.5-50 Hz). The overall analyses were followed by tests of the following specific comparisons: 1) all means were compared with the pre-exposure 1 ata control; 2) both exposure day 2 and day 3 excursion measurements were tested against their respective pre-excursion 1200-foot values to determine whether compressions from 1200 to 1600 feet produced changes; 3) the stable state at 1200 fsw (average of day 2 pre-excursion, day 3 pre-excursion and day 8 measurements at 1200 fsw) was compared with the sea level condition (average of pre-exposure and post-exposure 1 ata values) to determine whether exposure to 1200 feet affected tremor. Comparisons were considered statistically significant if $p \leq 0.05$. The data for the 3-7 Hz and the 8-12 Hz frequency bands were also tested to determine if there were differences between the two bands in their patterns of change as related to the pressure profile.

RESULTS

TREMOR MEASUREMENTS AT REST

Individual and mean values of integrated amplitudes and dominant frequencies for intentional and postural tremor are summarized for Phase I in Table I and for Phase II in Table II. The Phase I records were not always technically satisfactory for analysis of dominant frequency. Individual data for the resting state include all measurements from the rest subjects and pre-exercise values from the exercise subjects. Technical difficulties caused omissions in the tables. The pre-exposure control values for each subject in Phase II are averages of three or four measurements made over several days preceding the start of the exposure (Table II). Results of the integration of tremor amplitudes of Phase II after the tremor signals were separated into 3-7 Hz and 8-12 Hz frequency bands are given in Table III.

Mean integrated amplitudes of intentional and postural tremor measured during Phase I are compared with the

composite pressure-exposure profile for the four subjects in Fig. 1. For the first two exposure days only data from those modules in which measurements were obtained on all four subjects are plotted, except for the module at 1 ata on exposure day 1, when there were three subjects. All data, however, are given in Table I. Since measurements were made in only two subjects (LJ and CC) during the 800-1200 fsw excursion on exposure day 5, their averages for the other exposure days are also shown in Fig. 1 as open circles. The vertical bars on the depth profile show when comments reported by the subjects, or visible signs seen by observers were recorded. These included visible tremor, shakiness, jumpiness, edginess, muscle fasciculations or jerks, and "excess energy" in the legs (see Section E-1).

Intentional tremor index values in Phase I increased during actual compressions on exposure days 1 and 2, when compared with pre-compression values. Following each compression, there was a prompt return toward the pre-compression level. Compression on exposure day 5 did not elicit any increase in tremor in two subjects. The tremor values on day 2 were similar to those on day 1 following compression to 800 and 1200 fsw.

Postural tremor in Phase I increased during two of the four compressions (excursions to 1200 fsw on exposure days 1 and 5). The tremor values on days 2 and 5 were somewhat elevated when compared with day 1.

Average data for both intentional and postural tremor from Phase II are shown in Fig. 2 (integrated amplitude) and Fig. 3 (dominant frequency). Tremor measurements during the initial compression from the surface on exposure day 1, were made successfully only in two (MP, CC) of the four subjects (Table II). Vertical lines on the depth profile of Fig. 2 indicate signs and symptoms as described for Fig. 1.

Both intentional and postural tremor values increased during compressions in Phase II. Following each compression, tremor amplitudes promptly declined to pre-compression values. In other respects, the two types of tremor behaved differently.

Intentional tremor on exposure days 2 and 3 before and after the excursions was elevated compared with day 1.

Intentional tremor at stable pressure on day 8 was at the same level as on days 2 and 3. The post-exposure value was at the pre-exposure level shown in Fig. 2. The combined pre-excursion values for amplitude obtained at 1200 fsw on exposure days 2 and 3 and the single value on exposure day 8 are significantly different from the combined pre- and post-exposure sea level measurements. The amplitude of intentional tremor was significantly increased at 1600 fsw on exposure days 2 and 3 compared with the pre-excursion value.

In contrast, postural tremor amplitudes on exposure days 2 and 3 began and ended at the 1 ata control level. Postural tremor at stable pressure on day 8 was also at this control level. Amplitude of postural tremor increased significantly only during compression from 1200 to 1600 fsw on exposure day 3.

Comparison of the 3-7 Hz and 8-12 Hz frequency bands (Table III) by partitioning the data into a factorial design of compression profile and tremor frequency showed that for intentional tremor the significant variable was compression profile ($F = 2.60$, $df = 17,51$, $p \leq 0.01$). A similar result was found for postural tremor ($F = 2.30$, $df = 17,51$, $p \leq 0.05$). In the average amplitudes of both the intentional tremor and the postural tremor there was no significant interaction between the factors or a significant difference between the overall amplitude levels for the two bandwidth ranges. The pattern of change of the average amplitude of both the 3-7 Hz and 8-12 Hz bandwidths were similar to that of the 2.5-60 Hz amplitude (Fig. 2) for both types of tremor.

Average dominant frequencies of both intentional and postural tremor in Phase II are shown with the pressure profile in Fig. 3. None of the frequency changes is statistically significant when compared with pre-exposure control values. The only change which does show a significant difference is the dominant frequency of postural tremor measured during the excursion-compression on exposure day 2 compared with the pre-excursion value at 1200 fsw, which is lower than the sea level control. Some, but not all, of the increases in tremor amplitude (Fig. 2) are associated with parallel increments in dominant frequency (Fig. 3).

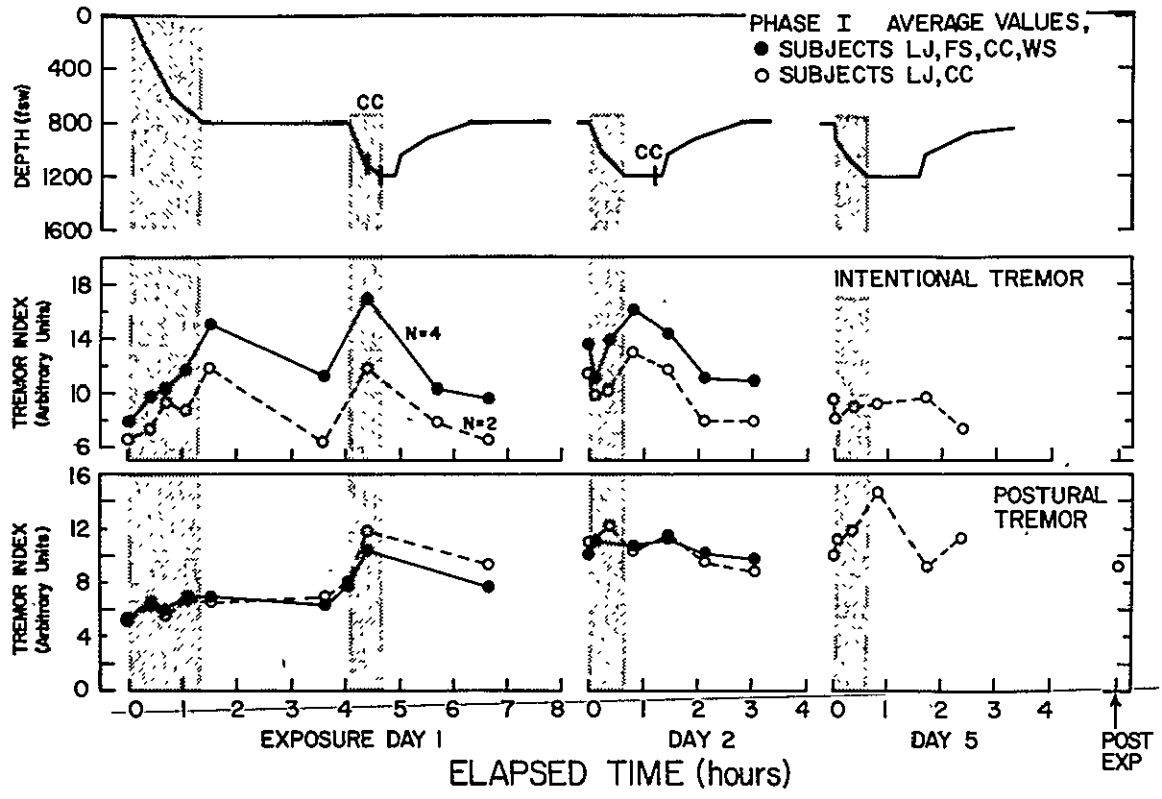

FIG. 1. Integrated amplitudes (tremor index) of intentional and postural tremor during compression and exposure to simulated depths of 800 and 1200 fsw in Phase I. Closed circles and solid lines indicate average data of subjects CC, LJ, FS and WS (except for sea level module on exposure day 1 where N = 3). Open circles and dashed lines show average data of subjects CC and LJ. See text for explanation of vertical bars on depth profile.

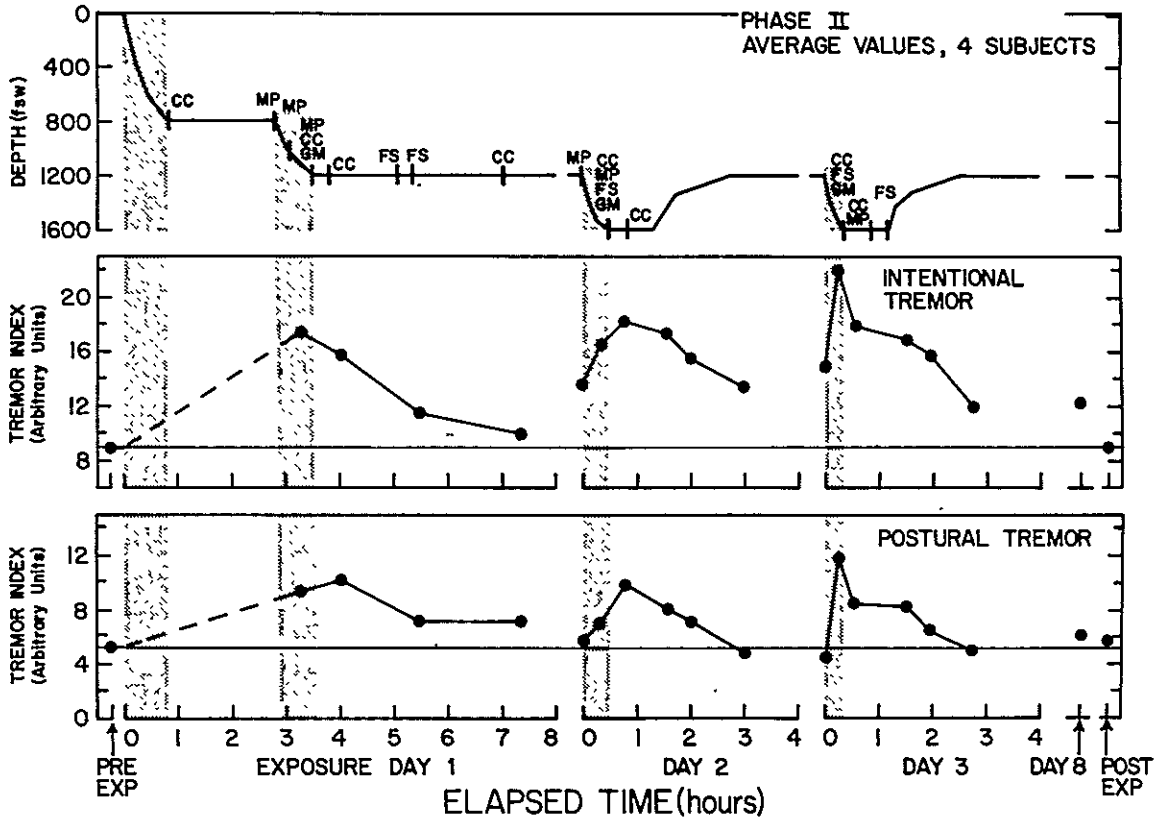

FIG. 2. Mean integrated amplitudes of intentional and postural tremor during compression and exposure to simulated depths of 800, 1200 and 1600 fsw in Phase II. All points represent average data of subjects CC, GM, MP and FS. Measurements during the initial compression on exposure day 1 were obtained only in subjects CC and MP and are shown in Table II. Horizontal lines drawn through the pre-exposure data are for reference. See text for explanation of vertical bars on the depth profile.

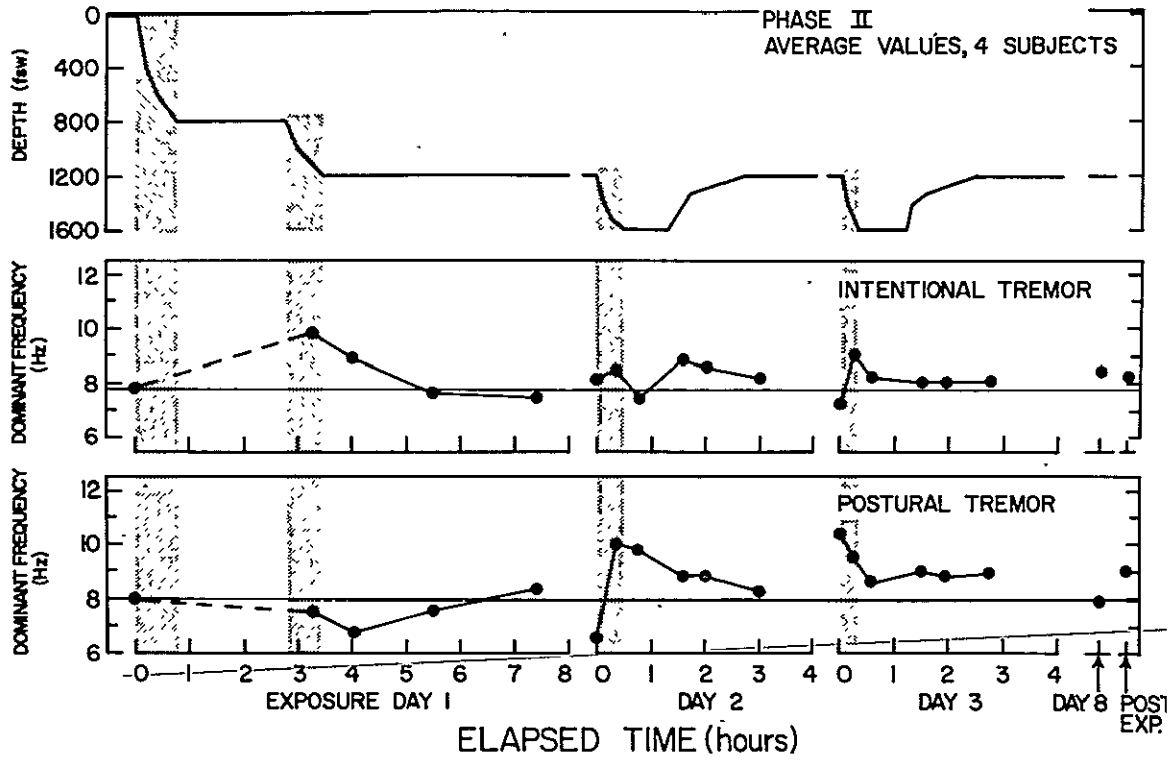

FIG. 3. Mean dominant frequencies of intentional and postural tremor during compression and exposure to simulated depths of 800, 1200 and 1600 fsw in Phase II. All points are average data of subjects CC, GM, MP and FS. See legend of Fig. 2 for additional details.

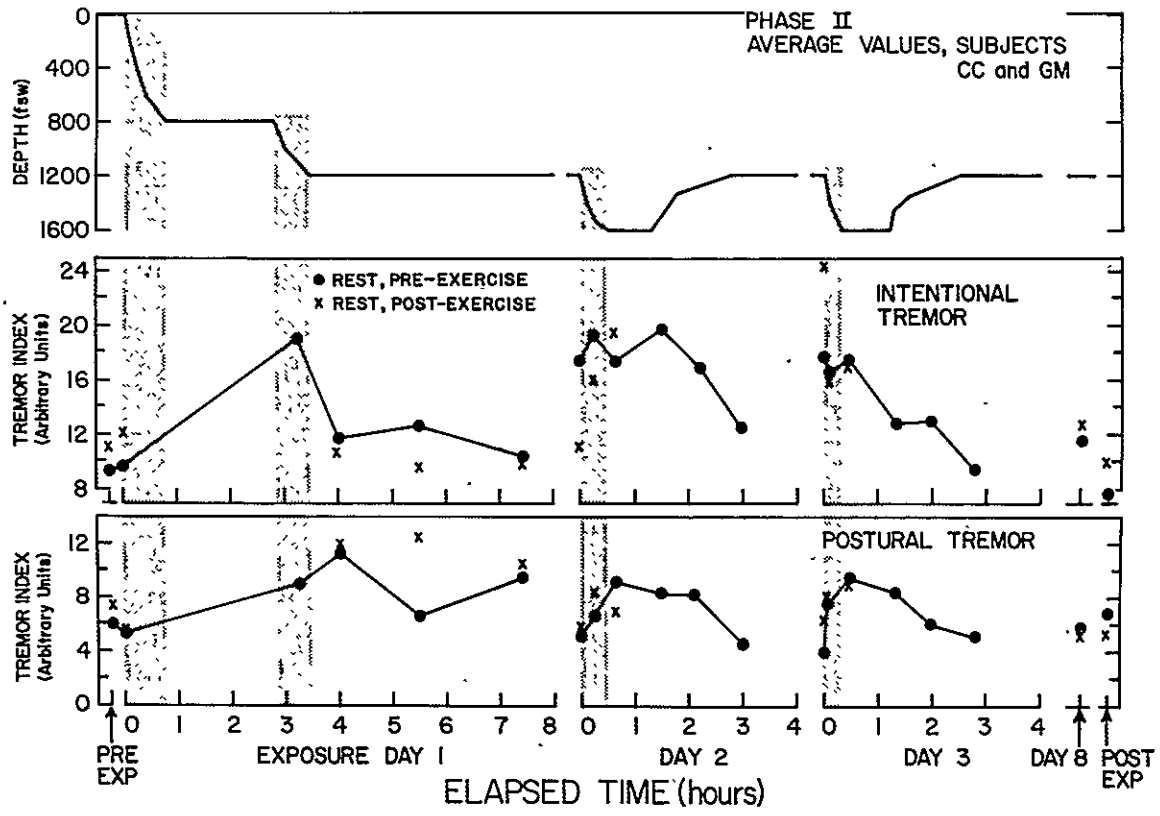

FIG. 4. Effects of light exercise on integrated amplitudes of intentional and postural tremor during compression and exposure to simulated depths of 800, 1200 and 1600 fsw in Phase II. All points are average data of subjects CC and GM.

TABLE I. Intentional and Postural Tremor at Rest During Compression and Exposure to Pressures Equivalent to 800 and 1200 fsw (Phase I)

	EXPOSURE DAY 1												EXPOSURE DAY 2							EXPOSURE DAY 5						POST-EXP.
Elapsed time (hr:min)	0	0:21	0:41	1:01	1:26	1:57	3.33	3:57	4:16	4:57	5:37	6:34	0	0:00	0:20	0:46	1:22	2.04	2:59	0	0:00	0:20	0:46	1:40	2:20	-
Depth range (fsw)	0	400-506	590-654	698-752	800	800	800	800-1010	1100-1159	1050-975	885-860	800	800	800-1000	1100-1150	1200	1050-980	885-855	800	800	800-1000	1100-1150	1200	1025-978	912-898	0
INTENTIONAL TREMOR																										
Integrated Amplitude (arbitrary units)																										
LJ	5.8	6.2	7.4	8.9	11.0	-	7.5	6.0	13.8	6.5	7.2	6.6	11.1	10.8	8.5	11.4	8.6	6.4	5.8	10.1	8.6	6.6	9.2	9.8	7.6	4.9
FS	10.3	14.9	12.9	17.4	16.9	17.9	17.7	16.6	21.0	16.7	15.1	14.6	12.1	10.7	14.1	19.7	15.3	10.7	12.8	-	-	-	-	-	-	-
CC	7.3	8.4	11.0	8.4	12.5	9.0	5.0	-	10.0	-	8.2	6.4	12.0	9.0	11.7	14.4	14.8	9.4	10.1	8.9	7.6	11.3	9.1	9.6	7.0	-
WS	-	9.3	9.4	11.7	19.7	15.0	14.7	8.4	22.4	14.8	10.5	11.2	19.3	13.6	21.4	18.7	19.0	17.7	14.7	-	-	-	-	-	-	-
Mean	7.8	9.7	10.2	11.6	15.0	14.0	11.2	10.3	16.8	12.6	10.3	9.7	13.6	11.0	13.9	16.1	14.4	11.1	10.9	9.5	8.1	9.0	9.2	9.7	7.3	-
±SEM	1.3	1.8	1.2	2.1	2.0	2.6	3.0	3.2	3.0	3.1	1.8	2.0	1.9	1.0	2.8	1.9	2.2	2.4	1.9	-	-	-	-	-	-	-
Dominant Frequency (Hz)																										
LJ	3	3	3	8	8	8	4	-	5	5	3	4	8	-	-	3	3	-	3	5	-	-	4	4	3	5
FS	-	8	8	8	8	8	8	8	8	8	8	7	8	-	8	8	7	7	-	-	-	-	-	-	-	7
CC	3	10	8	12	12	8	3	4	3	10	3	3	8	-	-	10	5	-	9	5	-	-	5	3	3	-
WS	5	4	4	4	5	8	8	4	8	5	3	3	3	-	6	-	-	3	-	-	-	-	-	-	-	5
Mean	3.7	6.3	5.8	8.0	8.2	8.0	5.8	5.3	6.0	7.0	4.2	4.2	6.8	-	-	7.0	5.0	-	-	-	-	-	-	-	-	5.7
±SEM	0.7	1.7	1.3	1.6	1.4	0	1.3	1.3	1.2	1.2	1.2	1.0	1.2	-	-	2.1	1.2	-	-	-	-	-	-	-	-	0.7
POSTURAL TREMOR																										
Integrated Amplitude (arbitrary units)																										
LJ	5.6	8.0	7.0	7.8	6.8	-	8.1	10.6	9.0	-	-	9.6	12.6	12.0	12.5	10.6	11.4	9.2	8.8	11.2	11.2	14.7	20.2	8.4	10.2	8.0
FS	4.7	6.2	5.9	5.9	7.9	7.1	6.1	8.6	11.4	7.7	7.6	7.1	4.9	9.3	10.1	10.9	9.1	8.9	9.0	-	-	-	-	-	-	-
CC	5.2	4.7	4.0	5.7	6.1	7.2	5.7	5.2	14.5	10.9	-	8.9	9.8	10.1	11.8	10.2	11.4	9.7	9.1	8.8	11.0	9.1	9.0	9.8	12.5	10.3
WS	-	7.1	6.9	8.3	7.0	5.4	5.2	6.0	6.4	9.8	8.0	4.8	13.4	13.6	14.1	11.6	13.3	13.1	12.1	-	-	-	-	-	-	-
Mean	5.2	6.5	5.9	6.9	6.9	6.6	6.3	7.6	10.3	9.5	7.8	7.6	10.2	11.2	12.1	10.8	11.3	10.2	9.8	10.0	11.1	11.9	14.6	9.1	11.4	9.2
±SEM	0.3	0.7	0.7	0.7	0.4	0.6	0.6	1.2	1.7	0.9	-	1.1	1.9	1.0	0.8	0.3	0.8	1.0	0.8	-	-	-	-	-	-	-
Dominant Frequency (Hz)																										
LJ	8	7	8	8	8	-	8	-	8	-	-	8	8	-	-	8	7	-	8	8	-	-	8	8	8	8
FS	8	8	8	8	9	8	9	8	9	8	9	8	8	-	9	9	9	9	8	-	-	-	-	-	-	8
CC	10	10	5	10	10	10	10	10	-	10	5	-	10	-	-	10	8	-	6	10	-	-	9	9	8	8
WS	-	6	6	6	6	6	5	8	7	8	5	8	10	-	10	8	-	4	8	-	-	-	-	-	-	9
Mean	8.7	7.8	6.8	8.0	8.2	8.0	8.0	8.7	8.0	8.7	6.3	8.0	9.0	-	-	8.8	8.0	-	7.5	-	-	-	-	-	-	8.3
±SEM	0.7	0.8	0.8	0.8	0.9	1.2	1.1	0.7	0.6	0.7	1.3	0	0.6	-	-	0.5	0.6	-	0.5	-	-	-	-	-	-	0.2

TABLE II. Intentional and Postural Tremor at Rest During Compression and Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

	PRE-EXP.	EXPOSURE DAY 1										EXPOSURE DAY 2							EXPOSURE DAY 3						EXP. DAY 8	POST-EXP.
Elapsed time (hr.min)	-	0	0.13	0·32	0:53	2.14	2.53	3·12	3.56	5:26	7:20	0	0·03	0:12	0:35	1.26	2.04	2.56	0	0:02	0:25	1:16	1:54	2.46	-	-
Depth range (fsw)	0	0	400-567	656-736	800	800	860-1030	1110-1160	1200	1200	1200	1200	1260-1430	1280-1560	1600	1425-1365	1285-1260	1200	1200	1280-1520	1600	1425-1375	1285-1260	1200	1200	0
INTENTIONAL TREMOR																										
Integrated Amplitude (arbitrary units)																										
MP	7.0	4.7	4.2	5 4	5.4	7.3	6.3	5.5	6.8	6 4	4.4	4.8	-	6.1	10 1	7.7	5.8	6.3	5.0	22.9	7.3	9.6	6.6	7.1	6.9	3.6
FS	10.4	11.7	-	-	-	-	-	26 8	33.4	14.6	14.8	15.4	22.4	21.8	28.5	22.5	21.6	22.6	19.2	31.5	28.4	32.1	30.1	21.5	18.4	16.3
CC	7.9	9 3	11.1	11.8	13.9	7.6	16.4	14.1	16.0	17.1	11.4	15 9	-	14.0	11.3	12.3	15.4	12.8	19.0	16 7	15.2	11.1	13.5	8.1	14.0	7.8
GM	11.1	10 1	-	-	-	-	-	24.1	7.5	8.1	9.1	18 8	17.4	24.3	23.4	26.9	18.7	12.2	16.3	16.4	20.0	14.4	12.5	10.7	9 1	7.6
Mean	9.1	9.0	-	-	-	-	-	17.6	15.9	11.6	9.9	13.7	-	16.6	18.3	17.4	15 4	13.5	14.9	21.9	17.7	16.8	15.7	11.9	12 1	8.8
±SEM	1.0	1.5	-	-	-	-	-	4.9	6.2	2.6	2.2	3.1	-	4.1	4.5	4.4	3.4	3.4	3.4	3.5	4.4	5.2	5.0	3.3	2.6	2.7
Dominant Frequency (Hz)																										
MP	-	6.0	6.5	6.0	6.5	7.0	6.0	13.0	12.0	6.0	6.0	6.5	-	6.0	6.0	8.0	8.0	6.0	6.5	8.5	7.0	7 0	7.0	7.0	8.5	7.0
FS	-	8.0	-	-	-	-	-	8.0	8.0	8.0	7.5	8.0	8.0	9.0	8.5	8.0	8.0	7.0	7.0	9.0	8.0	8.0	8.0	8.5	7.0	7.0
CC	-	9.0	9.0	10.0	9.0	9.0	9.5	10.0	9.0	9.0	9.0	10.0	-	8.0	8.0	9.0	9.0	9.0	9.5	7.5	9.0	9.0	9 0	9.0	9 0	12.0
GM	-	8.0	-	-	-	-	-	8.0	6.5	7.5	7.0	8.0	7.0	10.5	7.0	10.0	9.0	10.5	6.0	11.0	9.0	8.0	8.0	7.7	9.0	7.0
Mean		7.8	-	-	-	-	-	9.8	8.9	7 6	7.4	8.1	-	8.4	7.4	8.8	8.5	8.1	7.2	9.0	8.2	8.0	8.0	8.1	8.4	8.3
±SEM		0.6	-	-	-	-	-	1.2	1.2	0.6	0.6	0.7	-	0.9	0.6	0.5	0.3	1.0	0.8	0.7	0.5	0.4	0.4	0.4	0.5	1.3
POSTURAL TREMOR																										
Integrated Amplitude (arbitrary units)																										
MP	3.5	2 1	2.8	2.8	3.3	3.3	3.1	3.0	3.6	5 2	2.4	3.3	-	3.6	3.6	3.6	2.6	3.0	3.1	14.5	3.1	3.3	3.0	2.4	4.3	3.0
FS	5.5	8.0	-	-	-	-	11.0	16.5	14.9	10.1	7.3	8.6	11.2	10.6	17.4	11.4	8.7	6.3	6.9	17.4	11.0	12.6	10 4	6.7	8.4	6.5
CC	4.9	4.0	-	5.3	5 3	3.9	8.5	8.2	17.7	9.8	6.7	6.1	-	5.4	10.1	9.0	8.8	4 3	4.1	7.6	6.6	6.8	5.1	3.3	3.8	7.1
GM	7.0	6 8	-	-	-	-	-	9.8	4.6	3.6	12.3	4.3	7.6	8.0	8.2	7.6	7.6	4.9	3.8	7 7	12.4	9.9	7.1	7.1	7.8	6.6
Mean	5.2	5.2	-	-	-	-	7 5	9.4	10.2	7.2	7.2	5.6	-	6.9	9.8	7.9	6.9	4.6	4.5	11.8	8.3	8.2	6 4	4.9	6.1	5.8
±SEM	0.7	1.3	-	-	-	-	2.3	2.8	3.6	1.6	2.0	2.3	-	1.5	2 9	1.6	1.5	0.7	0.8	2.5	2.1	2.0	1 6	1.2	1.2	0.9
Dominant Frequency (Hz)																										
MP	-	5.0	6.0	6.0	4.0	5.0	6.0	6.5	4.0	6.0	7.0	4.0	-	9.0	9.0	7.0	7.0	6.0	10.0	8.0	8.0	9.0	9.0	8.0	11.0	10.0
FS	-	9.0	-	-	-	-	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	8.0	8.0	9.0	8.5	9.0	8.5	8.0	8.0	9.0	9.0	9.0
CC	-	8.0	-	5.5	8.0	6.0	5.0	5.0	4.0	6.0	7.0	4.0	-	10.0	7.0	10.0	10.0	4.0	8.0	6.0	8.0	9 0	9.0	5.0	4.0	5.0
GM	-	10.0	-	-	-	-	-	10.0	10.0	9.5	10.0	9.0	15.0	12.0	14.0	10.0	10.0	14.0	15.0	15.0	10.0	10.0	9.0	13.5	7.5	12.0
Mean	-	8 0	-	-	-	-	6.7	7.6	6.8	7.6	8.3	6.5	-	10.0	9.8	8.8	8.8	8.2	10.4	9.5	8.6	9.0	8.8	8.9	7.9	9.0
±SEM	-	1.1	-	-	-	-	1.2	1.1	1.6	0.9	0.8	1.4	-	0.7	1.5	0.8	0.8	2 2	1.6	1.9	0.5	0.4	0.2	1.8	1 5	1.5

TABLE III Intentional and Postural Tremor in Bandwidths of 3-7 Hz and 8-12 Hz at Rest During Compression and Exposure to Pressures Equivalent to 800,1200 and 1600 fsw (Phase II)

	EXPOSURE DAY 1						EXPOSURE DAY 2					EXPOSURE DAY 3						EXP. DAY 8	POST-EXP.
Elapsed time (hr:min)	0	0:53	2:14	3:12	3:56	5:26	0	0:12	0:35	1:26	2:04	2:56	0	0:02	0:25	1:16	1:54	2:46	-	-
Depth range (fsw)	0	800	800	1110-1160	1200	1200	1200	1280-1560	1600	1425-1365	1285-1260	1200	1200	1280-1520	1600	1425-1375	1285-1260	1200	1200	0
INTENTIONAL TREMOR (integrated amplitude)																				
3-7 Hz Bandwidth																				
MP	0.51	1.17	1.07	1.52	1.19	0.51	0.51	0.94	1.19	5.00	5.00	1 12	1.27	12.70	1.32	1.98	1.47	1.63	1.17	0.51
FS	1.17	-	-	13.65	25.71	8 25	11.27	10 79	18.41	8.87	8.81	12.30	13.57	12.38	14.29	18.73	12.46	8.89	7.94	7.62
CC	1.42	5.87	6.98	8.10	7.94	6.67	6.98	7.30	5.87	5.16	6.35	5.95	7.70	7.94	7.62	6.51	5.24	5.87	7.62	3.33
GM	4.92	-	-	6.35	3.52	3.83	7.14	9.52	10.16	8.25	8.57	6 19	8.25	8.10	10.32	9.52	5.40	6.35	5.95	3.17
Mean	2.00	-	-	7.40	9.59	4.82	6.48	7.14	8.91	6.82	7.18	6.39	7.70	10.28	8.39	9.18	6.14	5.68	5.67	3.66
±SEM	0.99			2.50	5.55	1.70	2.22	2.19	3.66	1.01	0.91	2.29	2.52	1.31	2.72	3.54	2.29	1.51	1.56	1.47
8-12 Hz Bandwidth																				
MP	0.25	0.25	0 25	0.71	0.25	0.25	0.30	0.22	0.28	3.65	2.38	0.66	0.30	11 75	0.74	1.27	0.30	0.75	0.28	0.75
FS	1.90	-	-	16.51	16.55	6.03	5.71	13.65	15.56	12.38	10.16	9.37	8.73	22.86	20.40	17.78	18.10	11.75	10.38	6.98
CC	2.86	7.62	5.63	4.60	4.29	2.54	5.40	6.83	5.71	8.89	3.49	4.44	3.97	6.19	10.48	6.35	5.56	5.71	6.98	3.17
GM	1.90	-	-	4.44	2.54	4.13	4.13	6.35	6.98	6.27	4.13	4.13	4.29	4.76	5.40	4.76	3.17	2.54	2.54	1.60
Mean	1.73	-	-	6.56	5.91	3.24	3.88	6.76	7.13	7.80	5.04	4.65	4.32	11.39	9.26	7.54	6.78	5.19	5.04	3.12
±SEM	0.54			3.43	3.64	1.22	1.24	2.74	3.16	1.86	1.74	1.79	1.73	4.11	4.21	3.57	3.92	2.42	2.26	1.38
POSTURAL TREMOR (integrated amplitude)																				
3-7 Hz Bandwidth																				
MP	0.22	0.19	0.25	0.22	0.67	0.20	0.25	0.29	0.25	0.25	0.32	0.22	0.20	1.75	0.22	0.22	0.20	0.18	0.24	0.21
FS	0.38	-	-	1.46	0.76	0.54	0.48	0.76	1.02	1.44	0.63	0.41	0.57	0.73	0.83	0.79	0.63	0.48	0.38	0.38
CC	0.25	0.15	0.37	1.02	0.86	0.70	0.63	0.44	1.05	0.54	0.44	0.25	0.38	0.67	0.41	0.54	0.54	0.60	0.41	0.40
GM	0.32	-	-	0.70	0.16	0.48	0.32	0.44	0.29	0.67	0.35	0.32	0.32	0.32	0.51	0.51	0.16	0.48	0.22	0.16
Mean	0.29	-	-	0.85	0.61	0.48	0.42	0.48	0.65	0.72	0.44	0.30	0.37	0.87	0.49	0.52	0.38	0.44	0.31	0.29
±SEM	0.04			0.26	0.16	0.10	0.08	0.10	0.22	0.25	0.07	0.04	0.08	0.31	0.13	0 12	0.12	0.09	0.05	0.06
8-12 Hz Bandwidth																				
MP	0.16	0.16	0.22	0.17	0.05	0.16	0.18	0.44	0.38	0.20	0.22	0.15	0.20	1.90	0.17	0.18	0.20	0.20	0.20	0.18
FS	0.70	-	-	1.77	1.43	0.67	0.73	0.57	1.33	0.95	0.79	0.51	0.70	1.59	1.40	1.40	0.95	0.73	0.76	0.73
CC	0.29	0.07	0 35	0.76	0 54	0.67	0.51	0.63	0.89	0.70	0.63	0.38	0.41	0.63	0.48	0.41	0.41	0.41	0.25	0.60
GM	0.73	-	-	1.08	0.48	1.30	0.35	0.83	0.67	0.70	0.70	0.68	0.65	0.76	1.11	1.11	1.11	0.63	0.54	0.54
Mean	0.47			0.94	0.62	0.70	0.44	0.62	0.82	0.64	0.58	0.43	0.49	1.22	0.79	0.78	0.67	0.49	0.44	0.51
±SEM	0.14			0.33	0.29	0.23	0.12	0.08	0.20	0.16	0.13	0.11	0.12	0.31	0.28	0.29	0.22	0.12	0.13	0.12

TREMOR BEFORE AND AFTER EXERCISE

Tremor measurements made at rest before and after exercise are summarized for exercise subjects CC and WS in Phase I and CC and GM in Phase II in Tables IV and V, respectively. Mean integrated amplitudes of intentional and postural tremor for subjects CC and GM before and after exercise are shown with the composite pressure-exposure profile of Phase II in Fig. 4. Most of the differences between the pre- and post-exercise mean values are small, and post-exercise values appear to be neither consistently greater nor less than pre-exercise values (Tables IV and V).

SOMATOSENSORY EVOKED RESPONSE

Mean latencies of the SSER components measured in Phase II are shown with the composite pressure-exposure profile in Fig. 5 (Table VI). Only the three values of the secondary N2 component obtained at 1200 fsw on exposure day 1 were significantly different from the pre-exposure control values. The remaining mean values of N2 and all values of P2 and N1 were remarkably similar to their respective controls despite repeated exposures to rapid compression and increased hydrostatic pressures.

DISCUSSION

In a previous experiment series (Predictive Studies III) saturation pressure equivalent to 1200 fsw was reached after nearly seven days of stepwise increase of helium pressures through experiment stages at 400, 700, 900 and 1200 fsw. Small but definite increases in intentional tremor were measured with the strain gauge tremor transducer between 400 and 900 fsw, and 900 and 1200 fsw, in spite of the slow compression (7).

In the present studies, sensitive methods for measurement of finger "intentional" tremor and arm "postural" tremor gave clear indications of increased involuntary motor activity in association with rapid compressions and at stable high pressures (Figs. 1 and 2). As shown in these

TABLE IV. Intentional and Postural Tremor Before and After Exercise During Compression and Exposure to Pressures Equivalent to 800 and 1200 fsw (Phase I)

	EXPOSURE DAY 1									EXPOSURE DAY 2				EXPOSURE DAY 5				POST-EXP.
Elapsed time (hr:min)	0	0:21	0:41	1:01	1:26	1:57	3:33	3:57	4:16	0	0:00	0:20	0:46	0	0:00	0:20	0:46	-
Depth range (fsw)	0	400-579	590-688	698-786	800	800	800	800-1080	1100-1191	800	800-1060	1100-1180	1200	800	800-1060	1100-1180	1200	1
INTENTIONAL TREMOR																		
Integrated Amplitude (arbitrary units)																		
CC-Rest	7.3	8.4	11.0	8.4	12.5	9.0	5.0	-	10.0	12.0	9.0	11.7	14.4	8.9	7.6	11.3	9.1	-
Exer.	4.7	9.8	12.7	9.8	11.9	7.5	8.0	9.0	9.4	12.4	15.5	14.5	14.6	11.5	9.5	15.8	15.9	12.9
WS-Rest	-	9.3	9.4	11.7	19.7	15.0	14.7	8.4	22.4	19.3	13.6	21.4	18.7	-	-	-	-	-
Exer.	6.4	17.9	10.4	14.4	11.4	13.6	13.9	15.4	22.3	14.7	27.7	21.3	20.7	-	-	-	-	-
Mean-Rest	-	8.9	10.2	10.1	16.1	12.0	9.9	-	16.2	15.6	11.3	16.6	16.6	-	-	-	-	-
Exer.	5.6	13.8	11.6	12.1	11.7	10.5	10.9	12.2	15.8	13.6	21.6	17.9	17.7	-	-	-	-	-
Dominant Frequency (Hz)																		
CC-Rest	3	10	8	12	12	8	3	4	3	8	-	-	10	5	-	-	5	-
Exer.	12	12	8	8	5	3	3	4	3	6	-	-	8	4	-	-	5	9
WS-Rest	5	4	4	4	5	8	8	4	8	3	-	6	-	-	-	-	-	5
Exer.	4	4	4	4	4	4	4	4	4	5	-	-	5	-	-	-	-	-
Mean-Rest	4.0	7.0	6.0	8.0	8.5	8.0	5.5	4.0	5.5	5.5	-	-	-	-	-	-	-	-
Exer.	8.0	8.0	6.0	6.0	4.5	3.5	3.5	4.0	3.5	5.5	-	-	6.5	-	-	-	-	-
POSTURAL TREMOR																		
Integrated Amplitude (arbitrary units)																		
CC-Rest	5.2	4.7	4.0	5.7	6.1	7.2	5.7	5.2	14.5	9.8	10.1	11.8	10.2	8.8	11.0	9.1	9.0	10.3
Exer.	5.0	5.2	6.2	8.0	7.2	8.6	5.1	8.9	10.4	9 2	10.6	9.0	9.1	8.1	18.0	11.1	13.0	8.4
WS-Rest	-	7.1	6.9	8.3	7.0	5.4	5.2	6.0	6.4	13.4	13.6	14.1	11.6	-	-	-	-	-
Exer.	6.1	7.6	9.0	10.2	7.8	6.4	6.0	6.2	5.5	13.3	14.4	14.0	14.6	-	-	-	-	-
Mean-Rest	-	5.9	5.4	7.0	6.6	6.3	5.4	5.6	10.4	11.6	11.8	13.0	10.9	-	-	-	-	-
Exer.	5.6	6.4	7.6	9.1	7.5	7.5	5.6	7.6	8.0	11.2	12.5	11.5	11.8	-	-	-	-	-
Dominant Frequency (Hz)																		
CC-Rest	10	10	5	10	10	10	10	10	-	10	-	-	10	10	-	-	9	8
Exer.	10	-	5	10	10	10	10	10	-	10	-	-	10	8	-	-	9	8
WS-Rest	-	6	6	6	6	6	5	8	7	10	-	10	8	-	-	-	-	9
Exer.	5	6	4	5	4	5	5	5	7	-	-	-	-	-	-	-	-	-
Mean-Rest	-	8.0	5.5	8.0	8.0	8.0	7.5	9.0	-	10	-	-	9.0	-	-	-	-	8.5
Exer.	7.5	-	4.5	7.5	7.0	7.5	7.5	7.5	-	-	-	-	-	-	-	-	-	-

TABLE V. Intentional and Postural Tremor Before and After Exercise During Compression and Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

	PRE-EXP.	EXPOSURE DAY 1										EXPOSURE DAY 2				EXPOSURE DAY 3			EXP. DAY 8	POST-EXP.
Elapsed time (hr:min)	0	0	0.13	0.32	0:53	2·14	2:53	3:12	3:56	5·26	7:20	0	0:03	0:20	0:35	0	0:02	0·25	-	-
Depth range (fsw)	0	0	400-640	656-792	800	800	860-1100	1110-1195	1200	1200	1200	1200	1260-1500	1280-1595	1600	1200	1280-1590	1600	1200	0
INTENTIONAL TREMOR																				
Integrated Amplitude (arbitrary units)																				
CC-Rest	7.9	9.3	11.1	11.8	13.9	7.6	16.4	14 1	16.0	17.1	11.4	15.9	-	14.0	11.3	19.0	16.7	15.2	14.0	7.8
Exer.	7.2	13.0	15.2	12.3	14.3	14.8	28.6	18.4	12.3	9.8	11.0	9.4	-	15.3	19.3	31.4	18.1	15.9	18.4	13.2
GM-Rest	11.1	10.1	-	-	-	-	-	24.1	7.5	8.1	9.1	18.8	17.4	24.3	23.4	16.3	16.4	20.0	9.1	7.6
Exer.	14.7	11.2	-	-	-	-	26.7	-	9.1	8.9	8.3	12.3	20.2	16.6	19.3	21.1	13.7	18·6	15.3	8.8
Mean-Rest	9.5	9.7	-	-	-	-	-	19.1	11.8	12.6	10.3	17.4	-	19.2	17.4	17.7	16.6	17.6	11.6	7.7
Exer	11.0	12.1	-	-	-	-	27.7	-	10.7	9.4	9.7	10.9	-	16.0	19.3	26.3	15.9	17.3	16.9	11.0
Dominant Frequency (Hz)																				
CC-Rest	-	-	9.0	10.0	9.0	9.0	9.5	10.0	-	9.0	9.0	10.0	-	8.0	8.0	9.5	7.5	9.0	9.0	12.0
Exer.	-	11.5	8.0	11.0	8.0	7.0	4.0	8.5	8.0	6.5	8.0	10.0	-	8.5	9.5	9.0	9.0	9.0	8.0	8.0
GM-Rest	-	8.0	-	-	-	-	-	8.0	6.5	7.5	7.0	8.0	7.0	10.5	7.0	6.0	11.0	9.0	9.0	7.0
Exer.	-	7.0	-	-	-	-	7.0	-	7.0	8.0	10.0	8.0	-	10.0	10.0	7.0	9.0	8.0	9.0	7.0
Mean-Rest	-	-	-	-	-	-	-	9.0	-	8.3	8.0	9.0	-	9.3	7.5	7.8	9.3	9.0	9.0	9.5
Exer.	-	9.3	-	-	-	-	5.5	-	7.5	7.3	9.0	9.0	-	9.3	9.8	8.0	9.0	8.5	8.5	8.5
POSTURAL TREMOR																				
Integrated Amplitude (arbitrary units)																				
CC-Rest	4.9	4.0	-	5.3	5.3	3.9	8.5	8.2	17.7	9.8	6.7	6.1	-	5.4	10.1	4.1	7.6	6.6	3.8	7.1
Exer.	4.6	5.9	5.2	7.4	4.6	4.6	9.9	7.9	14.7	14.8	11.2	7.4	-	7.5	8.9	4.9	6.3	6.1	4.3	3.4
GM-Rest	7.0	6.8	-	-	-	-	-	9.8	4.6	3.6	12.3	4.3	7.6	8.0	8.2	3.8	7.7	12.4	7.8	6.6
Exer.	10.5	5.0	-	-	-	-	4.8	-	9.2	10.2	10.0	4.4	-	9.4	5.3	7.8	9.3	11.9	6.1	7.2
Mean-Rest	6.0	5.4	-	-	-	-	-	9.0	11.2	6.7	9.5	5.2	-	6.7	9.2	4.0	7.7	9.5	5.8	6.9
Exer.	7.6	5.5	-	-	-	-	7.4	-	12.0	12.5	10.6	5.9	-	8.4	7.1	6.4	7.8	9.0	5.2	5.3
Dominant Frequency (Hz)																				
CC-Rest	-	8.0	-	5.5	8.0	6.0	5.0	5.0	4.0	6.0	7.0	4.0	-	10.0	7.0	8.0	6.0	8.0	4.0	5.0
Exer.	-	6.0	8.0	5.0	5.0	5.0	5.0	4.0	4.0	5.0	5.0	4.0	-	6.0	8.0	6.0	7.0	6.0	6.5	7.0
GM-Rest	-	10.0	-	-	-	-	-	10.0	10.0	9.5	10.0	9.0	15.0	12.0	14.0	15.0	15.0	10.0	7.5	12.0
Exer.	-	10.0	-	-	-	-	11.0	-	10.0	9.5	10.5	9.0	-	12.0	14.0	14.2	14.0	11.8	10.0	10.0
Mean-Rest	-	9 0	-	-	-	-	-	7.5	7 0	7.8	8.5	6.5	-	11.0	10.5	11.5	10.5	9.0	5.8	8.5
Exer.	-	8.0	-	-	-	-	8.0	-	7.0	7.3	7.8	6.5	-	9.0	11.0	10.1	10.5	8.9	8.3	8.5

TABLE VI. Central Nerve Conduction Times During Exposure to Pressures Equivalent to Depths of 800, 1200 and 1600 fsw (Phase II)

	PRE-EXP.	EXPOSURE DAY 1				EXPOSURE DAY 2			EXPOSURE DAY 3			EXP. DAY 8	POST-EXP.
Elapsed time (hr:min)	-	0	1:18	4:42	5:55	7:46	0	0:59	3:20	0	0:47	3:10	-	-
Depth (fsw)	0	0	800	1200	1200	1200	1200	1600	1200	1200	1600	1200	1200	0
N1 Component of SSER (msec)														
MP	19	18	15	15	18	15	15	18	15	15	15	15	15	15
FS	21	20	20	20	20	24	20	18	20	24	20	20	18	22
CC	21	25	20	20	20	20	25	25	25	25	25	25	30	22
GM	16	15	15	18	20	15	15	18	15	15	15	15	15	14
Mean	19.2	19.5	17.5	18.3	19.5	18.5	18.8	19.8	18.8	19.8	18.8	18.8	19.5	18.3
±SEM	1.2	2.1	1.4	1.2	0.5	2.2	2.4	1.8	2.4	2.8	2.4	2.4	3.6	2.2
P2 Component of SSER (msec)														
MP	53	55	60	50	50	60	55	60	54	56	80	60	55	50
FS	59	60	58	60	60	60	50	60	60	58	54	60	60	58
CC	60	70	75	90	90	90	80	80	80	80	85	80	80	80
GM	60	60	65	60	64	60	60	65	62	62	60	62	60	65
Mean	58.0	61.3	64.5	65.0	66.0	67.5	61.3	66.3	64.0	64.0	69.8	65.5	63.8	63.3
±SEM	1.7	3.2	3.8	8.7	8.5	7.5	6.6	4.7	5.6	5.5	7.5	4.9	5.5	6.4
N2 Component of SSER (msec)														
MP	113	114	115	155	155	130	135	145	135	130	155	125	130	110
FS	109	110	110	120	120	130	120	110	110	110	112	110	110	110
CC	108	100	130	145	143	175	120	110	110	105	100	100	110	100
GM	107	100	125	150	150	150	125	125	115	115	120	122	122	110
Mean	109.3	106.0	120.0	142.5	142.0	146.3	125.0	122.5	117.5	115.0	121.8	114.3	118.0	107.5
±SEM	1.3	3.6	4.6	7.8	7.7	10.7	3.5	8.3	6.0	5.4	11.8	5.8	4.9	2.5

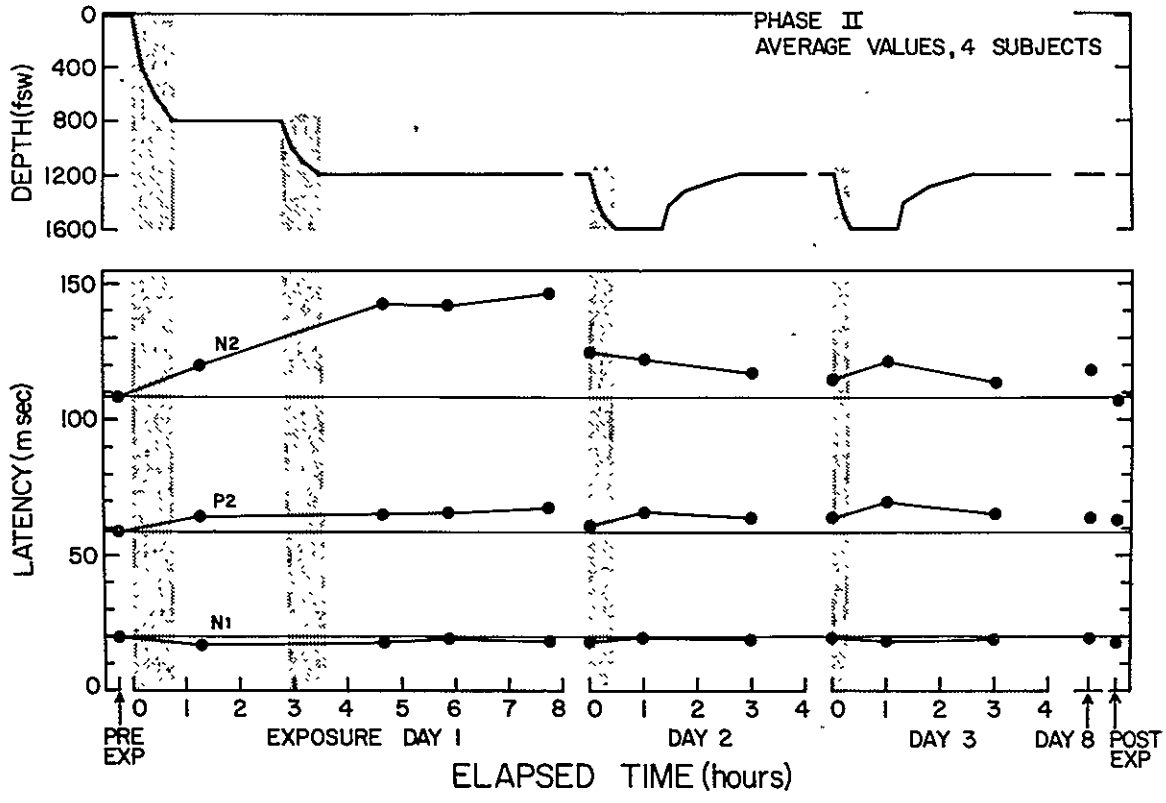

FIG. 5. Mean latencies of somatosensory evoked cortical responses during compression and exposure to simulated depths of 800, 1200 and 1600 fsw in Phase II. All points are averages for subjects CC, GM, MP and FS. Latencies are shown for primary (N1) and secondary (P2, N2) components of the somatosensory evoked response. Horizontal lines drawn through the pre-exposure data are for reference.

figures and in Section E-1, few signs of visible tremor or spasmodic twitches of muscles in arms or legs occurred concurrently in Phase I.

While the more severe compression exposures in Phase II resulted in only slightly greater levels of measured tremor than in Phase I (Figs. 1 and 2), they did elicit instances of effect (such as, shakiness, jumpiness, edginess, muscle fasciculations or jerks, or "excess energy" in the legs) more frequently--especially on exposure day 1 involving compression from 800 to 1200 fsw. These signs and symptoms occurred most frequently during the transient periods of maximal measured tremor (Fig. 2). However, it must also be emphasized that these grossly or visibly evident effects were brief in duration, and actual tremor was rarely directly visible during these periods.

The practical problem in interpreting tremor as measured by sensitive devices is that the finding of a doubling of a pre-existing small degree of normal tremor still did not indicate development of prominent or functionally significant tremor. These subjects evidently were not handicapped at any time by the tremor itself or by the other observed signs in the performance of either the tremor measurements or the large variety of other assigned tasks.

However, on exposure day 1 in Phase II as 1200 fsw was reached during the stressful initial sequence of compression from 1 ata to pressures equivalent to 400, 800 and 1200 fsw, symptoms such as nausea, fatigue and dizziness (without vertigo--except for brief periods of "spinning" as reported by GM and MP)[3] limited general subject competence for short periods of time (Sections E-1 and E-6). Nevertheless, even when such symptoms were absent, the measurement of increased tremor should be considered as indicating the presence of pressure effects, although small and not incapacitating. The data of the present study will be discussed in this context, with emphasis upon onset, persistence and possible adaptation. Despite the transient nature of the most visible

[3]These isolated reports of spinning episodes were not accompanied by signs of vertigo, for which there was continuous monitoring by electronystagmography (Section E-6).

changes, the small magnitude of any persistent effects, and the small number of subjects studied, some of the observed increases are statistically significant. Moreover, consistent response patterns which occurred during compression undoubtedly have a physiological basis related to acute increase in pressure.

TREMOR--INTENTIONAL AND POSTURAL

Influences of Initial and Excursion-Compressions

With few exceptions, the mean integrated amplitudes of both intentional tremor and postural tremor were consistently increased during and immediately following periods of rapid compression (Figs. 1 and 2). Also consistent were the partial or complete recoveries to pre-compression levels (although not always to 1 ata levels) while the subjects decompressed from excursions or remained at constant increased pressure. This similarity of response patterns for both tremor types is important to considerations of extent of effect and neural pathways involved (1). Thus, the effects of rapid compression on both types of tremor appear to be mediated by actions on more than one of the neural circuits, some elements of which may be common. The finding that the separate bands of low (3-7 Hz) and higher (8-12 Hz) frequency tremor spectra during the compression-pressure exposures showed no differences in response supports this conclusion.

Influences of Exposure to High Saturation Pressure

Even the control amplitudes of intentional tremor, measured at the stable holding pressure of 1200 fsw on exposure days 2, 3 and 8, were significantly greater than the controls obtained prior to compression at normal ambient pressure (Fig. 2). The observation that the first measurement of intentional tremor amplitude at stable saturation pressure on the morning of exposure day 2 at 800 fsw (Fig. 1) or day 2 at 1200 fsw (Fig. 2) was greater than the last measurement at the same pressure during the preceding evening is consistent with the previous finding that intentional tremor amplitude varies with the rest-activity cycle of night and day (3).

Influence of Exercise

The data do not show a consistent effect of prior exercise on either intentional or postural tremor (Fig. 4, Tables IV and V). Apparently there are no neuromuscular, circulatory or metabolic alterations which have a residual influence on the amplitude of tremor measured immediately after the cessation of exercise.

Influence of Temperature

Augmentation of tremor amplitude in association with ambient temperature rise of 7^{o}-15^{o}C has been observed (10,11). No correlations between tremor magnitude and skin or deep body temperature were found or were expected over a skin temperature range which was limited to 3^{o}C by the precise control of chamber ambient temperature during compression and decompression in this Predictive Study (Section E-15). In the opposite direction, a marked exaggeration of apparent tremor amplitude produced by shivering in response to a transient small decrease in chamber ambient temperature at the start of decompression did occur as a several-minute episode in subject FS .

Comparison of Intentional Tremor and Postural Tremor

While significant though small increases in average values of intentional tremor persisted at the stable pressure of 1200 fsw in Phase II, the 1200 fsw pre-excursion measurements of postural tremor were not different from pre-compression controls at 1 ata. During this phase, postural tremor adapted completely by returning to pre-exposure levels following excursions, while intentional tremor adapted only in part, returning to pre-excursion levels.

Other differences in postural and intentional tremor responses were found in Phase I (Fig. 1). The increase in tremor amplitude during the excursion from 800 to 1200 fsw on exposure day 2 was greater for intentional tremor than for postural tremor. In addition, the amplitudes of postural tremor at the stable pressure of 800 fsw in Phase I did not return entirely to sea level values as they did at 1200 fsw in Phase II. The inconsistency of the postural

tremor data (as compared with the data for intentional tremor) in relation to compression effect in Phase I, and between Phases I and II in pressure effect, and the apparent absence of a residual pressure effect during the exposures to the higher pressures of Phase II indicate that the pathways involved in the genesis of these two types of tremor were not affected identically by the exposures of Predictive Studies IV.

IMPULSE CONDUCTION

Except for the secondary N2 component of the somatosensory evoked cortical response, no measured conduction times were altered by rapid compression and exposure to increased hydrostatic pressures (Fig. 5 and Table IV). Thus no excitability changes were detected in the related sensorymotor pathways of the peripheral nerves, spinal cord, brainstem, thalamus and cortex. Even the N2 component was significantly increased only on the most extreme, initial compression to 1200 fsw on exposure day 1. Since the conduction latency of this secondary component includes synaptic delays within the central nervous system, the observed changes may have been caused in part by nonspecific effects such as fatigue which all subjects experienced during the latter half of exposure day 1 (Section E-1).

COMPARISON WITH OTHER INVESTIGATIONS

These findings in rapid compression (Predictive Studies IV) are consistent with effects observed in slow compression to the same 1200 fsw saturation pressure in Predictive Studies III (7). In each case measured intentional tremor increased, but tremor was not generally visible. Direct comparisons with most previous tremor studies are precluded by differences in methods and procedures (1). However, an earlier saturation exposure of six subjects at 1600 fsw (2) employed methods for measuring intentional tremor nearly identical to those used here. The mean integrated amplitude of intentional tremor at 1600 fsw in those subjects was approximately three times the sea level control value compared with maximum changes of two times the control level in Phases I and II of Predictive Studies IV (Figs. 1 and 2). Neither change is large. The subjects of the 1600-fsw

saturation study reported difficulty in the performance of manual tasks such as arterial catheterization and various diving procedures (2). Although they learned to improve their performance during the seven-day saturation exposure at 1600 fsw by compensating for tremor, their measured mean tremor amplitude did not diminish.

Conditions of saturation, other external influences or individual variability could account for the differences between increments of tremor amplitude in the different groups of subjects. However, feelings of well-being and psychomotor performance of subjects in Predictive Studies IV exceeded that of the subjects saturated at 1600 fsw in the earlier study (2). Although some decrements in measured psychomotor performance scores were found during and after the initial compression to 1200 fsw in this study (Section E-10), a high level of functional capability was demonstrated and sustained or improved during subsequent excursions to 1600 fsw on exposure days 2 and 3.

The decline in tremor amplitude during the first few hours at stable pressure after rapid compression to 800 or 1200 fsw (Figs. 1 and 2) is consistent with previous findings (1,4). Despite this early adaptation, there was a significant increase in measured tremor during most of the subsequent excursion-compressions, and even some of the pre-excursion control values of tremor amplitude were elevated. Persistence of increases in tremor amplitude also were found at the start and end of a seven-day sojourn at 1600 fsw (2). In Predictive Studies IV the sustained increase in tremor amplitude had no detectable functional correlates in the ability to participate in psychomotor performance testing (Section E-10), in the complex perceptual-motor skills involved in each subject's preparation for the measurement sequences (Section D), or in the paced performance of a complex underwater work task (Section F).

The mean changes in dominant frequency of tremor found in the subjects during compression were smaller than those reported by others (2), and they were not statistically significant (Fig. 3). In addition, most average dominant frequencies in Phase II (Fig. 3) were within or close to the range of 8-12 Hz which contains a large frequency component of all normal tremors (1).

C-3

Extensive measurements of postural tremor as one means of evaluating three different compression profiles in pressure exposures equivalent to depths of 1640 to 2001 fsw have been made (3). Although technical differences prevent quantitative comparison of those results with results in Predictive Studies IV, the observations are relevant here. The combined measurements of tremor amplitude in all three exposures included increments ranging from 100% to nearly 700% at the greatest pressures, as compared with sea level control values. Since two different subjects were studied in each exposure, it is not possible to determine the relative contributions of individual variations, compression rate and absolute hydrostatic pressure to the observed results. Tremor amplitude varied widely even at stable pressures and was consistently greater in the morning. Despite the marked increases in tremor amplitude and other hydrostatic effects, the subjects were considered "able to perform their work and psychometric tests equally well during the dives as at the surface level" (3).

SUMMARY AND CONCLUSIONS

Integrated amplitudes of intentional and postural tremor increased twofold during rapid compression to 800 and 1200 fsw. These initial increases were partially or completely reversed within the first two to four hours of continued exposure to the increased holding pressure. Despite this early apparent adaptation, the amplitudes of both forms of tremor were usually again increased during 400-fsw excursions beyond the 800 and 1200 fsw saturation pressures on subsequent exposure days. Control values of intentional tremor amplitude at 1200 fsw were significantly greater than sea level control values when measured on exposure days 2, 3 and 8. Both similarities and differences in response patterns of intentional and postural tremor were consistent with actions of hydrostatic pressure on multiple sites, including sites common to both tremor types.

The neuromuscular, circulatory and metabolic responses to 6 minutes of light exercise had no consistent influence on the amplitudes of intentional and postural tremor measured immediately after cessation of exercise.

Alterations in mean skin temperature were kept within a range of 3°C and had no correlation with tremor amplitude.

Mean changes in the dominant frequency of tremor were small and not statistically significant.

The conduction latency of the secondary N2 component of the somatosensory evoked cortical response was the single measured index of peripheral and central nerve conduction time which changed significantly. This change was found only during the first few hours after the initial compression to 1200 fsw and may have been caused by pressure or by nonspecific effects such as fatigue, or by both.

Neither the compression-induced twofold initial increment in tremor amplitude nor the smaller sustained augmentation of intentional tremor could themselves be considered large enough to have caused detectable functional impairment. While gross symptomatic changes induced in the subjects by intentionally rapid compression led to impairment of performance, there were no tremor-related deficits in the psychomotor performance testing, in the perceptual-motor skills required to prepare for the measurement sequences, or in the coordinated fine and coarse motor activity of complex underwater work tasks following excursion-compressions from 1200 to 1600 fsw.

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E-5. MUSCLE STRENGTH AND COORDINATION

R. Gelfand[1] and C.J. Lambertsen[1]

Rapid compression of men and animals to extreme hydrostatic pressures produces neuromuscular effects evidenced grossly by coarse tremors, motor incoordination, muscle fasciculations and spastic movements (1-5,7,9,10). While the ultimate physiological and anatomical origins of these effects are not known investigations of discrete neural and neuromuscular loci are being accomplished (6).

In preparing for this study it was considered possible that, regardless of mechanism, visible effects such as tremor might be accompanied by measurable changes in coordinated application of muscle strength. It was therefore an intent of Predictive Studies IV to examine effects of compression and hydrostatic pressure on muscle strength and coordination, as possible components of any neuromuscular dysfunctions generated by pressurization. Several aspects of the overall program provided information concerning muscle action during the compression and pressure exposures.

METHODS

With the exception of grip strength, methods used for all of the measurements contributing to appraisal of muscle function are described in detail in other sections of this report. This section will review these appraisals of muscle function, including results obtained in: psychomotor measurements (Section E-10), certain pulmonary function studies (Section E-12), measurements of frequency and

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pattern of pulmonary ventilation (Section E-14), diaphragmatic electromyography (Section E-14), vocal communications (Section E-9), bicycle ergometry (Section E-14), visual function (Section E-8) and practical underwater work (Section F).

Hand grip muscle strength was measured by a dynamometer (Jamar Adjustable Dynamometer, Asimov Engineering Co., Los Angeles, California). With this instrument, grip force is hydraulically transmitted by a dual piston-cylinder arrangement to a bourdon gauge calibrated in pounds of applied force. Force exerted was read as the difference between the peak deflection of the gauge when the subject applied his maximal effort (retained by a peak-reading indicator) and the baseline deflection when no force was applied. Dynamometer malfunction at increased pressure prevented planned measurement of hand grip strength until Phase II.

Grip strength measurements were incorporated into the "exercise" measurement module of subject CC in Phase II and were made during the second and third minutes of light exercise on the bicycle ergometer. Only one trial per experiment module was made on the first day of compression, from sea level to 1200 fsw. On the next day, during the first excursion to 1600 fsw, three grip strength readings were recorded for each module. Thereafter the measurements were discontinued when the dynamometer was damaged during decompression to the surface for inspection.

RESULTS AND DISCUSSION

On the first day of rapid compression and exposure to 800 and 1200 fsw the subjects experienced essentially all of the overt neuromuscular effects which have previously been observed with compression to extreme hydrostatic pressures (see Section E-1). Not all of these effects occurred in each subject. Certain aspects of neuromuscular function were not detectably affected in any subject, and the degree to which particular derangements did occur also varied with the individual (Section E-1).

COORDINATED GENERATION OF FORCE

Hand Grip Strength

The forces developed during the limited hand grip measurement of muscle strength are shown in Table I, with the depths and times at which the measurements were made, and pertinent indications of the status of the subject. Due to practical problems the first reading during pressurization on exposure day 1 was not obtained until the subject had been compressed to 550 fsw. Subsequent readings showed a gradual decline in force throughout the remainder of the compression exposure, from a high of 85 pounds at 550 fsw, to a low of 60 pounds at 1200 fsw. During this period of compression the subject experienced ankle and shoulder pain, general weakness and fatigue. The first force measurement on the second day, at 1200 fsw, was up to 82 pounds, approximately equal to the initial period of compression from 1 ata. During the excursion to 1600 fsw force readings fluctuated between 82 and 67 pounds.

The dynamometer measurements summarized in Table I are consistent with a progressive decline in muscle strength throughout the first day of compression to 800 and 1200 fsw. This does not necessarily indicate actual decrement in capacity for force generation by the muscle itself, since a maximal effort may not have been expended for each measurement. Subject CC concurrently had an effort-dependent reduction in maximum voluntary ventilation, which is generated by the power of the respiratory muscles, and reported that he felt some general weakness immediately prior to compression from 800 to 1200 fsw. Even if the apparent decrement in grip strength was real, effective strength remained since subject CC still was able to exert a force of 60 pounds at the end of the long, strenous day of physiological stress and compression to 1200 fsw.

The fluctuations of grip strength as measured during the excursion to 1600 fsw on the second exposure day (Table I) do not correlate with the excursion profile. Consecutive trials over a two-minute interval show that higher readings were usually followed by a decline in force on subsequent trials. Possibly the initial trials with

TABLE I. Dynamometer Measurements of Muscle Strength and Concurrent Effects During Compression-Pressure Exposures to 800, 1200 and 1600 fsw (Subject CC, Phase II)

MEASUREMENTS			CONCURRENT EFFECTS		
Elapsed Time (hr:min)	Depth (fsw)	Force Exerted (lbs)	Elapsed Time (hr:min)	Depth (fsw)	Comments
Exposure Day 1					
0	0	No test	0:30	600	Mild joint pain right ankle (beginning 500 fsw) and right shoulder (starting 560 fsw) but only on motion.
0:22	550	85			
0:41	728	70	0:50	800	"All right" on arrival at 800 fsw; still "little" joint pain (right ankle, right shoulder) on motion; "little bit trembly"; "not quite as sharp as normal"; "about 95%."
1:02	800	70	1:56	800	No joint pains, no dizziness, "95% mentally"; normal appetite, hungry, had lunch.
2:23	800	70	2:50	800	Prior to 1200 fsw excursion, "thinking is a little bit distant, like 90 or 95%, like about 4 o'clock in the morning"; generally weak; no dizziness, headache, nausea or joint pain; "kind of faint when doing pulmonary functions."
3:02	1020	70			
3:21	1155	65	3:30	1200	On arrival at 1200 fsw, "mentally okay, about 90 to 95%"; "just a little bit edgy and jumpy"--"as if I'm all wound up tight"; "no quivering in legs"--"have all kinds of energy going through my legs and I can't get rid of it."
3:45	1200	65	3:45	1200	Increased intention tremor of hands; both legs also show coarse tremor; right leg jerking rhythmically; motion can be stopped voluntarily, but occurs periodically even while supporting body weight; feels "tight in all my muscles," "all strung out," "energy to burn"; no nausea or dizziness.
4:46	1200	60	5:20	1200	Feels tired, looks exhausted, completes module.
			5:45	1200	Allowed to rest and sleep for over an hour.
			6:56	1200	Awakened for final modules; "okay," "feel some tiredness but otherwise I am sharp"; legs were shaking "a little bit" during module.
7:11	1200	60	7:41	1200	Completed final module; looks very tired, exhausted, but otherwise without grossly observable effects; effectively removed experiment leads, stowed equipment, helped to prepare chamber living systems, had full dinner and uneventful night.
Exposure Day 2					
-1:15	1200	82,77,72	0	1200	Good breakfast and lunch prior to excursion; competent rigging of electrodes and apparatus; "wasn't fed enough"; had slight pain in right knee which went away; mental capacity "just about" normal; "not really" dizzy but "balance is a little bit off"; no vertigo.
-0:48	1200	82,77,72			
0:11	1520	77,72,72	0:20	1600	On arrival at 1600 fsw, mentally clear, had no dizziness or nausea; "occasionally I feel a little bit off-balance"; "a little bit jumpy"; "a little bit higher strung" than normal; "100% better" than previous night at 1200 fsw.
0:34	1600	77,82	0:45	1600	Slight increase in hand tremor; no leg tremor during module; "a little off-balance," "not really dizzy."
1:32	1370	67,67,67	1:15	1600	Prior to decompression, no apparent problems.
2:10	1252	82,72,67			
3:01	1200	67,67,67	2:44	1200	Apparently fine on arrival at 1200 fsw; had full dinner.

the higher readings required an extreme effort which was difficult to deliver repeatedly and consistently.

Measurement of muscle strength during rapid compression has not previously been reported. However, the effect of exposure to stable hydrostatic pressures to the equivalent of 610 meters of sea water on capacity to sustain static force (isometric contraction) has been measured (8). In two semi-reclining subjects, a steady load (force) of 16 kg was imposed parallel to the brachial biceps, and the maximum time this load could be maintained was measured. The load selected was approximately 50% of the maximum force which could be exerted by this muscle group. Only small changes, both increases and decreases in the force maintenance time, were seen over the course of the multi-day exposure to high ambient pressure. There were no signs of discrete muscle fatigue related to the pressure exposure, and electromyographic recordings were unaltered (8).

Subjective estimates of fatigue (gross fatigue, articular and muscular disturbances and a detailed inventory of fatigue sites) were also made in the same study (8). While both subjects reported some fatigue during the slow eight-day compression, these effects were very slight and without definite location, and differed markedly in characteristics between the two participating subjects. Both subjects reported considerably more fatigue during decompression than at the highest pressures. The lower half of the body was most affected and the authors postulated that this might have been due to a decompression-related impairment of circulation (8). It is also important to recognize that, due to the small diameter of the chamber compartment used, the subjects were not able to stand erect and move about normally throughout the many day exposure to extremely high pressures. This is in contrast with the full activity practical at the 1200 to 1600 fsw maximum pressures of this Predictive Study.

It thus appears that elevated hydrostatic pressure per se did not affect muscle strength or cause fatigue in Predictive Studies IV. The stresses of rapid compression from 1 ata to high pressure in conjunction with the almost continuous physical activity of carrying out experiments over a 12-hour period did elicit general, subjective fatigue and a decrease in grip strength in this study.

These changes were transient, having disappeared by the time measurements were begun on the subject's second exposure day. The effects of the subsequent rapid excursion compression, which covered a lesser pressure range and a shorter time span, were negligible.

Respiratory Muscle Strength and Coordination in Pulmonary Ventilatory Functions

To study muscle strength and coordination in its relation to respiratory function, the composite forces generated by inspiratory and expiratory muscles were measured. Measurements were made in terms of maximum inspiratory and expiratory pressure changes which could be voluntarily exerted by the subject against a closed airway, as a "dynamometer" index of respiratory muscle strength. Intrathoracic pressure was sensed via a balloon catheter with the balloon located in the lower third of the esophagus. Subjects CC and GM participated in this during Phase II as part of the related pulmonary function studies (Section E-12).

Peak intrathoracic pressures capable of being generated with an occluded airway during forced inspiration and expiration were not consistently affected by exposure to compression and high ambient pressure (Table II). Subject CC recovered by the next morning from the decline recorded on initial compression during his first exposure day. Decreased esophageal pressures accompanied the decrement in the maximum voluntary ventilation at 1200 fsw in subject CC (Section E-12). However, these were found to be effort-dependent rather than related to muscle weakness per se. This subject, who began with a relatively low maximum expiratory force, developed increased capacity for exerting expiratory pressure during the first four days of exposure.

Frequency and pattern of ventilation during the light exercise on the bicycle ergometer were similar at all depths to values obtained for equivalent levels of exercise at sea level (Section E-14). No evident incoordination of the respiratory act was found. Electromyography of the diaphragm also showed no impairment of muscular function, either in the form of "tremor" or as incoordination within the respiratory cycle.

TABLE II. Maximum Voluntarily Generated Respiratory Muscle Forces at 0, 800, 1200, 1600 fsw in Phase II

Depth (fsw)	SUBJECT CC			SUBJECT GM		
	Clock Time	Maximum Inspiratory Force (cm H_2O)	Maximum Expiratory Force (cm H_2O)	Clock Time	Maximum Inspiratory Force (cm H_2O)	Maximum Expiratory Force (cm H_2O)
Exposure Day 1						
0	1140	72	74	1152	111	189
800	1328	49	58	1423	111	237
800	1447	52	72	1540	116	220
1200	1618	39	56	1821	102	215
1200	1943	-	-	1946	101	202
1200	1943	43	46	2105	82	196
Exposure Day 2						
1200	1314	66	72	1404	108	205
1600	1428	34	55	1548	87	220
1200	1657	50	88	1801	119	210
Exposure Day 3						
1200	1215	69	83	1343	119	209
1600	1340	66	107	1453	118	210
1200	1557	66	112	1721	96	165

TABLE III. Psychomotor Abilities Pertinent to Neuromuscular Function

Psychomotor Test	Ability Tested	Major Effects Observed on Initial Compression Days	
		Phase I 0-400-800-1200-800	Phase II 0-400-800-1200
Rest Subject (FS)			
One-hand Compensatory Control Test	Manual tracking	"Learning" offset by time-related decrement (possibly fatigue).	Single large decrement at 800 fsw; small time-related decrement (possibly fatigue).
Key Insertion Test	Finger dexterity	Compression-related decrements	Compression-related decrements.
Visual Reaction Time	Reaction time	No sustained compression or pressure effects. Results obscured by change in test strategy by subject.	Transient compression or pressure-related decrements. Quick recovery.
Wrench and Cylinder Test	Manual dexterity	Transient decrement, compression-related.	Time-related decrement (fatigue?).
Exercise Subjects (CC,GM,WS)			
Grip Speed Test	Wrist-finger speed	Small time-related decrement (fatigue?) (CC). Essentially no effect (WS).	Small time-related decrement (CC). Transient compression-pressure-related decrement (GM).

PSYCHOMOTOR FUNCTIONS

The psychomotor measurements employed were designed to test motor coordination and speed, rather than muscle strength itself. They therefore bear upon coordination but only indirectly upon generation of force by muscle. The tests most pertinent to neuromuscular function included those concerned with manual tracking, reaction time, finger dexterity, manual dexterity, and wrist-finger speed (see Section E-10).

Continuous visual monitoring of the subjects indicated no evident defect in performance of the coordinated functions cited above. Quantitative performance measurements (Section E-10) for the first exposure day did show improvement, gradual decrement throughout the hours of compression, transient decrements, and an occasional prominent single decrement which may have been related to attention level (Table III). There is no definite indication of either failure of muscle strength or failure of motor coordination. However, it is probable that general and/or compression-related fatigue influenced motor function throughout the experiment day.

The effects cited in Table III and discussed in detail in Section E-10 were accompanied by small increases in tremor of the hands, involuntary muscle twitches and "ratchety" movements of the extremities. These were of a degree not expected to be functionally critical except in the performance of intricate tasks. Except as indicated in Section E-1, compensatory activity provided the ability to carry out the detailed technical maneuvers of the experiments even during the compressions.

EVALUATION OF NEUROMUSCULAR EFFECTS ON OVERALL FUNCTIONAL CAPABILITY

Competence in Verbal Communications

Since comprehensible articulation is a complex neuromuscular event which requires the coordinated actions of laryngeal, mouth and facial muscles, it was used as a fine index of compression effect on the neuromuscular functions

involved. Aside from the well-known physical distortion of speech caused by the helium-oxygen environment, phonation was not detectably impaired during either of the two compression exposures (Phases I and II). Even while manifesting severe subjective symptoms under the most stressful conditions, all subjects were able to communicate intelligibly with personnel located outside the chamber by means of electronic reconstitution of "helium speech" (Section E-9).

Eye Movement Coordination

By use of the surface electrode system employed for electronystagmography (Section E-6), combined with voluntary tracking of a moving pendulum, measurement of the coordination of external muscles of the eye was accomplished in each rest subject. No detectable alterations of eye movement coordination occurred throughout the compressions and periods at stable increased pressure.

Performance of Specific Physical Activity--Bicycle Ergometry

The coordinated leg and body exercise of bicycle ergometry used for study of ventilatory control in physical work was performed by subjects CC and WS in Phase I and subjects CC and GM in Phase II (Section E-14).

All but two of the scheduled exercise module periods were completed successfully by the subjects. Performance of the muscular exercise was generally smooth and precisely coordinated. In certain limited periods interference with this performance occurred. On his first exposure day in Phase I, CC had hip and ankle pains which caused intermittent interruptions of the exercise periods scheduled immediately prior to and during compression from 800 to 1200 fsw. In addition, when he was temporarily unable to exercise due to severe nausea, vomiting and dizziness on arrival at the 1200-fsw pressure on the first day of Phase II, subject GM was also instructed to dismount from the bicycle and be seated. Impairment of muscle or neuromuscular function did not appear--symptomatically or objectively--to contribute to any of these work interruptions.

Underwater Work Performance

Operational capability of the diver-subjects was evaluated by the underwater work task performed by CC and MP on their fifth exposure day and by GM and FS on their ninth (Section F). Direct observation, and subsequent analysis of video tapes and motion picture films, showed no evident impairment of muscle strength or coordination as each of the divers dismantled and reassembled wellhead components on excursing from 1200- to 1600-fsw pressure equivalents. The average time required to complete the task at 1600 fsw was 12.4 minutes as compared with an average time of 11.2 minutes at the underwater control state of 5 fsw.

SUMMARY AND CONCLUSIONS

Rapid compression and exposure to 400, 800 and 1200 fsw on exposure day 1 produced nonlimiting neuromuscular effects such as some increase in tremor, muscle fasciculations and myoclonic jerks (Section E-1). These effects were accompanied by, but were not necessarily responsible for, decrements in motor speed and coordination; these, while not grossly evident, were measurable (Section E-10). Hand grip strength tended to be reduced in the one subject (CC) who performed these measurements. Even with the more severe conditions of Phase II, the small magnitude of these decrements was such that limitations would probably be encountered only during the performance of the most strenuous physical tasks. Recovery from the overall effects of initial compression to 1200 fsw was sufficiently rapid that further compression to 1600 fsw on the second day of exposure produced much less evident neuromuscular effects than those observed on the initial day at 800 and 1200 fsw.

Many aspects of specific neuromuscular function were not detectably impaired even under the conditions of initial compression when symptoms of malaise and nausea occurred. The subjects instrumented themselves with precision, carried out the technically difficult physiological experiment series, and maintained the associated apparatus in the chambers throughout the exposure. After five days of exposure to high ambient pressures equivalent to 1200 fsw (and probably prior to this as well), each of four

divers had the ability to perform a complex underwater work task which required motor speed, strength and coordination; this was carried out as effectively and nearly as rapidly at 1600 fsw as had been done previously at 5 fsw. It can be judged that prominent or progressive muscular functional degradation was not a consequence of the rapid pressurizations employed in this predictive program.

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E-6. VESTIBULAR FUNCTION AND BALANCE

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The role of the vestibular apparatus and associated sensory and integrative systems is recognized as important in motion sickness associated with the sea, in aviation, and even in space and exploration, where elaborate mechanisms have been devised to protect man from changes in his gravitational environment (12). Current efforts to extend man's ability to work on the ocean floor have exposed divers to severe stresses of the vestibular apparatus. Decompression, presumably due to bubble formation, can produce transient or permanent loss of vestibular function (22,23,30). Supersaturation of tissues with inert gases leading to bubble formation can also occur due to the isobaric counter-diffusion phenomenon, producing vestibular dysfunction (24,34). Furthermore,many symptoms which occur during compression and exposure to high pressures and should not involve bubble formation resemble symptoms of vestibular disturbance (4,7,8,13,16,25,27,29).

The vestibular system functions in conjunction with visual orientation and proprioception to maintain spatial orientation (both sense of location and actual body orientation) through vestibulospinal and vestibulo-ocular reflexes (11). When vestibular dysfunction is present,

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complex symptoms such as malaise with pallor, sweating, drowsiness, nausea and vertigo may result, and dysmetria, nystagmus and inability to maintain balance may be observed. In diving these can be life-threatening.

The involvement of the vestibular system in decompression was recognized as early as 1873, when vertigo, vomiting and dizziness were recognized as symptoms of caisson disease, and it was noted that "affectations of the ear are mentioned by every writer on the subject of compressed air" (22,23). Subsequently, vestibular dysfunction was increasingly reported in diving experiments. The actual incidence of vestibular dysfunction may be even greater than reports indicate because symptoms such as malaise, weakness and nausea can be vague and not accompanied by physical signs (22,23). The high incidence (65%) of otological injury detected by examinations of professional divers (in spite of a negative history) (36), and the fact that hypofunction of the labyrinths has been detected in older divers substantiate the suspicion that vestibular injury may occur more frequently than has been reported. Modern saturation diving studies have reported a frequency of vestibular-type symptoms in as many as 50% of the subjects (23).

The possible involvement of the vestibular system in disorders related to compression and to high hydrostatic pressure per se is a recent concept. Reports of nausea, vomiting, dizziness, and vertigo, along with EEG changes, drowsiness, malaise, tremor and muscle fasciculations (16) have implicated neurophysiological dysfunction as a possible limiting effect in deep simulated diving experiments. Progressive decline of auditory-evoked response has suggested otoneurological injury which is central (36). Changes in visual-evoked response have also been observed (Section E-3) and are suggestive of pressure effects on the central nervous system. Direct evidence of an association between high pressure and vestibular dysfunction has recently been reported (14), and altered balance function has been observed at high pressure (6).

Predictive Studies IV provided an opportunity to extend observations of vestibular and balance functions to rapid compressions, with tracking of effects both during the act of compression and during exposure to stable high pressures. Repetitive measurements during pre-compression, compression and post-compression periods allowed for

subjective and objective evaluations of the transient and stable-state effects of compression and of hydrostatic pressure on the vestibular system.

MATERIALS AND METHODS

PRE-EXPOSURE AND POST-EXPOSURE CLINICAL EVALUATIONS

The six diver-subjects had comprehensive physical examinations which included an otolaryngological history and physical examination with laryngoscopy, nasopharyngoscopy and pneumatic otoscopy, before and after the compression-pressure-decompression exposures. The audiological examination included pure-tone thresholds and speech discrimination (Section E-7). The pre- and post-exposure vestibular function tests consisted of electronystagmography (ENG) with bithermal caloric testing, ocular fixation, pendulum tracking and optokinetic evaluation. Posturography (quantitative Romberg) and electrical vestibular stimulation (positive and negative polarization) were also performed.

TESTS DURING COMPRESSION-PRESSURE EXPOSURES

The vestibular function battery of tests, performed during experiment modules (Section D), required eight minutes to complete. These tests consisted of continuous ENG recordings, ocular fixation and pendulum tracking, posturography and electrical vestibular stimulation. The rest subjects (LJ and FS in Phase I, MP and FS in Phase II) were given the entire test battery while the exercise subjects (CC and WS in Phase I, CC and GM in Phase II) were monitored with continuous ENG recordings and ocular fixation.

Electronystagmography

Electronystagmographic recordings were obtained with silver-silver chloride electrodes applied to alcohol-cleaned skin at the outer canthus of each eye. The ground reference electrode was centered at the bridge of the nose.

The signals were coupled to the recording system by battery-operated D.C. preamplifiers (Princeton Applied Research model 113) for isolation from the A.C. line. Direct and velocity signals were inscribed on a strip chart recorder (Beckman model RM, with type 9859 direct nystagmus couplers and type 9841 nystagmus velocity couplers). Eye movement to the right produced an upward deflection. Deflection sensitivity was set so that 1^{o} eye deflection was shown by 1 mm pen deflection, with ocular fixation spots used for calibration. The rest subjects sat erect with eyes closed and looking straight ahead performing mental arithmetic aloud during recording periods. The exercise subjects were monitored with continuous ENG recordings. Recorder tracings were evaluated for signs of vestibular disturbance (spontaneous nystagmus).

Pendulum Tracking and Ocular Fixation

The subjects sat erect while visually tracking a brass cylinder suspended on a wire. This pendulum initially traversed an arc of 20^{o} at 10^{o}/second. While in the pendulum tracking position the subjects' eyes traversed an arc of 20^{o} between two spots painted on the chamber hull, each 10^{o} from center. Recorder tracings were examined for abnormalities in tracking and/or fixation responses.

Posturography

Recordings of balance function were obtained with a statometer constructed at the Naval Medical Research Institute. Strain gauges provided both x- and y- (lateral and sagittal) axis electrical outputs representing functions related to but not identical with the time course of change in body weight distribution (18,20,21). The electrical signals were recorded on magnetic tape (Ampex SP 300) for subsequent analysis.

Only the rest subjects in Phase I (LJ, FS) and in Phase II (MP, FS) participated in the study of balance function. Six minutes were allotted for posturography in each experiment module. The subject rose from the seated position, stepped onto the statometer and secured a safety harness designed to prevent injury if complete loss of balance occurred. He then stood quietly with arms at the

side (quantitative Romberg) and with eyes open as the strain gauge bridges were balanced to bring the statometer signals onto the scale of a strip chart recorder. After two minutes, the subject was instructed to close his eyes. Two minutes later, electrical vestibular stimulation began.

Quantitative analysis. The frequency range of interest extended to very low frequencies (3,21,31); therefore analog methods for elimination of static unbalance voltages from tape-recorded statometer signals could not be used since they cause distortion. A digital processing method was designed and implemented on a PDP-11 computer (Digital Equipment Corporation) as follows: The x- and y-axis analog signals from the tape recorder were passed through a two-channel low-pass filter (Krohn-Hite model 3342) with frequency response flat to 0 Hz and a 48 dB/octave roll-off set at 2.0 Hz. These signals were digitized and stored in computer memory, and the average (D.C. or zero frequency) value of the time segment selected for analysis was calculated. This value was subtracted from the original, providing a time varying unbalance signal symmetrical about zero volts, with frequency response flat to 0 Hz but with the 0 Hz component removed. Initial transients which occurred as the subjects positioned themselves on the statometer were excluded from the analyses. Positive and negative areas of the x- and y-axis signals, obtained by digital integration, were summed and divided by the time interval over which the signal was analyzed to obtain average sway amplitudes in arbitrary units. In this method of analysis (19), with D.C. offset voltages eliminated and no distortion of low frequencies, the time spent "off-balance" and the instantaneous amplitudes of the off-balance signals are weighted linearly. Absolute calibration of x- and y-axis responses were not obtained; therefore only relative changes in x- and y-axis responses are meaningful for purposes of comparison.

Electrical Vestibular Stimulation

Electrical stimulation of the labyrinth was used to circumvent several problems associated with caloric stimulation. Water irrigation was impractical in these studies since measurements of vestibular function were integrated into rapidly paced multi-experiment modules. In addition,

caloric stimulation is super-threshold and may itself induce vomiting, while electrical stimulation is a generative stimulus that can be applied at threshold levels and then stopped abruptly.

Polarization of the labyrinth was performed with a constant current stimulator which was applied to the mastoid as the subject stood on the statometer. Negative (stimulatory) polarization was applied to the test ear at 10-second intervals for a duration of 10 seconds using intensities of 0.3,0.4,0.6,0.8,1.0 and 2.0 ma. The negative polarization current was then applied to the other ear at 1.0 and 2.0 ma. Statometer signals were visually analyzed for evidence of altered thresholds.

RESULTS

PRE-EXPOSURE EXAMINATIONS

Prior to exposure all subjects had normal vestibular responses to bithermal caloric testing, ocular fixation and pendulum tracking. Optokinetic evaluation, posturography and electrical vestibular stimulation revealed no abnormalities. When electrically stimulated at 0.4 ma, all subjects experienced a sensation of rotation and tended to sway in the appropriate direction: away from the ear stimulated by negative polarization and toward the ear stimulated by positive polarization.

SYMPTOMS AND SIGNS DURING COMPRESSION-PRESSURE EXPOSURES

Symptoms typical of vestibular disturbance were reported by each subject during one or more of the compressions of Phases I and II, particularly during compressions from 1 ata. These symptoms included headache, drowsiness, dizziness, unsteadiness, nausea and vomiting (Section E-1). Figures 1-4 combine the results of the sway measurements with condensed versions of Figs. 1 and 2 in Section E-1, and show those comments by both rest and exercise subjects which can be associated with vestibular function derangements.

These symptoms were accompanied by the usual signs of vestibular dysfunction only once, when several short bursts of spontaneous nystagmus were observed in subject FS in Phase II at 1600 fsw on exposure day 3 (Fig. 3A).

Eye pursuit remained smooth and no ocular dysmetria was detected either during compressions or at pressures of 800, 1200 and 1600 fsw.

Symptoms usually resolved within several hours at stable pressures (Figs. 1-4). The intensity of the symptoms was greater during exposure to the more rapid compressions and higher pressures of Phase II. The exercise subjects experienced more intense symptoms than the rest subjects (Section E-1).

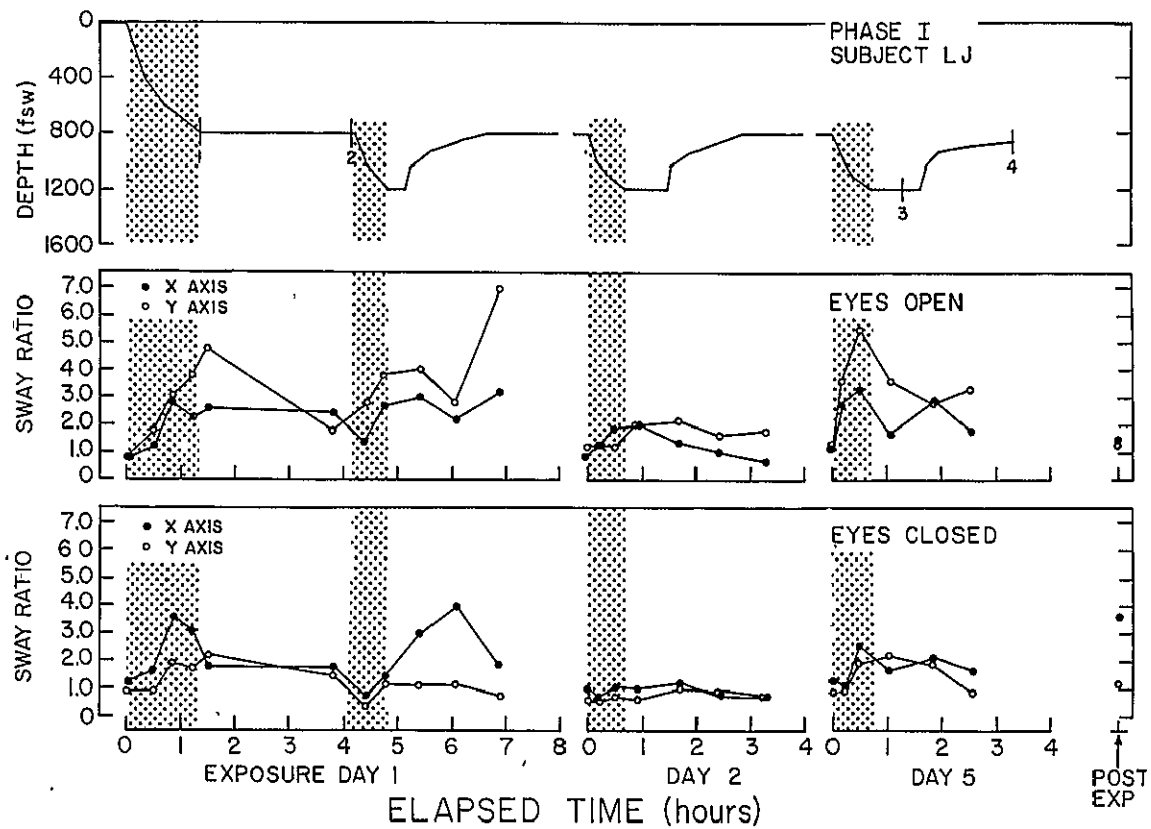
QUANTITATIVE POSTURAL SWAY MEASUREMENTS

The results of the quantitative analyses of the statometer signals are shown in Tables I-IV. A reference value for each subject was obtained by averaging measurements made at 1 ata during the interval between Phases I and II and those made immediately prior to compression to 800 fsw in Phase II (Tables I-IV). The ratios of all other sway values to these reference values were calculated and are plotted in Figs. 1A-4A, allowing comparison of compression and pressure effects on x and y axes and in the eyes-opened and eyes-closed situations.

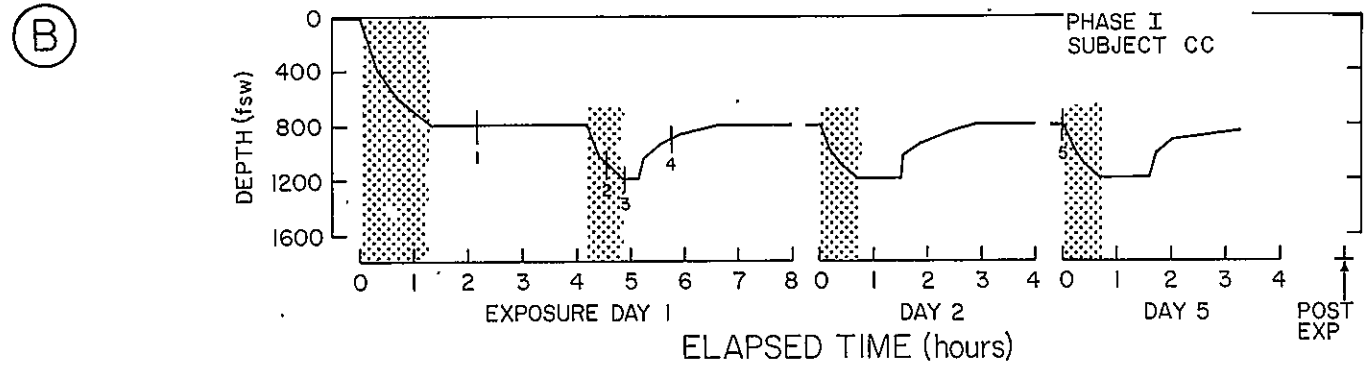
In the majority of cases, average sway amplitudes were larger for eyes closed than for eyes open and were greater in the x axis than in the y axis.

Data for subject LJ in Phase I (Fig. 1A) show markedly increased sway during compression from 0 fsw to 800 fsw and during the first excursion to 1200 fsw. The largest increases(up to 700% greater than control)occurred during the initial compression and during the excursion-decompression. Partial recovery occurred during the several-hour "hold" at 800 fsw, before the excursion to 1200 fsw. During the exposures, sway ratio increases were greater when eyes were open than when eyes were closed. With eyes open, the y-axis sway was affected more than the x-axis sway; the reverse occurred with eyes closed.

(A)



Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject LJ
1	Day 1	1:20	At about 500 fsw, had temporary sensation of clockwise rotation during electrical vestibular stimulation of right and left mastoid regions.
2		4:10	Had strong sensation of counterclockwise rotation during stimulation of left mastoid; right mastoid stimulation produced weak sensation.
3	Day 5	1:19	Felt nauseated and thought the vestibular electrical stimulation gave unusually strong responses; dizzy on standing.
4		3:23	Vestibular decompression sickness (see text and Section G-4).



Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject CC
1	Day 1	2:10	Had headache and joint pain.
2		4:33	Starting at about 1000 fsw, felt "a little slow" and "had a rocking motion." Described feeling as: "a little bit shaky," "achy," "seasick and queasy." Felt tired and fell asleep on the bicycle ergometer during some of the mental function tests.
3		4:53	Felt shaky; sitting on exercise bicycle he performed his roles but had trouble concentrating on tasks; seemed inattentive; slightly nauseous; "the air is hot and thick in here"; headache and dizziness gradually subsiding.
4		5:45	No longer dizzy but still "just a little bit sick to my stomach."
5	Day 5	0	Diarrhea in a.m. Still had headache and queasiness on beginning compression.

FIG. 1. Sway ratios during exposures to compression and pressure equivalents of 800 and 1200 fsw for subject LJ. Numbered vertical lines on the depth profiles refer to the symptoms listed below the graph for A, subject LJ and B, subject CC.

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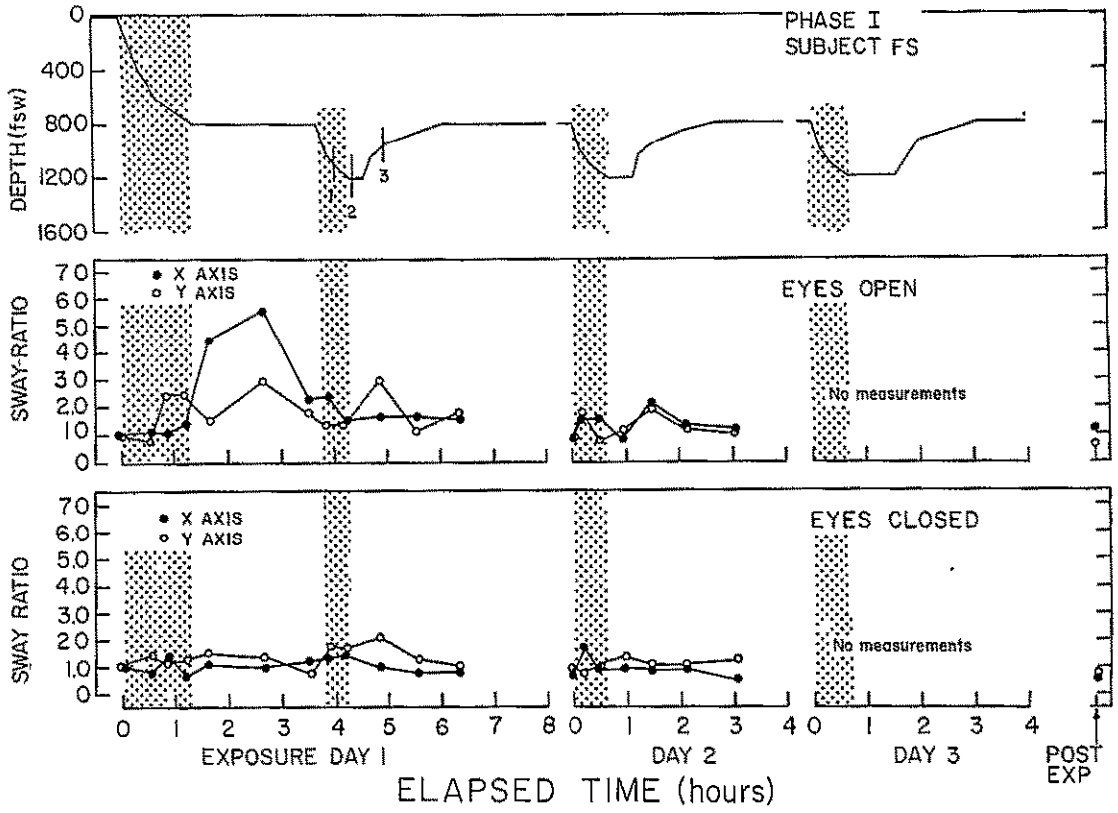
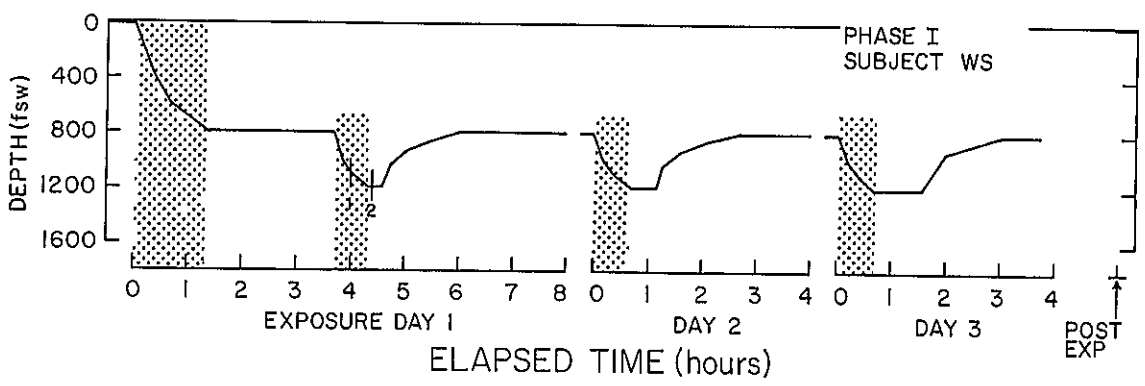


Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject FS
1	Day 1	4:01	Had "just a bit of a headache"--"right behind my eyes," "otherwise I'm all right." Headache was worse while watching flashing light for visual evoked response.
2		4:21	"I have a sort of dizziness when I get up and down, but I think that's just part of the headache. I pretty much feel O.K." Dizzy feeling on standing started soon after leaving 800 fsw.
3		4:54	Still had headache; no dizziness.

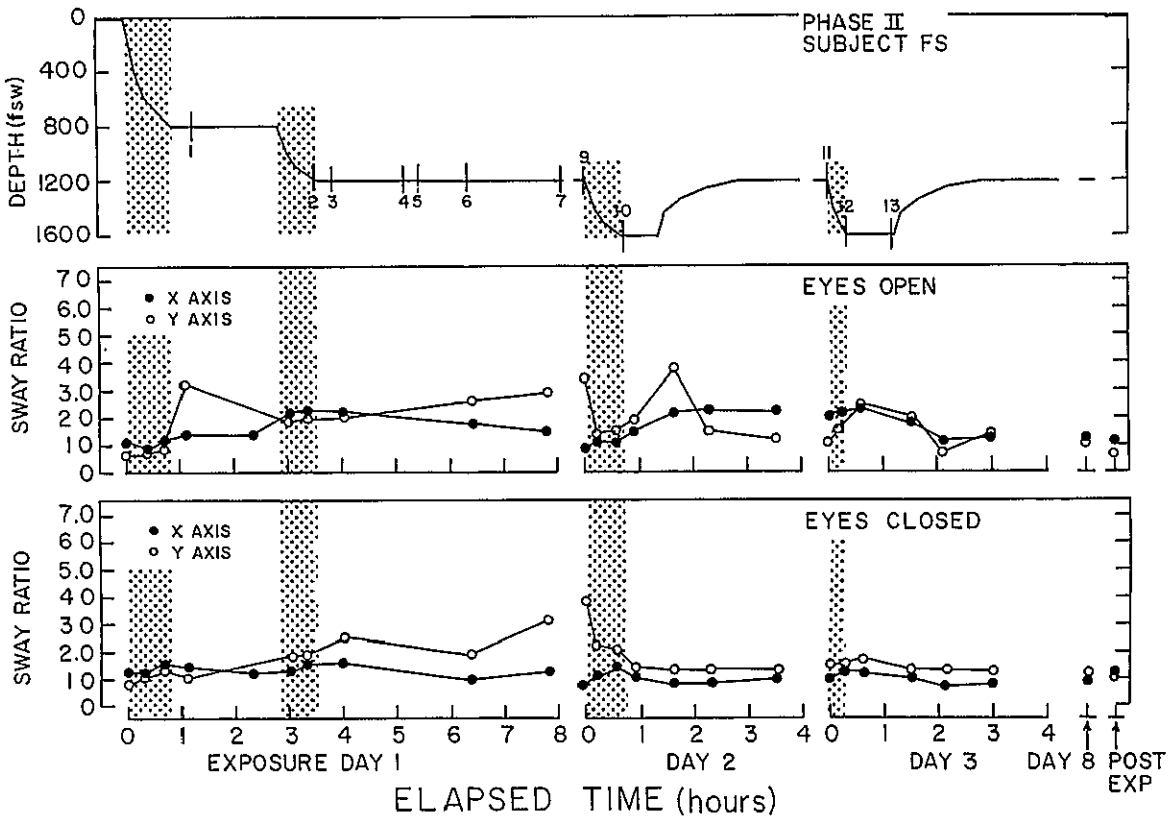
B



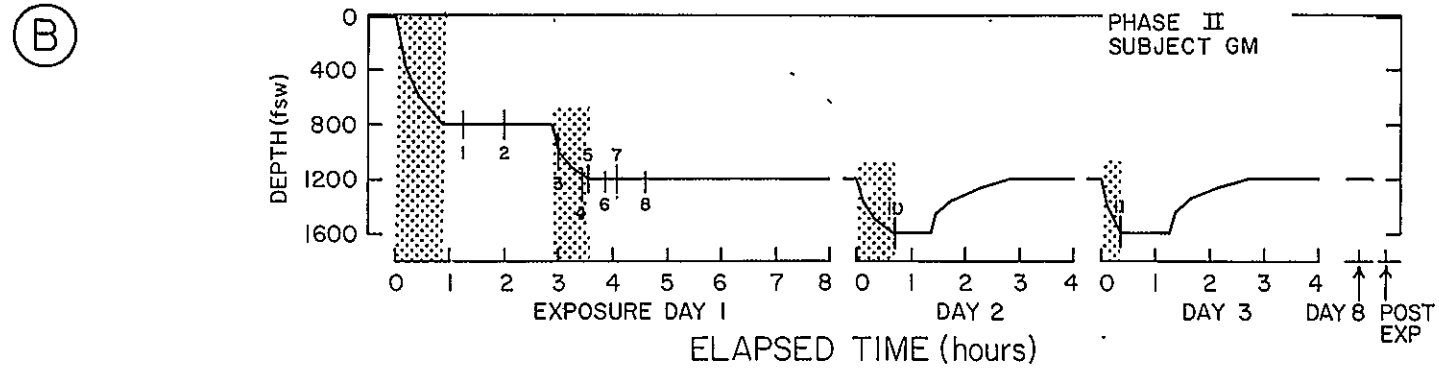
Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject WS
1	Day 1	4:01	Slightly dizzy upon standing: lightheaded, not vertigo.
2		4:21	Dizziness "went away before we got to the bottom." No other problems.

FIG. 2. Sway ratios during exposures to compression and pressure equivalents of 800 and 1200 fsw for subject FS. Symptoms listed are for A, subject FS and B, subject WS.

Ⓐ



Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject FS
1	Day 1	1:12	"Little bit" lightheaded.
2		3:30	On arrival at 1200 fsw felt "slightly lightheaded," only discomfort is "slight headache."
3		3:50	Became nauseated while standing on balance board (statometer) prior to vestibular electric shock.
4		5:08	Still nauseated but ready for next module.
5		5:25	Three min into module showed some postural incoordination, intermittent coarse tremor of muscles of neck, trunk and limbs.
6		6:50	Had headache again, eyes felt as though he must strain to see the test numbers; not blurred.
7		8:09	Still had headache.
8		9:00	Slight nausea. Headache not prominent. Essentially no supper (said he was not hungry).
9	Day 2	0	Had slight headache and "squeamishness" before breakfast. Felt fine now. Vestibular stimulation was stopped momentarily--was "too strong."
10		0:40	Lost headache during compression; "feels a little bit slow mentally"; unsteadiness on balance board (statometer) at 1480 and 1580 fsw, increasing when eyes are closed; hungry.
11	Day 3	0	Showed slightly increased sway on balance board.
12		0:20	Less postural sway on balance board than during previous excursion.
13		1:15	Some unsteadiness in standing to get on balance board; short bursts of nystagmus.



Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject GM
1	Day 1	1:12	"Tiny bit" lightheaded.
2		1:57	Suddenly became nauseated and vomited on drinking orange juice following about five glasses of water during compression, after 58 min at 800 fsw. Sweating; felt "hot" and "dizzy" on vomiting. Felt fine, "almost 100% " immediately after vomiting but with periodic recurrence of slight nausea. Felt better every time temperature dropped.
3		3:00	Felt disoriented and "little bit dizzy" on ocular fixation at 950 fsw. Required conscious effort not to fall from bicycle at about 1050 fsw.
4		3:26	"Dizzy," felt as though "turning to right." Dizziness was severe. At 1180 fsw mentally clear but felt "as though rocking back and forth," with increasing nausea.
5		3:30	Still "a bit dizzy" but slowly subsiding. Ten min after arrival at 1200 fsw dizziness "suddenly getting much better."
6		3:50	Excused from "exercise" module due to extreme feeling of fatigue.
7		4:03	Felt "a tiny bit weak" but much less disoriented. Gradually recovering.
8		4:32	Vomited small amount, asked permission to remove esophageal tubes, still slightly nauseated, still slight headache.
9		9:00	Essentially no supper (said he was not hungry).
10	Day 2	0:40	Had slight dizziness and slight nausea on intentional rapid head movement; slight lightheadedness and "slight" problem of concentration (says "mind" is 99%).
11	Day 3	0:20	Mentally "not quite 100%," "a little bit of dizziness on very rapid head movements and slight nausea.

FIG. 3. Sway ratios during exposures to compression and pressure equivalents of 800, 1200 and 1600 fsw for subject FS. Symptoms listed are for A, subject FS and B, subject GM.

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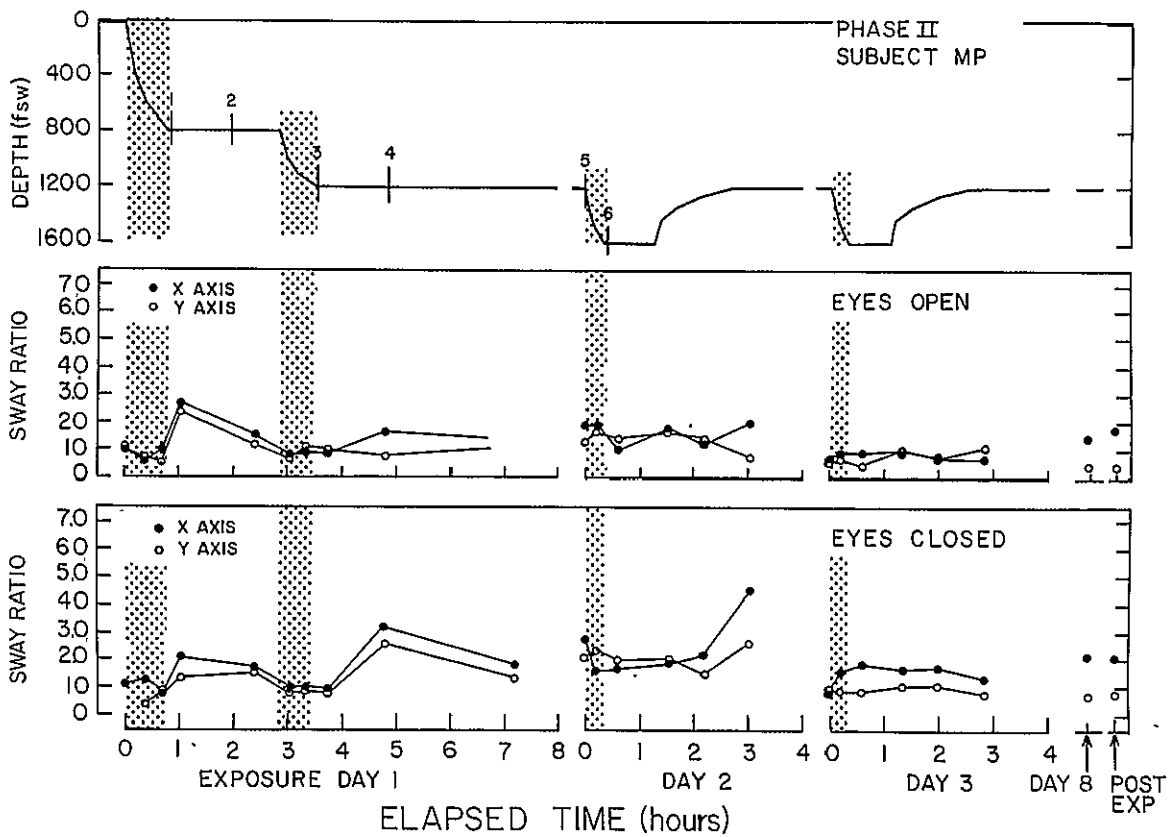
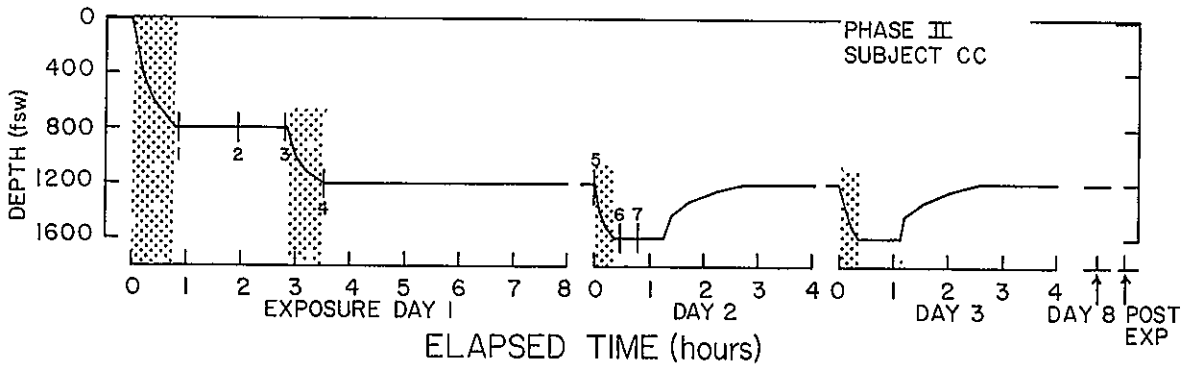


Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject MP
1	Day 1	0:50	"A little dizzy."
2		1:56	Some "dizziness" on standing, no vertigo, no nausea, things "jump" a little bit.
3		3:30	On arrival at 1200 fsw, felt mentally clear, "just a little bit shaky," no headache or nausea, "spinning a little" but "if I turn 5 or 6 degrees it reverses direction."
4		4:50	A little dizzy during electrical vestibular test but "otherwise feel fine."
5	Day 2	0	"A little bit slow and a little bit tired," little dizzy "if I move really fast."
6		0:20	"Not really dizzy," "wouldn't want to try standing up without holding onto something," "spinning a little bit," only with eyes closed on balance board.

(B)



Comment Number	Elapsed Time		Symptoms of Vestibular Dysfunction Subject CC
1	Day 1	0:50	"Not quite as sharp as normal," "about 95%."
2		1:56	"95% mentally."
3		2:50	"Thinking is a little bit distant, like 90 or 95%, like about 4 o'clock in the morning." Generally weak, "kind of faint when doing pulmonary functions."
4		3:30	"Mentally O.K., about 90 to 95%."
5	Day 2	0	Mental capacity "just about" normal. "Not really dizzy," but "balance is a little bit off." No vertigo.
6		0:20	"Occasionally I feel a little bit off-balance."
7		0:45	"A little off-balance," "not really dizzy."

FIG. 4. Sway ratios during exposures to compression and pressure equivalents of 800, 1200 and 1600 fsw for subject MP. Symptoms listed are for A, subject MP and B, subject CC.

TABLE I. Sway in Anterior-Posterior (x Axis) and Lateral (y Axis) Directions with Eyes Open and with Eyes Closed (Phase I, Subject LJ)

Exposure Day	Depth Range (fsw)	Elapsed Time[a] (hr:min)	EYES OPEN				EYES CLOSED			
			x Axis		y Axis		x Axis		y Axis	
			Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio
1 ata measurements			66		25		96		83	
			78		38		68		107	
			42				65			
			84				95			
			49							
Reference value[c] (Mean of 1 ata data)			64	1.0	32	1.0	81	1.0	95	1.0
1	0	0	49	0.8	25	0.8	95	1.2	83	0.9
	500-520	0:31	75	1.2	57	1.8	140	1.7	85	0.9
	659-671	0:51	178	2.8	98	3.1	289	3.6	178	1.9
	777-789	1:11	146	2.3	121	3.8	253	3.1	164	1.7
	800	1:31	166	2.6	153	4.8	142	1.8	210	2.2
	800	3:49	155	2.4	58	1.8	148	1.8	146	1.5
	1000-1020	4:25	91	1.4	85	2.7	62	0.8	39	0.4
	1150-1160	4:45	173	2.7	121	3.8	114	1.4	118	1.2
	975-965	5:24	195	3.0	130	4.1	243	3.0	100	1.1
	860-855	6:04	140	2.2	90	2.8	324	4.0	108	1.1
	800	6:52	203	3.2	222	6.9	150	1.9	69	0.7
2	800	0	52	0.8	38	1.2	78	1.0	55	0.6
	1000-1020	0:11	76	1.2	39	1.2	54	0.7	51	0.5
	1150-1160	0:31	112	1.8	38	1.2	83	1.0	65	0.7
	1200	0:54	127	2.0	65	2.0	80	1.0	53	0.6
	975-965	1:41	80	1.3	66	2.1	95	1.2	97	1.0
	853-848	2:24	66	1.0	50	1.6	67	0.8	77	0.8
	800	3:19	36	0.6	57	1.8	56	0.7	70	0.7
5	800	0	79	1.2	40	1.3	109	1.3	90	0.9
	1000-1020	0:11	181	2.8	111	3.5	92	1.1	81	0.9
	1150-1160	0:31	213	3.3	176	5.5	211	2.6	183	1.9
	1200	1:02	105	1.6	113	3.5	138	1.7	205	2.2
	975-965	1:51	176	2.8	85	2.7	173	2.1	183	1.9
	888-883	2:31	114	1.8	101	3.2	138	1.7	88	0.9
	1050[d]		649	10.1	135	4.2	140	1.7	52	0.5
Post-exp.	0	-	91	1.4	38	1.2	291	3.6	107	1.1

[a] Elapsed time is at the center of the 2-minute measurement interval (minutes 10-12 of module).

[b] Average sway amplitudes are in arbitrary units.

[c] See text for explanation of reference values. For this subject the post-exposure measurement was included in the calculation of the y-axis reference values.

[d] Measurements made shortly after resolution of nystagmus following recompression for decompression sickness therapy.

TABLE II. Sway in Anterior-Posterior (x Axis) and Lateral (y Axis) Directions with Eyes Open and with Eyes Closed (Phase I, Subject FS)

Exposure Day	Depth Range (fsw)	Elapsed Time[a] (hr:min)	EYES OPEN				EYES CLOSED			
			x Axis		y Axis		x Axis		y Axis	
			Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio
1 ata measurements			47		51		57		78	
			90		73		92		55	
			39		28		139		68	
			60		48		114		75	
Reference value[c] (Mean of 1 ata data)			59	1.0	50	1.0	101	1.0	69	1.0
1	0	0	60	1.0	48	1.0	114	1.1	75	1.1
	496-515	0:33	65	1.1	42	0.8	76	0.8	99	1.4
	644-654	0:53	65	1.1	125	2.5	137	1.4	81	1.2
	742-752	1:13	82	1.4	130	2.6	71	0.7	89	1.3
	800	1:37	268	4.5	72	1.4	112	1.1	101	1.5
	800	2:40	330	5.6	152	3.0	99	1.0	87	1.3
	800	3:30	136	2.3	88	1.8	126	1.2	47	0.7
	1000-1020	3:52	144	2.4	65	1.3	131	1.3	117	1.7
	1150-1160	4:12	88	1.5	69	1.4	155	1.5	107	1.6
	975-965	4:48	98	1.7	151	3.0	99	1.0	143	2.1
	860-855	5:32	99	1.7	59	1.2	77	0.8	93	1.3
	800	6:20	95	1.6	86	1.7	80	0.8	70	1.0
2	800	0	49	0.8	40	0.8	70	0.7	63	0.9
	1000-1020	0:11	93	1.6	92	1.8	167	1.7	47	0.7
	1150-1160	0:31	93	1.6	40	0.8	92	0.9	68	1.0
	1200	0:56	51	0.9	61	1.2	88	0.9	92	1.3
	975-965	1:26	124	2.1	95	1.9	86	0.9	62	0.9
	860-855	2:06	77	1.3	67	1.3	89	0.9	66	1.0
	800	3:02	70	1.2	55	1.1	55	0.5	85	1.2
Post-exp.	0	-	66	1.1	32	0.6	63	0.6	47	0.7

[a]Elapsed time is at the center of the 2-minute measurement interval (minutes 10-12 of module).

[b]Average sway amplitudes are in arbitrary units.

[c]See text for explanation of 1 ata reference value.

TABLE III. Sway in Anterior-Posterior (x Axis) and Lateral (y Axis) Directions with Eyes Open and with Eyes Closed (Phase II, Subject FS)

Exposure Day	Depth Range (fsw)	Elapsed Time[a] (hr:min)	EYES OPEN				EYES CLOSED			
			x Axis		y Axis		x Axis		y Axis	
			Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio
1 ata measurements			47		51		57		78	
			90		73		92		55	
			39		28		139		68	
			71		35		136		55	
Reference value[c] (Mean of 1 ata data)			62	1.0	47	1.0	106	1.0	64	1.0
1	0	0	71	1.1	35	0.7	136	1.3	55	0.9
	550-580	0:23	50	0.8	35	0.7	129	1.2	70	1.1
	718-733	0:42	77	1.2	42	0.9	171	1.6	101	1.6
	800	1:05	85	1.4	151	3.2	154	1.5	70	1.1
	800	2:22	86	1.4	-	-	142	1.3	-	-
	1020-1040	3:03	134	2.2	88	1.9	142	1.3	112	1.8
	1155-1165	3:22	142	2.3	93	2.0	166	1.6	122	1.9
	1200	4:00	135	2.2	96	2.0	161	1.5	159	2.5
	1200	6:24	112	1.8	120	2.6	100	0.9	121	1.9
	1200	7:49	92	1.5	134	2.9	138	1.3	201	3.1
2	1200	0	58	0.9	161	3.4	76	0.7	241	3.8
	1410-1430	0:13	67	1.1	65	1.4	117	1.1	141	2.2
	1550-1560	0:32	69	1.1	71	1.5	146	1.4	125	2.0
	1600	0:55	92	1.5	91	1.9	101	1.0	90	1.4
	1385-1375	1:38	129	2.1	178	3.8	87	0.8	81	1.3
	1265-1260	2:18	141	2.3	70	1.5	90	0.8	82	1.3
	1200	3:34	136	2.2	56	1.2	103	1.0	83	1.3
3	1200	0	123	2.0	48	1.0	102	1.0	92	1.4
	1510-1530	0:12	135	2.2	70	1.5	128	1.2	99	1.5
	1600	0:35	140	2.3	112	2.4	123	1.2	110	1.7
	1415-1405	1:31	112	1.8	88	1.9	101	1.0	77	1.2
	1260-1255	2:10	68	1.1	34	0.7	78	0.7	85	1.3
	1200	3:01	83	1.3	59	1.3	82	0.8	81	1.3
8	1200	-	74	1.2	53	1.1	93	0.9	78	1.2
Post-exp.	0	-	71	1.1	27	0.6	128	1.2	65	1.0

[a] Elapsed time is at the center of the 2-minute measurement interval (minutes 10-12 of module).

[b] Average sway amplitudes are in arbitrary units.

[c] See text for explanation of 1 ata reference value.

TABLE IV. Sway in Anterior-Posterior (x Axis) and Lateral (y Axis) Directions with Eyes Open and with Eyes Closed (Phase II, Subject MP)

Exposure Day	Depth Range (fsw)	Elapsed Time[a] (hr:min)	EYES OPEN				EYES CLOSED			
			x Axis		y Axis		x Axis		y Axis	
			Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio	Sway Amplitude[b]	Ratio
1 ata measurements			141		45		51		37	
			141		162		144		125	
			138		155		88		128	
			138				101			
Reference value[c] (Mean of 1 ata data)			140	1.0	121	1.0	96	1.0	97	1.0
1	0	0	138	1.0	-	-	101	1.1	-	-
	550-583	0:23	89	0.6	70	0.6	122	1.3	42	0.4
	729-745	0:42	137	1.0	60	0.5	78	0.8	73	0.8
	800	1:03	380	2.7	292	2.4	204	2.1	128	1.3
	800	2:24	215	1.5	146	1.2	166	1.7	148	1.5
	1020-1040	3:03	113	0.8	83	0.7	100	1.0	73	0.8
	1155-1165	3:22	120	0.9	137	1.1	93	1.0	91	0.9
	1200	3:46	119	0.9	120	1.0	91	0.9	76	0.8
	1200	4:47	242	1.7	97	0.8	304	3.2	249	2.6
	1200	7:12	193	1.4	142	1.2	175	1.8	124	1.3
2	1200	0	269	1.9	162	1.3	267	2.8	201	2.1
	1520-1540	0:12	267	1.9	205	1.7	157	1.6	229	2.4
	1600	0:35	144	1.0	171	1.4	164	1.7	192	2.0
	1370-1360	1:33	257	1.8	200	1.7	183	1.9	191	2.0
	1265-1260	2:11	171	1.2	171	1.4	207	2.2	145	1.5
	1200	3:02	284	2.0	83	0.7	445	4.6	256	2.6
3	1200	0	103	0.7	68	0.6	77	0.9	90	0.9
	1510-1530	0:12	120	0.9	80	0.7	150	1.6	92	0.9
	1600	0:35	119	0.9	52	0.4	184	1.9	84	0.9
	1380-1370	1:20	144	1.0	115	1.0	162	1.7	102	1.1
	1265-1260	1:59	101	0.7	82	0.7	165	1.7	106	1.1
	1200	2:50	101	0.7	128	1.1	126	1.3	74	0.8
8	1200	-	207	1.5	43	0.4	207	2.2	72	0.7
Post-exp.	0	-	256	1.8	43	0.4	199	2.1	82	0.8

[a] Elapsed time is at the center of the 2-minute measurement interval (minutes 10-12 of module).

[b] Average sway amplitudes are in arbitrary units.

[c] See text for explanation of 1 ata reference value.

Subject LJ's sway ratios at 800 fsw before the excursion on day 2 began were at the 0 fsw control levels. Sway changes in this excursion were minor when compared with day 1. Sway during the first measurement on exposure day 5 was also at the 0 fsw level. The excursion on this day resulted in sway increases comparable to those of day 1, and the largest increases were again with eyes open.

Subject LJ reported symptoms typical of vestibular dysfunction on three occasions, each time in association with electrical vestibular stimulation (Fig. 1A). He was asymptomatic on day 2, and during compression on day 5. However, at 1200 fsw on exposure day 5, LJ felt nauseous and dizzy on standing; he reported a strong response to vestibular stimulation. Although he felt well at the beginning of decompression, he subsequently developed an unequivocal and well documented case of vestibular decompression sickness (Section G-4). Sway following successful recompression therapy was essentially at the control level with eyes closed but was considerably increased with eyes open (Table I).

Subject FS in Phase I (Fig. 2A, Table II) had virtually no change in sway ratios with eyes closed on exposure day 1 but showed large transient increases (400%-600% over control) with eyes open. These peak increases occurred during compression to 800 fsw and following arrival at that depth. There was considerable recovery by the time the excursion to 1200 fsw began. The excursion itself resulted only in transitory increases in y-axis sway.

Sway was at the 0 fsw control level before the excursion on exposure day 2. Essentially no changes from control occurred with eyes closed during this excursion, and small transient increases during the excursion-compression and decompression occurred with eyes open.

In Phase II, changes in sway for subject FS generally resembled those in Phase I (Fig. 3A). The peak increase in sway with eyes open during the compression to 800 fsw was smaller than in Phase I. The excursions from 1200 to 1600 fsw in Phase II elicited somewhat larger sway responses than the 800-1200 fsw excursion in Phase I. Sway at 1200 fsw on day 8 and at 0 fsw after decompression were at control levels.

Subject FS reported symptoms reminiscent of vestibular dysfunction on several occasions in Phase I (Fig. 2A) and with considerably greater frequency in Phase II (Fig. 3A). These symptoms were associated directly with electrical vestibular stimulation only once (Fig. 3A, comment 9).

Subject MP in Phase II exhibited only transient sway increases following compressions on exposure day 1 (Fig. 4A) On day 2, sway was somewhat elevated compared with the 0 fsw level with no systematic changes during the excursion. Day 3 began with sway ratios at the control level. Only eyes-closed sway in the x-axis increased during the excursion on that day. Sway ratios at 1200 fsw on day 8 and at 0 fsw after the saturation-decompression were at control levels in the y axis but were above control levels in the x axis in both eyes-open and eyes-closed conditions.

Subject MP reported symptoms typical of vestibular dysfunction on six occasions (Fig. 4A), associated with electrical vestibular stimulation only once (comment 4).

Subject LJ first showed increased sway in the first measurement at about 500 fsw during the compression from 0 fsw on day 1, in Phase I with both eyes open and closed (Fig. 1A). For FS on day 1 of Phase I, increased sway first appeared at about 650 fsw with eyes open (Fig. 2A). For the same subject in Phase II, increased sway first appeared just after 800 fsw was reached, with eyes open (Fig. 3A). Increased sway in MP also first appeared just after reaching 800 fsw, with eyes open and closed (Fig. 4A).

CHARACTERISTICS OF SWAY PATTERNS

The average sway amplitude is one of many ways the statometer signals can be analyzed. While it measures alterations in balance function, the same numerical value of sway can be obtained with many different sway patterns. It is necessary to employ additional methods of data analysis (19,33) to characterize completely the time-varying sway signals. Currently it is not known which characteristics of these signals are most significant in diagnosing the physiological disturbances that cause altered sway patterns. To illustrate the changes in pattern which occurred in Predictive Studies IV, original recordings

obtained with subject LJ on the statometer are reproduced in Figs. 5-7. These tracings illustrate five major characteristics of sway:

1) very low frequency baseline fluctuations

2) low frequency fluctuations (in the range 0.1-0.5 Hz)

3) high frequency fluctuations (in the range 1-2 Hz)

4) abrupt anterior-posterior or lateral changes without sustained baseline shift

5) abrupt sustained shifts in baseline.

In Fig. 5A at 1 ata, the y-axis baseline was stable with low amplitude, high frequency fluctuations. When eyes were closed, a transient baseline disturbance occurred, and a low frequency component appeared with the high frequency component superimposed. The x-axis baseline was less stable, with very low frequency fluctuations upon which was superimposed a high frequency component. Figure 5B was recorded at approximately 660 fsw during compression from 1 ata. The x-axis baseline was unstable (drifting). Abrupt postural changes without sustained shift in baseline occurred. The higher frequencies were less prominent in the x axis with eyes open than at 1 ata and were almost completely absent with eyes closed. Compared with the x axis, the y axis baseline was more stable and the peak-to-peak sway amplitudes were smaller. With eyes closed, high frequency amplitude in the y axis was greater than at 1 ata.

Tracings shown in Fig. 6 were recorded at 800 fsw prior to compression and at 1200 fsw following compression on exposure day 2. They are similar to the pre-exposure control at 1 ata, except for occasional brief bursts of high frequency instability. At 800 fsw (Fig. 6A), the baseline shifted immediately prior to the eyes closing.

Tracings in Fig. 7 were recorded on exposure day 5. At 800 fsw, before compression began, the y axis with eyes open was similar to the recording at 1 ata, while with eyes closed there was increased amplitude of higher frequencies (Fig. 7A). The x axis showed higher amplitude, low frequency activity than at 1 ata. During decompression, approximately 1 hour

prior to the onset of LJ's vestibular decompression sickness on day 5 (Sections G-1 and G-4), the tracing showed some increased sway at low frequency in the x axis with the eyes closed (Fig. 7B). A review of preceding recordings on that day showed that a change in this sway pattern first occurred during decompression at approximately 1000 fsw (1 hour and 20 minutes before the onset of the decompression sickness). The tracing, which was recorded with eyes open after successful recompression therapy of the vestibular decompression sickness, showed a highly unstable baseline in the x axis (Fig. 7C). Correction of the initially rapidly drifting baseline occurred, thus maintaining equilibrium (Fig. 7C).

VESTIBULAR STIMULATION

Quantitative analysis of the statometer recordings during electrical stimulation of the mastoid was not possible since the subjects usually responded to the stimulation with a change in body orientation which frequently produced little deflection of the statometer tracings. However, observations of the subjects and visual interpretation of the recordings produced no objective evidence of hyperactivity of the labyrinth. No abnormal sway was detected in response to the stimulations, and the subjects' ability to compensate for the electrical stimulation by a re-orientation of body position remained at a high level throughout the exposures to compression and pressure.

Despite this lack of objective evidence of altered labyrinthine function, there was subjective evidence of labyrinthine hyperactivity. Subject LJ in Phase I reported a transient sensation of rotation during vestibular stimulation at 500 and 800 fsw on exposure day 1, and "strong response" to vestibular electrical stimulation on day 5 at 1200 fsw (Fig. 1A). MP reported feeling dizzy when stimulated electrically during Phase II on exposure day 1 at 800 fsw and on day 2 at 1600 fsw (Fig. 4A). FS held onto instrumentation cables during electrical stimulation on day 1 of Phase II at 1200 fsw (observation recorded in tape log) and stimulation was momentarily stopped on day 2 before compression at 1200 fsw, because it was "too strong" (Fig. 3A).

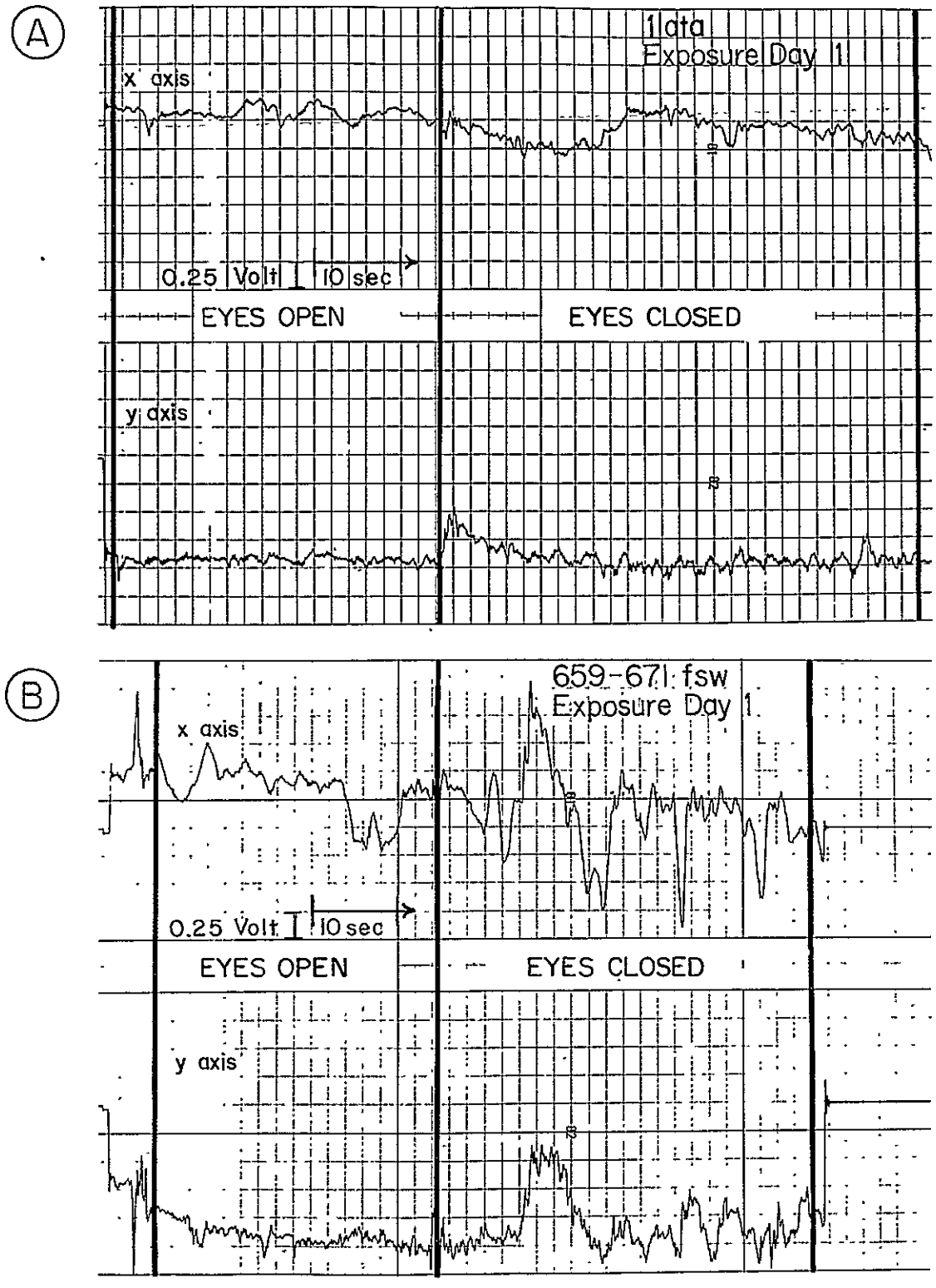


FIG. 5. Effect of compression on balance. Sway recordings obtained on subject LJ. A, at 0 fsw; B, during compression to pressures equivalent to 659-671 fsw. Anterior-posterior and lateral sway are indicated as x and y axes, respectively.

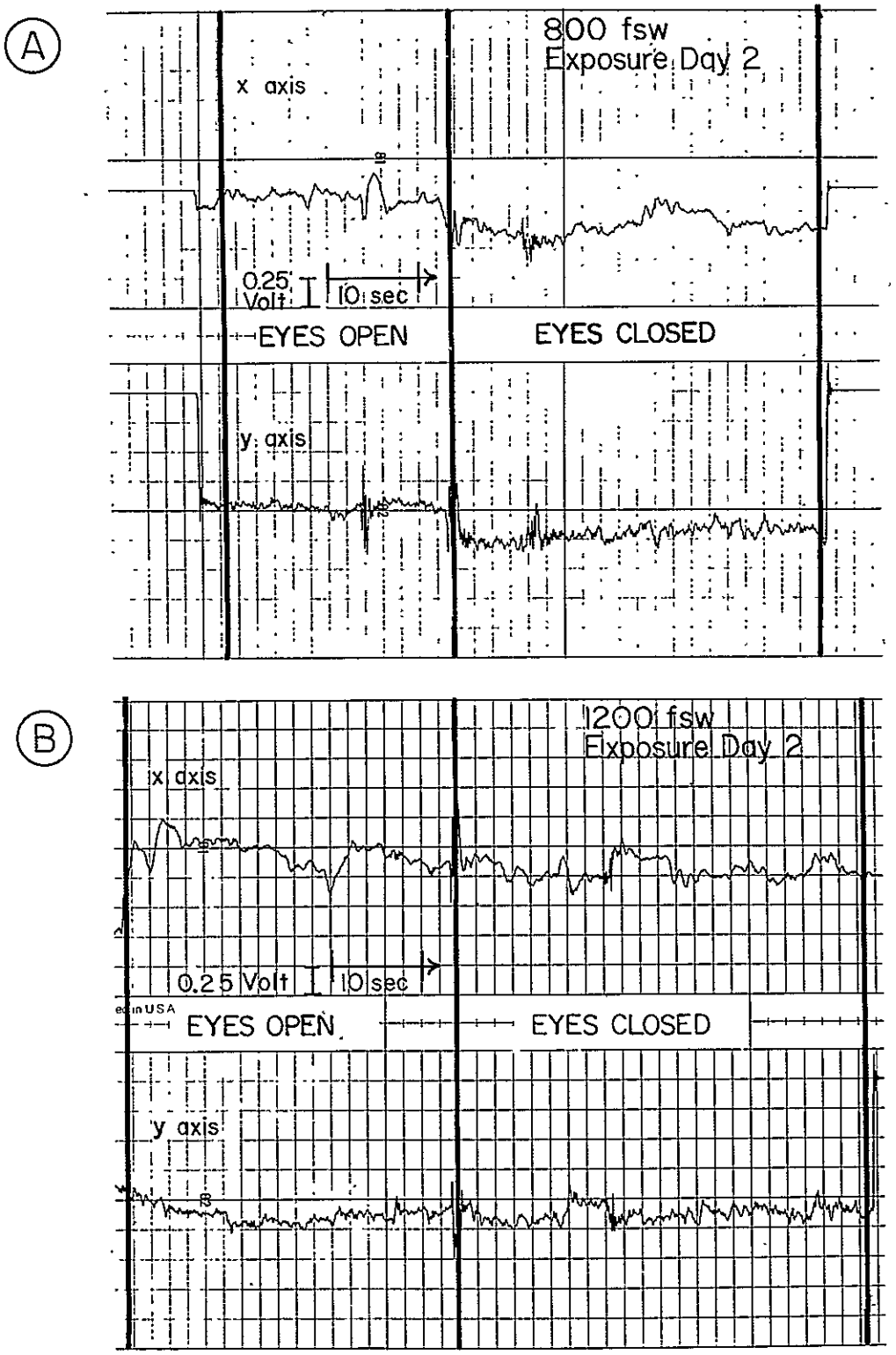


FIG. 6. Effect of compression on balance. Sway recordings obtained on subject LJ at pressures equivalent to: A, 800 fsw and B, 1200 fsw.

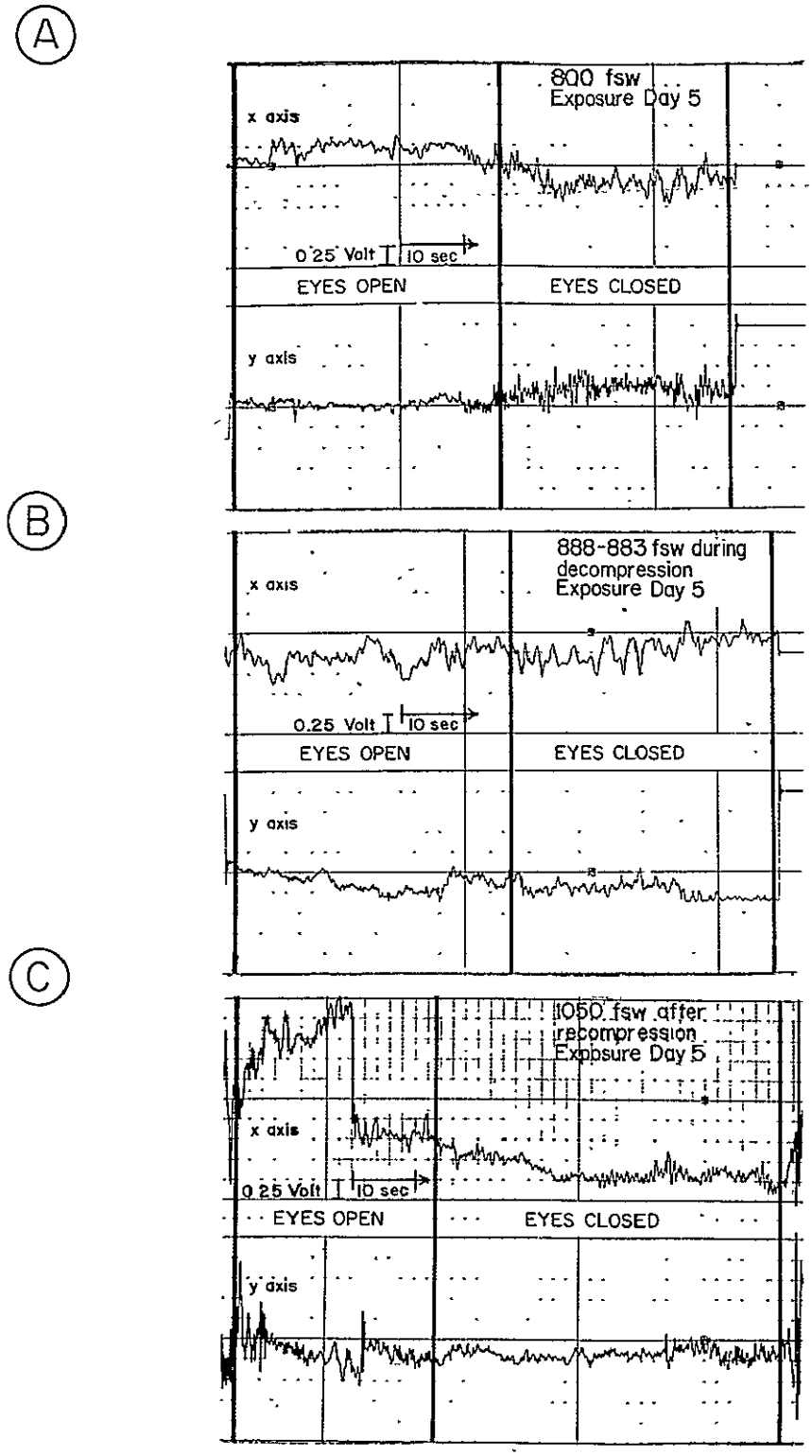


FIG. 7. Effect on balance of vestibular decompression sickness. Sway recordings obtained on subject LJ at pressure equivalent to: A, 800 fsw; B, at 888-883 fsw during decompression from 1200 fsw approximately 1 hour prior to onset of vestibular decompression sickness; and C, at 1050 fsw after recompression and resolution of vestibular decompression sickness.

DECOMPRESSION FROM EXCURSIONS

During decompression from 1200 to 800 fsw in Phase I on his fifth exposure day, subject LJ experienced spinning vertigo (Fig. 1A, Sections G-1 and G-4). The onset of vertigo occurred while LJ was instrumented for measurements, so it was possible to observe spontaneous nystagmus by electronystagmography immediately as symptoms began (Fig. 8A); recording continued throughout the episode. Initially, the nystagmus was right-beating with a slow component velocity of 14°/sec. Five seconds after the onset, the slow component velocity had increased to 35°/sec and reached a maximum of 36°/sec at seven minutes (Fig. 8B). The nystagmus displayed suppression by ocular fixation. Recompression was initiated with the subject reclining. The symptoms resolved quickly with recompression (Sections G-4 and E-7) and the nystagmus reversed to the left, decreasing to 4°/ sec (slow component) before stopping 30 minutes after the initial onset. Following treatment, no abnormality could be detected either by eye tracking or by ocular fixation tests.

Subject MP developed spinning vertigo following decompression from 1600 to 1200 fsw in Phase II. He was not instrumented at the onset of symptoms, but no nystagmus was observed five minutes after recompression to 1400 fsw and symptoms resolved quickly with recompression. These symptoms could not be identified either as true vestibular decompression sickness or as a consequence of efforts to equalize middle ear pressures (Section G-4).

POST-EXPOSURE EXAMINATIONS

Vestibular testing at 1 ata after the compression-pressure exposures were completed included ENG with caloric stimulation, pendulum tracking, and ocular fixation. No change from pre-exposure test results was detected in any of the six subjects.

DISCUSSION

Compression to high pressure in helium-oxygen atmospheres results in such manifestations as increased tremor, ratchety movements of the limbs, spontaneous muscle

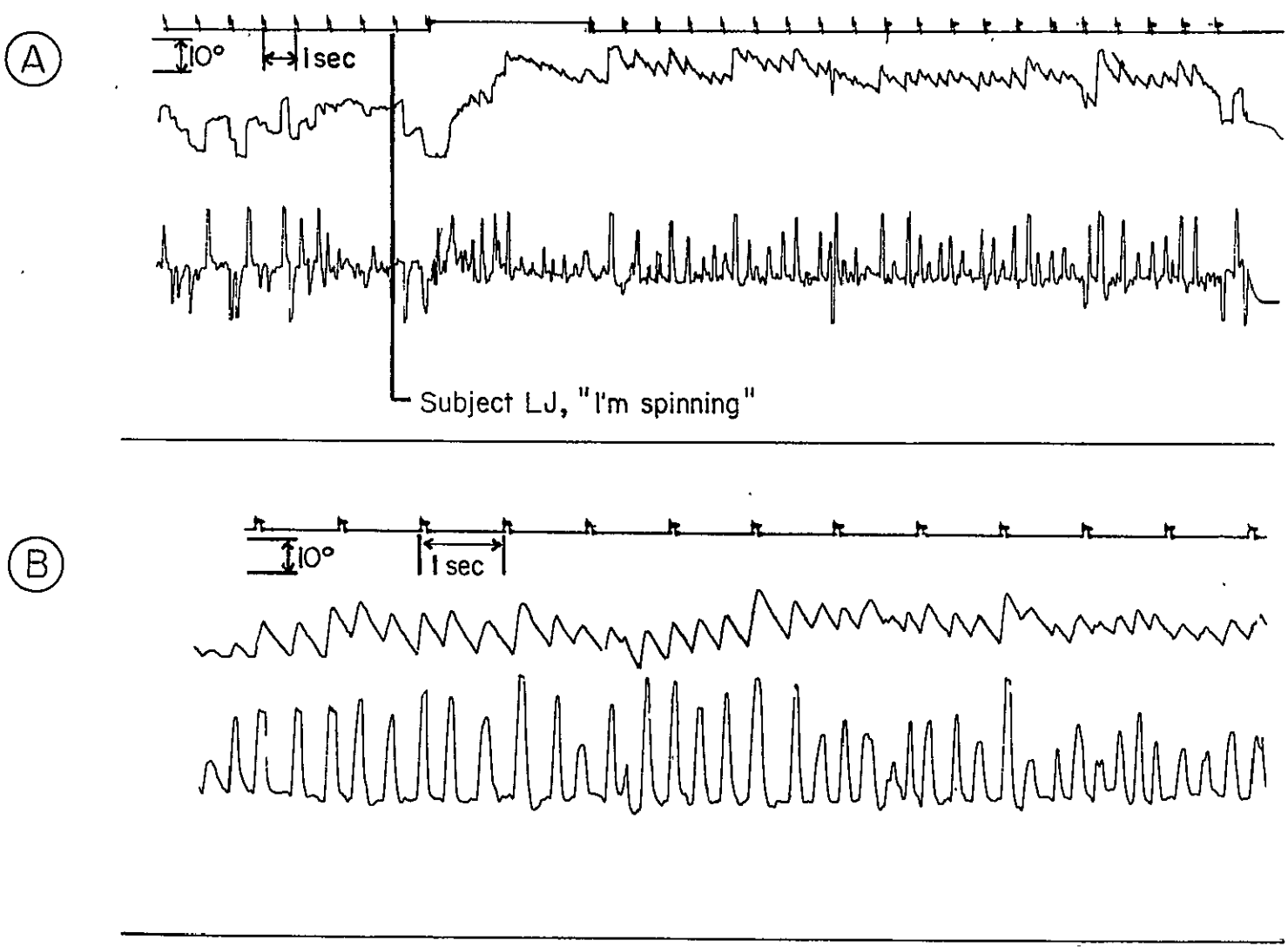


FIG. 8. Nystagmographic recordings associated with vestibular decompression sickness in subject LJ. Upper trace is eye position; lower trace is velocity. Eye movement to the right produces upward deflection. A, Recorded at onset of symptoms; B, recorded seven minutes after onset, during maximum nystagmus.

fasciculations and EEG changes (Section E-1) (4,7,8,13,16, 25,27,29). These signs are usually accompanied by symptoms associated with disturbances of vestibular function, including nausea, drowsiness and dizziness. Since EEG changes have been present in exposures to compression and high pressure, and since the signs of vestibular dysfunction have usually not been present, it has been assumed that the signs and symptoms of compression and pressure are all central nervous system in origin. However, increased sensitivity of the vestibulo-ocular reflex at pressures greater than 41 ata has recently been reported (14), and compression-pressure effects on the vestibular apparatus cannot be ruled out.

Furthermore, the absence of objective signs such as spontaneous nystagmus, alterations in eye pursuit ability and ocular dysmetria is not inconsistent with the presence of symptoms of vestibular dysfunction. As in many disorders, vestibular dysfunction displays a spectrum of symptoms, many in the presence of only minimal signs. This has been demonstrated quantitatively: a symptomatic response to electrical stimulation of the labyrinth at the round window of a human subject could be elicited at an electric current level five to ten times less than the level required to produce objective signs (28). Thus it is possible that even in the absence of visible signs, peripheral effects in the inner ear may contribute to the symptoms of compression and pressure.

HYPERSENSITIVITY OF THE VESTIBULE

With one exception, no directly measurable signs of labyrinthine dysfunction associated with the compression-pressure exposures occurred, even when subjective symptoms were most severe. This exception involved several brief episodes of spontaneous nystagmus (subject FS in Phase II, at 1600 fsw on day 3 [Fig. 3A]) and occurred during a symptom-free excursion.

Although use of the statometer in conjunction with electrical stimulation of the vestibule did not give objective evidence of vestibular hypersensitivity, such hypersensitivity was probably responsible for subjective reports during electrical vestibular stimulation of sensations of rotation (LJ, Fig. 1A), stimulation "too strong"

(FS, Fig. 3A) and feeling dizzy (MP, Fig. 4A). Other symptoms reported by the exercise subjects of spinning, rotation, dizziness, or nausea related to standing (WS, Fig. 2B) or to head movements (GM, Fig. 3B) also suggest such hypersensitivity. All but one of these symptoms occurred during or soon after compressions on the first three days of exposure, at pressures of 500,800,1200 and 1600 fsw. None was reported during excursions from 1200 to 1600 fsw for underwater work at 1600 fsw (Section F) on exposure days 4 (MP, CC) and 9 (FS, GM). Furthermore, the symptoms generally became less severe within several hours of their onset (Section E-1). Thus, any vestibular hypersensitivity (or decreased threshold) appeared to be related principally to compression and to adapt rapidly to to the effects of pressure. Persistent effects of hydrostatic pressure appear to be minimal. This finding is consistent with the absence of vestibular symptoms during very slow compression to pressures of 1000 fsw and beyond in other studies (6,24,34).

These results provide the basis for postulating that rapid compression causes vestibular hypersensitivity or reduces its threshold. The physiological basis for this proposed effect is not known.

POSTURAL SWAY

Increased body sway, a sign of altered postural control, accompanied typical symptoms of vestibular derangement in Predictive Studies IV. Although increases in average sway amplitudes of several hundred percent were commonly observed, even greater changes (up to 1000% in subject LJ following therapy for vestibular decompression sickness, Table I) did not result in gross loss of balance.

Increased postural sway was first observed in the range of 500-800 fsw in all four subjects tested (LJ and FS in Phase I; FS and MP in Phase II). This effect on the body's postural control usually occurred before symptoms of vestibular dysfunction were reported. Balance rail tests have demonstrated altered postural control even earlier than statometer measurements (6).

Increases in sway usually occurred with compression or decompression (Figs. 1A-4A). Recovery was often substantial within several hours of arrival at stable pressures but was not always complete in that time span. Sway values, however, were usually at the 0 fsw control level at the beginning of exposure days 2 and 3. Thus, increased sway and symptoms of vestibular dysfunction were associated principally with compression rather than with pressure per se.

Sway was more affected by compression with eyes open than with eyes closed for subjects LJ (Fig. 1A) and FS (Figs. 2A and 3A). The opposite was true for MP (Fig. 4A). The results for LJ and FS are not necessarily inconsistent with the usual observation that, under normal conditions, body sway is quantitatively greater with eyes closed than with eyes open (10), since measurement periods began soon after each subject rose abruptly from the seated position, and initial measurements were always with eyes open. While disturbances within the visual system could contribute to eyes-open sway, the only changes observed in vision during the compression-pressure exposures were not functionally significant (Section E-8). The small changes observed in visual-evoked response and the stimulation of the visual system by the VER measurements (five to seven minutes before balance measurements in Phase I, five minutes before measurements in Phase II) probably did not affect the sway measurements.

The temporal patterns of altered sway in the x and y axes were usually similar. The relative magnitudes of these effects, however, were often different; greater changes in sway in one axis occurred as frequently as greater changes in sway in the other axis.

The conclusion here that altered postural stability is primarily a nonpersisting function of compression is at variance with data from another study in which the onset of increased sway measured by statometer during relatively slow compression occurred at a simulated depth greater than 1000 fsw (6). Balance function continued to deteriorate during relatively slow compression to the maximum simulated depth of 1600 fsw, with little adaptation during six days at that depth; the increased sway persisted during the saturation-decompression. The discrepancy between the findings of that study (6) and Predictive Studies IV has not been explained.

The statometer tracings showed distinct changes in the patterns of sway during the compression-pressure-time exposures (Figs. 5-7). Alterations in all five of the major characteristics used here to describe sway were observed. The complex nature of the statometer output signals corresponds to the complexity of postural control (11, 18), and a combination of analytical and data reduction methods will be necessary to relate sway patterns to specific functional disturbances (3, 19). The significance of these pattern changes in terms of the nature or loci of the vestibular or CNS disturbances which have caused them is not entirely known. Higher sway frequencies (approximately 2 Hz) are normal and associated with the proprioceptive system (9, 21). Sway frequencies in the range of 0.2-0.3 Hz, sometimes found in normal sway patterns, are more prominent in individuals with various vestibular disturbances and are elicited by caloric stimulation (9,21).

Altered postural control has been associated with numerous other factors in addition to vestibular disorders, some of which may be of direct relevance to this study. Fatigue and severe anxiety have been reported to affect sway (3,5,33), changing the sway amplitude distribution pattern and the low frequency amplitudes, respectively (33). Individual differences are large (2,3,6,21).

Other factors which affect postural control are also relevant to this study. Central nervous system depressants such as diazepam (21), hypoxia (5) and alcohol (15) alter postural stability as do other CNS-active drugs (21,35), and neurological (35) and emotional disorders (33). Nitrogen narcosis at 10 ata of compressed air increases body sway (2), in the absence of any change in the vestibulo-ocular reflex arc (1). Thus, increased body sway can result from disturbances of central as well as of peripheral function, and the increased average sway amplitudes observed in Predictive Studies IV cannot be assumed to result from vestibular hypersensitivity alone.

Altered sway patterns associated with the compression-pressure exposures in Predictive Studies IV included increased "low frequency" sway (Figs. 5 and 6); this is consistent with the symptomatic evidence of vestibular hypersensitivity. Increased amplitude of "high frequency" sway

(Figs. 5 and 6) may reflect increased activity of foot and leg muscles attempting to compensate for the increase in low frequency sway.

The fact that most of the "energy" in the power spectral density of normal sway is concentrated at extremely low frequencies (3,21,31) is important to analysis of statometer output by methods using integration for area analysis. Distortion of these low frequency components must be avoided while at the same time the D.C. component present in the output of direct coupled amplifiers must be eliminated. If low frequency components are progressively attenuated, such as by a low pass electronic analog filter, then relationships between sway measured in experimental situations and sway measured in control situations may be changed:

Let C = control sway

E = experimental sway

then fractional change in sway = $(E - C)/C$

If experimental circumstances introduce high frequency components into E, with C concentrated at low frequencies, analog filtering may reduce C disproportionately more than E. As a result, the ratio (E-C)/C can assume artificially large values. Thus, the results of sway measurements obtained in different laboratories may not be comparable if different methods are used to remove the D.C. component.

SIMILARITIES BETWEEN SYMPTOMS OF COMPRESSION-PRESSURE AND MOTION SICKNESS

Even though the causes of motion sickness and compression-pressure sickness may differ, many symptoms of rapid compression and pressure are also associated with vestibular disturbance induced by motion sickness (12,26,32). Symptoms of acute motion sickness include: epigastric awareness and discomfort, nausea, vomiting or retching; changes in the skin (pallor, flushing, subjective warmth, cold sweating); increased salivation; headache; and dizziness. The victim often desires a cooler environment. Moderate-to-severe drowsiness, to the point where the subject is unable to follow instructions, is common. The latter has been a disturbing aspect of compression to high pressure,

particularly since EEG changes indicating lowered alertness or attentiveness have been observed at the same time.

Great variations in individual susceptibility to motion sickness have been found (17,26). Emotional states, anxiety and fear are probably not factors, but uncomfortable heat and alcohol (even after the concentration in blood returns to zero) may be important as predisposing factors (17). Symptoms of vestibular dysfunction can be elicited or exacerbated by stimuli other than motion, such as decreased circulating blood volume and hypotension (30,32), low CNS P_{O_2} due to low inspired P_{O_2} or decreased brain blood flow (5,32), noise (23), and odors (17). Even if such stimuli are not primary causes of symptoms, they may decrease the threshold to onset.

Many of these factors are also relevant to compression-pressure exposures. Individual variability in sway responses to compression and pressure has been reported (6) and was observed in Predictive Studies IV. Variability in the incidence of symptoms was also observed (Section E-1). Heat was probably a factor (Section E-15) but significant cardiovascular alterations did not occur (Section E-13). Noise is a problem generally present in pressure chamber operations and may have been an exacerbating factor in these studies, in spite of attempts to control its intensity (Sections E-7 and E-9).

The physiological aspects of the etiology and expressions of motion sickness are not fully understood (12). While the primary initiating factor in motion sickness may be disturbances of the inner ear mechanisms (26), effects on central nervous system structures are involved in the generation of symptoms. This is consistent with the conclusion of this study that central nervous system disturbances and vestibular hypersensitivity both may be involved in the generation of symptoms associated with rapid compression to high pressure.

MEASUREMENTS BEFORE AND AFTER VESTIBULAR DECOMPRESSION SICKNESS

Since subject LJ was instrumented for measurements during decompression, electronystagmographic recordings at the onset and resolution of his vestibular decompression

sickness on exposure day 5 were observed (Fig. 8). Prompt recompression therapy rapidly reversed this sign of vestibular dysfunction precipitated by the decompression from 1200 to 800 fsw.

Postural sway was also measured before the onset of LJ's decompression sickness, and after the recompression therapy (Table I and Fig. 1). Sway was near the control level before the excursion started but increased markedly during compression, returning only partly toward the control level during the remainder of the excursion. It is not known whether the abrupt increase in sway during compression and the subsequent onset of decompression sickness are related.

CLINICAL EXAMINATIONS

Repeated exposures of working divers to compression, pressure and decompression may eventually result in otological injury (36). However, the rapid compression rates, high pressures, and decompressions in Predictive Studies IV had no residual effects on otological function as evidenced by comparison of the post-exposure with the pre-exposure examination results.

SUMMARY

Rapid compression from 1 ata to pressures equivalent to 800 and 1200 fsw elicited symptoms typical of vestibular dysfunction such as headache, drowsiness, dizziness, nausea, vomiting and altered states of alertness. These symptoms were usually accompanied by increased body sway and subjective evidence of vestibular hypersensitivity, but not by nystagmus. Eye pursuit was unaffected and ocular dysmetria was not detected. The symptoms, subjective feelings of vestibular hypersensitivity, and alterations in postural control improved within hours and were largely absent by the morning of the second exposure day.

There were few signs and symptoms during excursions from 800 to 1200 fsw on exposure days 2 and 5 in Phase I and during excursions from 1200 to 1600 fsw on days 2 and 3 in Phase II. Excursions for underwater work from 1200 to

1600 fsw on exposure day 5 for two subjects, and on day 9 for the other two subjects were symptom-free.

Acknowledgments

The statometer and tape recorder employed for measurement and recording of the postural sway data were provided by Dr. Arthur Bachrach and Dr. Thomas Berghage of the Naval Medical Research Institute (NMRI). Dr. John Spencer was responsible for the direct recording of statometer measurements.

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E-7. AUDITORY FUNCTION

W.P. Potsic[1], L.D. Lowry[2], R. Overlock[3], R. Gelfand[3] and C.J. Lambertsen[3]

Hearing loss in divers is a serious problem that may occur suddenly or may present itself as a progressive decrease in acuity over many years. The hearing deficit may be conductive, neurosensory or be due to both conductive and neurosensory factors. In professional divers the severity of hearing loss appears to be related to the years of professional activity and, therefore, to the number of exposures to otic barotrauma (13). There is also a high incidence (65%) of tympanic membrane abnormalities in divers with hearing loss, suggesting that barotrauma has taken place whether or not it was of concern to the diver (13).

Since either acute or chronic deficits in auditory acuity may affect diver performance and safety, the understanding and prevention of these otological effects assume increasing importance as diving operations require greater compression rates, depths and durations in undersea and compression chamber exposure. Dizziness, vertigo and a progressive decline in the auditory-evoked cortical response have occurred following rapid compression to depths greater than 500 fsw (9). This suggests that otoneurological derangement may be induced by rapid compression, exposure to

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extreme hydrostatic pressure, or both. If actual injury is induced it can cause a neurosensory hearing deficit that is either transient or permanent.

In the present study, to investigate compression and hydrostatic effects on auditory function, audiological and otolaryngological examinations were performed on four subjects before and after rapid compression to saturation exposure at a stable pressure equivalent to 800 fsw, with repetitive excursions to 1200 fsw (Phase I). Similar examinations were conducted on two of the same and two additional subjects before and after saturation exposure at 1200 fsw with repeated excursions to 1600 fsw (Phase II). Audiometry was also performed at increased ambient pressures in three of these subjects within 30 to 40 minutes after rapid compression from 1 ata to the pressure equivalent of 800 fsw and from 800 to 1200 fsw. Two subjects were tested after rapid compression from 1200 to 1600 fsw. In addition, tests were done on two subjects in conjunction with recompression therapy of decompression sickness associated with symptoms of inner ear disturbance.

METHODS

PRE- AND POST-EXPOSURE EVALUATIONS

The pre- and post-exposure otolaryngological examinations consisted of detailed history and physical examination including laryngoscopy, nasopharyngoscopy and pneumatic otoscopy. Audiological examination included pure tone audiometry by air and bone conduction, speech reception thresholds and speech discrimination. Electronystagmography was also performed.

AUDIOMETRY AT INCREASED AMBIENT PRESSURES

Auditory thresholds at increased ambient pressures were measured by bone conduction rather than with air conduction audiometry, since auditory sensitivity is known to be decreased at high pressure by gas density-related, reversible conductive hearing impairment (4,5,12). Use of earphones as the sound source is complicated by influences

of increased gas densities upon the earphones themselves (5). Bone conduction sensitivity is unaffected by the physical properties of the ambient gas, since the skull vibrator is an electromechanical device which is stable at pressure and does not require extensive recalibration with each pressure change (11). If hydrostatic pressure were to produce auditory changes, they were expected to be neurosensory due to neurophysiological dysfunction, rather than conductive. Such changes could not be unequivocally detected by air conduction audiometry.

Bone audiometry is relatively free of contamination from variations in force of application when the force is substantial (2). However, the method of audiometry used does have reduced sensitivity since a bone vibrator which was calibrated to the mastoid (1) was applied to the middle of the forehead (the measurements were made with an audiometer [Maico model H-1] with a skull vibrator [Maico model C] calibrated to an artificial mastoid [B + K model 4152]). Sensitivity was also decreased by the masking effect of some residual noise in the chamber (3,10).

The forehead position was selected for ease of application by the subject and good test/retest reliability. The subject held the skull vibrator against his forehead, and bone conduction thresholds were determined by the method of ascending limits (7) at frequencies of 250, 500, 1000, 2000 and 4000 Hz. The chamber sound level was reduced during audiometry by turning off all noise-generating devices in the chamber, and an external sound-attenuating intercommunications headset (David Clark Co. type 10 B-A) was worn, further reducing ambient noise contamination to about 35-50 dB. Voice communication also was stopped, the subject indicating hearing the tone by raising his index finger.

RESULTS

PRE-EXPOSURE EVALUATIONS

None of the subjects, all of whom were professional offshore divers, had a history of subjective auditory deficit or vestibular symptoms which would suggest past otoneurological injury. The physical examinations of all

subjects showed no abnormalities. The appearance of the tympanic membrane, and mobility tested by pneumatic otoscopy, were both normal. Electronystagmography on all subjects showed appropriate responses to caloric stimulation.

Hearing deficits were found in the pre-exposure audiometric examinations. Only one subject (CC) had a completely normal audiogram. The five remaining divers had mild high tone neurosensory hearing losses above 4000 Hz. Subject GM also demonstrated a neurosensory impairment at 250 and 500 Hz (Fig. 1), while LJ had a unilateral, moderate-to-severe neurosensory impairment at 6000-8000 Hz (Fig. 2A).

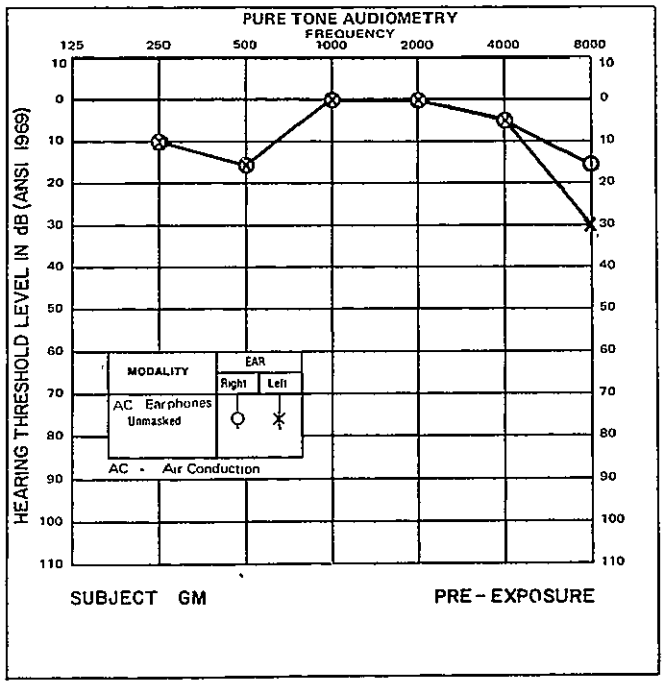

FIG. 1. Pre-exposure air conduction audiogram of subject GM obtained as part of comprehensive medical examinations.

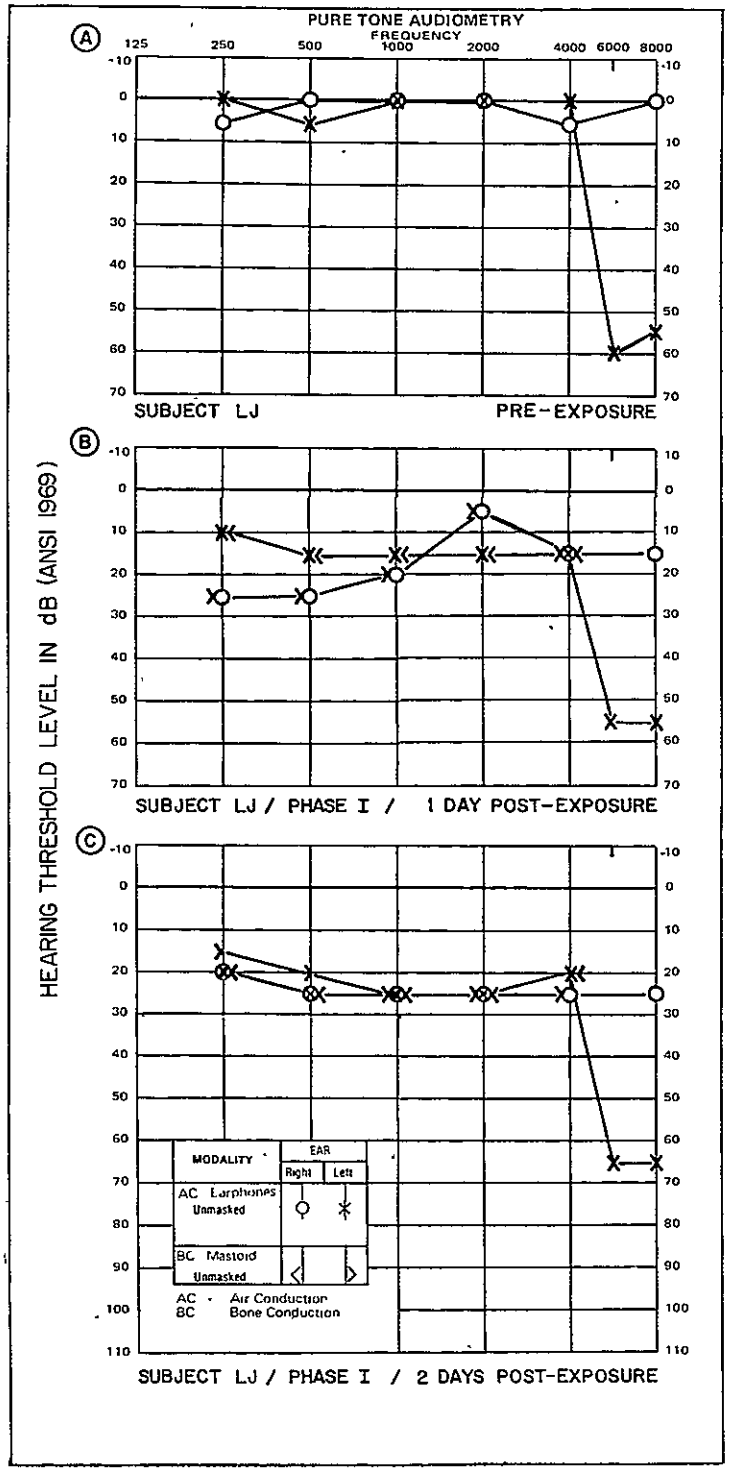

FIG. 2. Audiograms of subject LJ. A, Pre-exposure air conduction audiogram. B, Post-exposure air and bone conduction audiogram of subject LJ, one day after completion of decompression from the Phase I pressure exposure. C, Same as B, one day later.

AUDITORY FUNCTION AT INCREASED AMBIENT PRESSURES

No deficits in auditory acuity were detected in the three subjects who performed the audiometric measurements. No subjects manifested any auditory symptoms in either Phase I or II.

Audiometric data obtained during the pressure exposures of Phases I and II are summarized in Tables I-III for subjects LJ, MP and FS. Fluctuations of 5 or 10 dB are seen in sound-isolated test booths and may be compounded by other factors such as the subject's general physical condition and motivation (6). In the chamber environment, even larger individual variation can be expected but this would not indicate real changes in auditory sensitivity if not consistently present.

AUDIOMETRY DURING TREATMENT OF VESTIBULAR DECOMPRESSION SICKNESS

Near the end of the 800-1200-800 fsw excursion-decompression in Phase I (on his fifth exposure day), subject LJ experienced vestibular decompression sickness and was rapidly recompressed to 1050 fsw (Sections G-1 and G-4). Audiometry by bone conduction from the forehead and the right and left mastoid about 1 hour after the onset of symptoms demonstrated no abnormality (Table I).

Subject MP had vestibular decompression sickness in Phase II on his fifth exposure day 58 minutes after decompressing to 1200 fsw from an excursion to 1600 fsw. Approximately 5 minutes later, after therapeutic recompression to 1400 fsw, no significant change in bone conduction audiometry was detected (Table II, Sections G-1 and G-4).

POST-EXPOSURE EVALUATIONS

Post-exposure audiometry on the day following decompression demonstrated mild threshold variations in all subjects, possibly related to fatigue. This was most prominent in LJ (Fig. 2B) who showed further variation the following day (Fig. 2C). Audiometry one month following exposure did not significantly differ from pre-exposure

TABLE I. Effect on Auditory Thresholds of Exposure to Pressure (Subject LJ, Phase I)

Exposure Day	Depth (fsw)	Auditory Threshold (dB) 250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz
1	0	45	40	40	25	15
	800	35	40	40	25	20
	800	40	40	40	35	25
	800	35	30	35	35	30
2	800	35	40	35	35	25
	1200	35	40	35	35	30
	800	35	35	30	25	15
5	800	40	35	35	35	20
	1200	40	30	40	35	25
5[a]	Forehead	35	40	40	30	40
	R. Mastoid	30	30	30	25	15
	L. Mastoid	35	30	30	20	10
Post-exp.	0	40	35	30	25	15

[a]Measurements made one hour following recompression to 1050 fsw for decompression sickness therapy.

TABLE II. Effect on Auditory Thresholds of Exposure to Pressure (Subject MP, Phase II)

Exposure Day	Depth (fsw)	Auditory Threshold (dB) 250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz
1	0	25	25	35	15	15
	800	30	30	45	25	20
	800	35	30	35	30	15
	1200	35	40	40	25	20
	1200	35	35	45	30	25
	1200	40	45	45	35	5
2	1200	15	30	35	15	5
	1600	40	45	50	30	15
	1200	30	30	35	15	0
3	1200	25	20	40	30	10
	1600	25	35	30	30	15
	1200	30	30	30	20	15
5[a]	Forehead	25	30	40	25	10
	R. Mastoid	15	15	30	20	15
	L. Mastoid	20	20	35	20	15
	Forehead	30	30	50	40	10
	R. Mastoid	20	10	30	35	20
	L. Mastoid	25	15	35	35	10
8	1200	30	35	40	25	15
Post-exp.	0	30	20	35	20	20

[a]Two consecutive measurements started approximately five minutes after recompression to 1400 fsw for decompression sickness therapy.

TABLE III. Effect on Auditory Thresholds of Exposure to Pressure (Subject FS, Phases I and II)

Exposure Day	Depth (fsw)	Auditory Threshold (dB) 250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz
Phase I						
1	0	35	30	25	25	25
	800	35	35	50	30	30
	800	35	45	45	35	30
2	800	35	35	30	25	20
	800	40	30	40	35	30
Post-exp.	0	55	40	35	35	30
Phase II						
1	0	30	30	35	30	20
	800	35	35	45	30	25
	800	-	-	-	-	-
	1200	25	35	35	30	15
	1200	35	35	35	25	20
	1200	30	35	40	30	20
2	1200	40	35	35	30	15
	1600	35	30	35	35	20
	1200	30	30	45	30	15
3	1200	40	35	35	35	15
	1600	35	35	30	30	15
	1200	40	40	35	30	10
8	1200	35	35	35	30	15
Post-exp.	0	30	35	30	35	25

audiometry in any of the subjects. Repetition of the complete pre-dive otolaryngological and audiological examinations revealed no detectable changes from pre-exposure results.

DISCUSSION

The possibility that rapid compression and exposure to extreme hydrostatic pressures could cause a neurosensory hearing deficit was taken into account in planning the Predictive Study because of the association of these conditions with otoneurological symptoms and a progressive decline in the auditory-evoked response with increased pressure (9). In agreement with previous findings all subjects in Predictive Studies IV experienced nausea and dizziness in varying degrees (transient slight dizziness to vomiting) (Section E-1), and four subjects tested showed some evidence of postural disequilibrium (Section E-6) during rapid compression and exposure to depths of 800, 1200 and 1600 fsw. However, transient otological signs were perceived only once (Section E-6) and no hearing impairment by bone conduction audiometry could be detected at any depth or rate of compression studied.

These results with a bone conduction method are consistent with the concept that changes in auditory acuity measured at increased ambient pressures by air conduction audiometry are reversible, conductive hearing impairments caused by alterations in gas density and in the acoustic properties of compressed gases, and that they are not neurosensory in origin (8).

The occurrence of vestibular decompression sickness in two subjects at a time when it was possible to perform controlled audiometry in the pressure chamber provided an opportunity to examine auditory function during the initial treatment of this condition. No temporary threshold shift or permanent hearing impairment was detected. These results, along with the rapid resolution of nystagmus and vertigo, confirmed the effectiveness of immediate recompression therapy of vestibular dysbarism (Section G-4).

CONCLUSIONS

Rapid compression to high pressures evoked symptoms of dizziness and nausea which may have been related to oto-neurological effects. No otological signs and no hearing impairment were found in any of the six subjects. Auditory function tested soon after the onset of and rapid recompression therapy for vestibular decompression sickness in two subjects revealed no shifts in auditory threshold. Immediate post-decompression tests showed mild auditory threshold variations in all subjects, evidently due to fatigue. Subsequent examinations revealed no audiological, vestibular or otolaryngological changes from pre-exposure control measurements.

Acknowledgment

The authors are especially gratified for the direct advice and assistance provided by Dr. J.C. Farmer, Professor of Otolaryngology, Duke University, in planning this study of hearing in compression.

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E-8. VISUAL FUNCTION

D.J. Montabana[1] and C.J. Lambertsen[1]

Studies of visual function during prolonged exposures to simulated depths over the range of 0 to 1200 fsw have not reported inert gas or hydrostatic pressure effects on vision (2-4). However, exposures in which vision has been studied have involved slow rates of compression. It was considered in planning the present study that the neural influences produced by rapid compression might have a demonstrable effect on the visual system. The present studies provided opportunities for both subjective and objective evaluation of visual function during exposure to hydrostatic pressures of 0-800-1200-1600 fsw and at rates generally faster than previously employed in compressions to the higher pressures. For this reason, repeated measurements of visual function were made both during stages of compression and during the protracted exposures at stable elevated pressures. Of the four visual function tests which were used, two (measuring central visual acuity and accommodation) were self-administered by the supervised subjects at saturation pressures and during the stable pressures at the extremes of transient excursions. Two (measurement of color discrimination and peripheral visual fields) were administered only at saturation pressures on days when transient compressions did not occur. These four measures of visual integrity provided useful indicators of specialized central nervous system function and are relevant to performance in diving operations.

METHODS

The specific tests selected for use in compression states were in addition to the comprehensive clinical

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ophthalmological evaluations of visual function performed before and following the pressure exposures.

EXAMINATIONS PRIOR TO COMPRESSION

Prior to the start of the study all subjects underwent an ophthalmological examination which included measures of muscle balance, ocular pressure, applanation tonometry and refraction, as well as slit lamp and full external examinations. All were found to be normal both clinically and perceptually.

MEASUREMENTS DURING PRESSURE

In addition to the clinical examinations, psychophysical measures of visual function provided baseline controls prior to compression and were performed during actual exposures to pressure.

CENTRAL ACUITY

Three copies of the chart were used, each having one of the three test columns uncovered. There were six possible test instructions (the order of reading letters or numerals), providing eighteen different tests to minimize complications due to learning.

A general instruction required the subjects to read and record the finest possible set of letters or numerals first, followed by the next coarsest acuity level. This procedure assured that a score would not be lost if a subject made all incorrect responses on a single set. The test chart was located on a modified MacBeth illuminator stand as shown in Fig. 1. The subject was seated in front of the chart with his eye position maintained at a fixed distance of 35.3 cm from the chart surface by means of a restraining device which rested on the bridge of his nose and cheekbones. Chamber lights were dimmed and the chart was viewed under the illumination of the MacBeth source which provided 0.95 log lux illuminance (the source was that of Illuminant C which is approximately equivalent

to average daylight, having a color-correlated temperature of 6750° K). Measurements were taken monocularly, first for the right eye (left eye patched), and then for the left eye (right eye patched), since results of binocular measurements would represent only the better of the two eyes. The patch was translucent to prevent changes in the subjects' level of light adaptation.

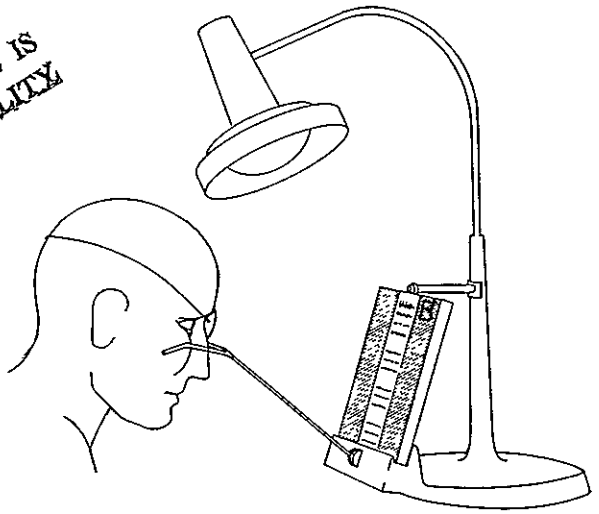

FIG. 1. Drawing of the modified MacBeth illuminator stand used for acuity measurements. The head restraining device ensures accurate placement of the subject's eyes at a fixed distance from the test chart.

ACCOMMODATION

A modified Adler Near-Point Rule (6) was used to measure accommodative ability by determining the closest distance at which the subject could accommodate. A fine-letter target (4 x 4 array of letters) attached to a millimeter rule was initially positioned to be in sharp focus; it was then slowly moved toward the eye (with the rule angled inward toward midline to avoid the problem of convergence of the eyes) while the subject was actively accommodating to the decreasing distance until blurring began at the

near-point. Accommodative near-points were measured separately for each eye, recording a total of five readings per eye. All trials were performed with the chamber lights on and with the eye not being tested, occluded with a translucent eye patch. Baseline measurements were obtained for each subject several days prior to compression. Following one-half hour of practice during which no scores were taken, 20 or more trials over a two-day period were scored to establish control values.

COLOR VISION

Three different measures of color vision were obtained. The Farnsworth-Munsell 100-Hue Test of Color Discrimination (1) served as a preliminary comprehensive screening examination for color deficiency. All subjects scored normally in this test. The two tests of color vision administered during the actual pressure exposures were the Farnsworth Dichotomous Test (D-15 Panel) and the Farnsworth Tritanopic Test Plate. Both were presented monocularly to each eye under the MacBeth light source at the same illuminance level employed for the acuity test.

PERIMETRY

Peripheral visual field measurements were made monocularly for each eye with one subject (acting to perform the measurement) presenting the stimuli in a fully darkened chamber. Perimetric fields were plotted every 30^{o} on a Rodenstock Projection Perimeter employing the 1.12 mm test spot (13' visual angle). Field luminance of the hemisphere was fixed at 0.50 log mL while test luminance was maintained at 2.0 log mL. To assure that proper fixation was maintained throughout each test, the subject was continuously monitored by the subject-experimenter with the viewing device built into the perimeter. The stimulus was presented randomly in any one of 12 field locations, with each presentation being from the "not seen" to the "seen" mode.

TEST CIRCUMSTANCES

For Phases I and II of the overall study program, the measurements of acuity and accommodation were performed on

only the rest subjects (Phase I: subjects LJ and FS; Phase II: subjects MP and FS) just before compressions and during maximum pressures of the excursions. Periods of one and one-half minutes were allotted for measurements, first of visual acuity and then visual accommodation. These measurements were also made at one atmosphere, before and after the pressure exposures. Additional measurements were made in Phase II at the stable saturation pressure of 1200 fsw.

Color vision and peripheral visual field measurements were made on all subjects in both Phases I and II, at one atmosphere before and after the exposures, and at the saturation pressures.

SUBJECT TRAINING

Each subject was trained to measure and record acuity, accommodation and color vision. During four separate training sessions, the tasks were repeated until the test package could be completed efficiently and with reproducible results within the allotted three minutes. The subjects were trained in pairs for the perimetry measurements, with each member of each team acting as subject and experimenter until consistent levels of performance were reached.

RESULTS

At no time in either the active compression or the stable periods at increased pressure during Phase I or II were visual manifestations such as scintillations or other subjective phenomena reported by any of the six subjects.

The data obtained from all subjects in both phases are summarized in Tables I, II and III.

VISUAL ACUITY

Phase I (0-800-1200), subject LJ (Fig. 2A). Visual acuity showed virtually no change from pre-compression control levels either during the stable pressure periods on the first compression day or on succeeding days. The only exception was an apparent change in the left eye from 20/20 to approximately 20/25 while at the 1200 fsw highest excursion

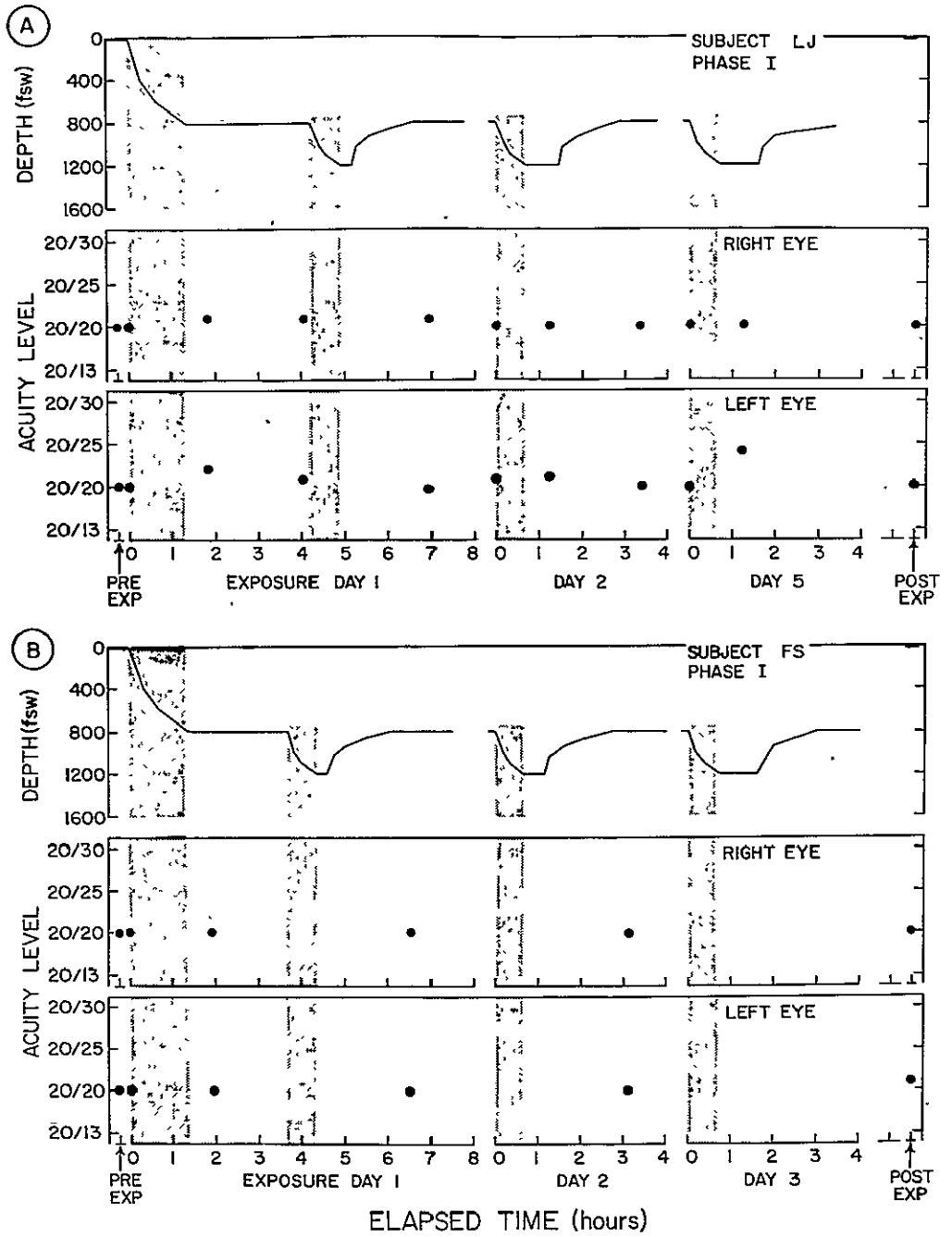

FIG. 2. Visual function (acuity) during exposure to pressures equivalent to 800 and 1200 fsw. Acuity level is given in standard Snellen numbers. (Phase I; A, subject LJ; B, subject FS.)

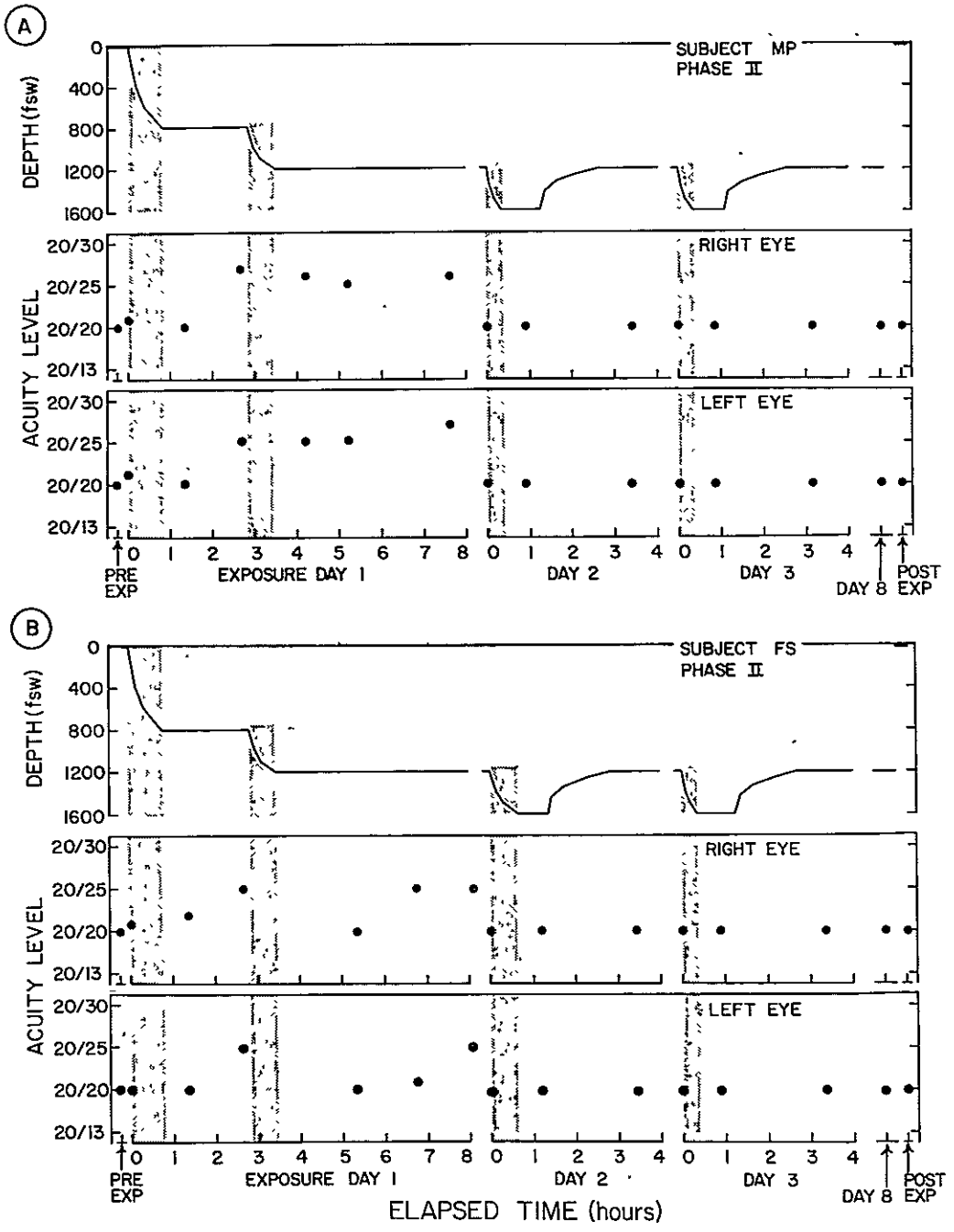

FIG. 3. Visual function (acuity) during exposure to pressures equivalent to 800, 1200 and 1600 fsw. Acuity level is given in standard Snellen numbers. (Phase II; A, subject MP; B, subject FS.)

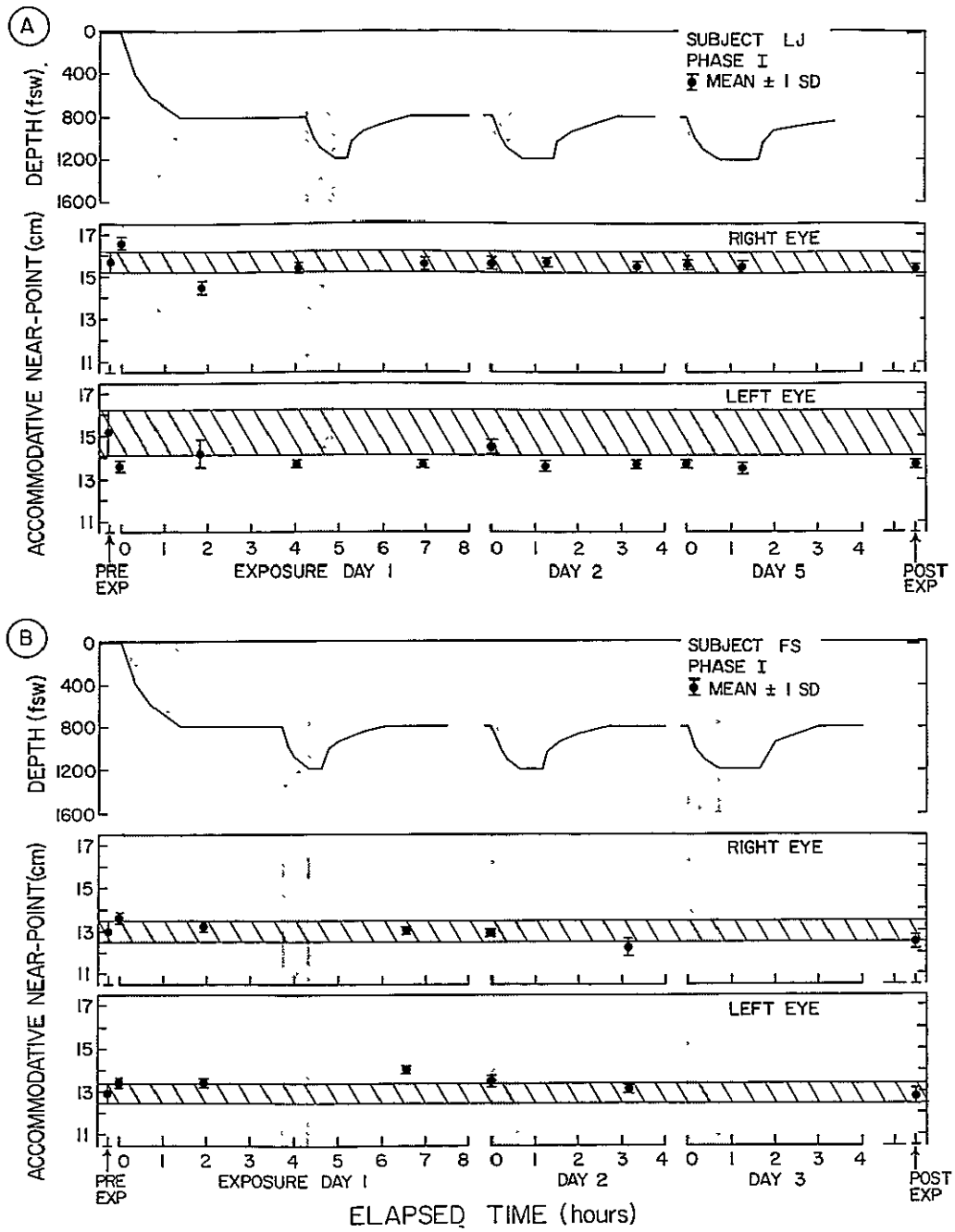

FIG. 4. Visual function (accommodation) during exposure to pressures equivalent to 800 and 1200 fsw. Near-point measurements are the average of five trials. (Phase I; A, subject LJ; B, subject FS.)

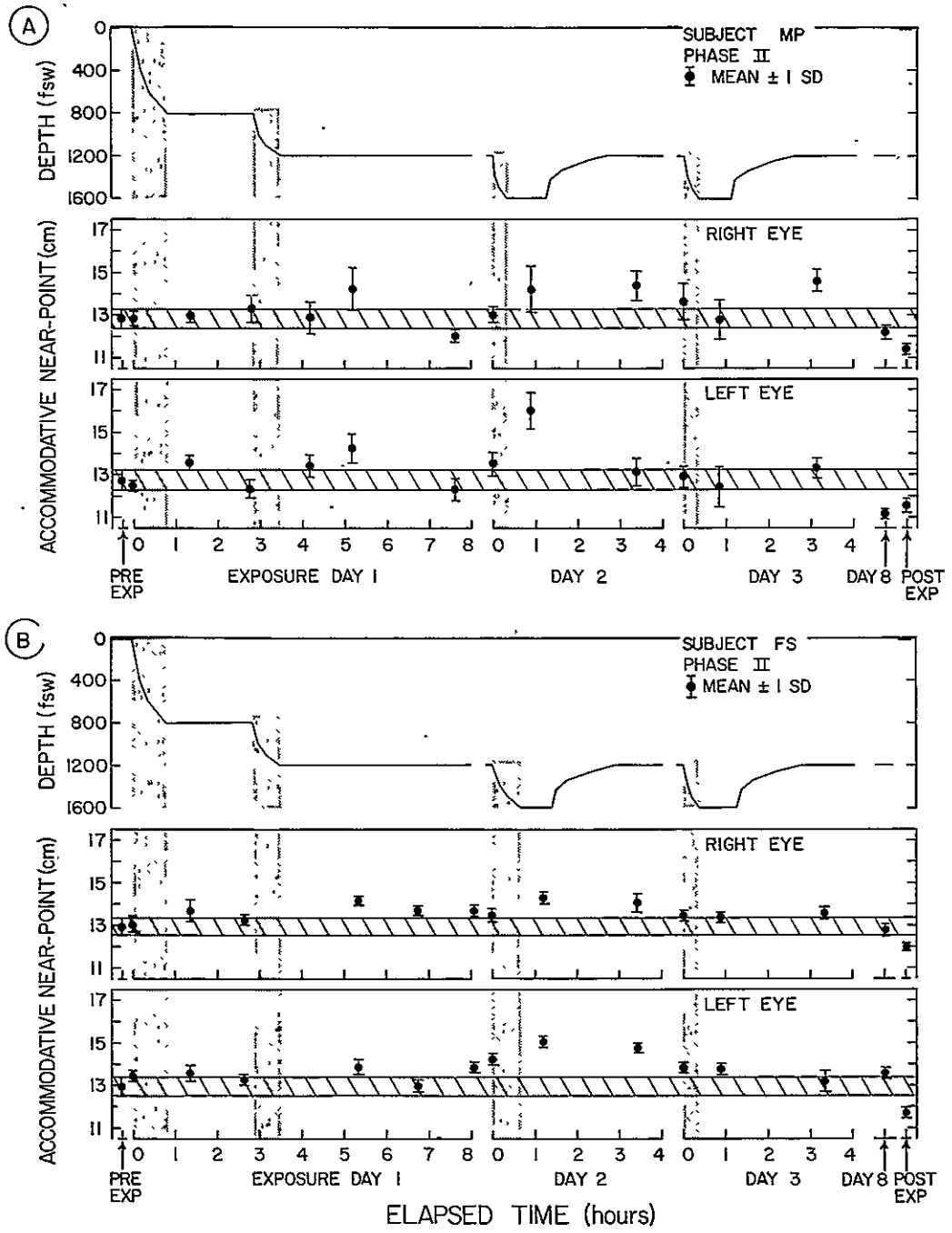

FIG. 5. Visual function (accommodation) during exposure to pressures equivalent to 800; 1200 and 1600 fsw. Near-point measurements are the average of five trials. (Phase II; A, subject MP; B, subject FS.)

TABLE I. Visual Function Tests During Exposure to Pressure (Subject LJ, Phase I)

Exposure Day	Depth (fsw)	Acuity		Accommodation			
				Right Eye		Left Eye	
		Right Eye	Left Eye	Mean	SD	Mean	SD
Pre-exp. baseline	0	20/20	20/20	Mean SD N	15.7 0.5 30	Mean SD N	15.2 1.0 25
1	0	20/20	20/20 [a]	16.6	0.22	13.6	0.20
	800	20/20-1	20/20-2	14.5	0.27	14.2	0.54
	800	20/20-1	20/20-1	15.4	0.20	13.7	0.10
	800	20/20-1	20/20	15.6	0.26	13.7	0.10
2	800	20/20	20/20-1	15.6	0.26	14.5	0.23
	1200	20/20	20/20-1	15.7	0.10	13.6	0.16
	800	20/20	20/20	15.5	0.20	13.7	0.09
5	800	20/20	20/20	15.6	0.13	13.7	0.10
	1200	20/20	20/20-4	15.5	0.24	13.5	0.20
Post-exp.	0	20/20	20/20	15.4	0.16	13.7	0.10

[a] The number following the standard Snellen figure indicates the number of errors made by the subject.

TABLE II. Visual Function Tests During Exposure to Pressure (Subject MP, Phase II)

Exposure Day	Depth (fsw)	Acuity		Accommodation			
				Right Eye		Left Eye	
		Right Eye	Left Eye	Mean	SD	Mean	SD
Pre-exp. baseline	0	20/20	20/20	Mean SD N	12.8 0.4 20	Mean SD N	12.6 0.6 20
1	0	20/20-1	20/20-1	12.8	0.36	12.4	0.16
	800	20/20	20/20	13.0	0.25	13.5	0.33
	800	20/25-2	20/25	13.3	0.62	12.3	0.39
	1200	20/25-1	20/25	12.9	0.71	13.4	0.49
	1200	20/25	20/25	14.2	0.93	14.2	0.69
	1200	20/25-1	20/25-2	12.0	0.28	12.3	0.56
2	1200	20/20	20/20	13.0	0.33	13.5	0.52
	1600	20/20	20/20	14.2	1.03	16.0	0.87
	1200	20/20	20/20	14.4	0.63	13.1	0.65
3	1200	20/20	20/20	13.6	0.85	12.9	0.49
	1600	20/20	20/20	12.8	0.90	12.4	0.92
	1200	20/20	20/20	14.6	0.56	13.3	0.48
8	1200	20/20	20/20	12.2	0.29	11.1	0.20
Post-exp.	0	20/20	20/20	11.4	0.17	11.5	0.29

TABLE III. Visual Function Tests During Exposure to Pressure (Subject FS, Phases I and II)

Exposure Day	Depth (fsw)	Acuity		Accommodation			
				Right Eye		Left Eye	
		Right Eye	Left Eye	Mean	SD	Mean	SD
Pre-exp. baseline	0	20/20	20/20	Mean SD N	13.0 0.4 37	Mean SD N	13.0 0.4 36
Phase I							
1	0	20/20	20/20	13.6	0.24	13.4	0.19
	800	20/20	20/20	13.2	0.16	13.4	0.14
	800	20/20	20/20	13.0	0.11	14.0	0.13
2	800	-	-	12.9	0.09	13.5	0.23
	800	20/20	20/20	12.2	0.39	13.1	0.15
Post-exp.	0	20/20	20/20-1	12.5	0.28	12.7	0.38
Phase II							
1	0	20/20-1	20/20	13.1	0.36	13.5	0.19
	800	20/20-2	20/20	13.7	0.49	13.6	0.34
	800	20/25	20/25	13.3	0.22	13.3	0.20
	1200	20/20	20/20	14.2	0.18	13.9	0.32
	1200	20/25	20/20-1	13.7	0.22	13.0	0.26
	1200	20/25	20/25	13.7	0.24	13.9	0.21
2	1200	20/20	20/20	13.5	0.32	14.3	0.19
	1600	20/20	20/20	14.3	0.23	15.1	0.16
	1200	20/20	20/20	14.1	0.37	14.8	0.19
3	1200	20/20	20/20	13.5	0.15	13.9	0.09
	1600	20/20	20/20	13.4	0.18	13.8	0.23
	1200	20/20	20/20	13.6	0.24	13.2	0.45
8	1200	20/20	20/20	12.8	0.26	13.6	0.20
Post-exp.	0	20/20	20/20	12.0	0.04	11.7	0.13

pressure on day 5, which occurred while the subject reported feeling nauseous and lightheaded.

Phase I, subject FS (Fig. 2B). This subject showed no changes in acuity in the relatively few measurements made on him in this phase.

Phase II (0-800-1200-1600, subjects MP and FS (Fig. 3A,B). Both subjects showed a mild decrease in acuity after their compressions from 0 to 800 fsw and from 800 to 1200 fsw. The visual changes were associated with effects such as "mental slowness," "shakiness," "spinning" and fasciculations of thigh and abdominal muscles in subject MP, while subject FS had headache, nausea and coarse tremor (Section E-1). No changes in acuity for either subject were associated with subsequent excursions to 1600 fsw.

VISUAL ACCOMMODATION

Standard deviations (SD) of 20 or more pre-exposure trials were used to establish the regions of variation of controls for this measurement.

Phase I, subjects LJ and FS (Fig. 4A,B). No systematic changes in accommodation were associated with exposure to pressure. Almost all measurements which were outside their control regions deviate by less than 1/2 cm and show closer near-points than the controls.

Phase II, subject MP (Fig. 5A). The standard deviation for the measurement sequences of five trials was larger for this subject than for subjects LJ or FS and was usually larger during the pressure exposures than at sea level. Measurements which were outside the variation established by individual controls occurred at 1200 fsw following compression from the surface, during and after the first 1200-1600 fsw excursion, and at 1200 fsw following decompression from the second excursion. These deviations occurred in the presence of previously described symptoms on the first two exposure days and in the absence of symptoms on the third day (Section E-1).

Phase II, subject FS (Fig. 5B). Although exposure of FS to pressure in Phase II had slightly greater effects on accommodation than he experienced in Phase I (Fig. 4B), the magnitude of these effects was small, Minor deviations from control measurements occurred on each of the first three exposure days with the largest changes found during and after the first 1200-1600 fsw excursion on day 2. These deviations occurred in the presence of more numerous symptoms on day 1 than on days 2 or 3 (Section E-1).

COLOR VISION AND PERIMETRY

Phase I, subjects CC, LJ, FS, WS. Both the D-15 panel and Farnsworth Tritanopic Test Plate measurements were made at 800 fsw on exposure day 4 for CC and LJ and on day 5 for FS and WS. Color vision of all four subjects was completely normal. Visual fields (both eyes, all four subjects) were well within normal limits, both at one atmosphere and at 800 fsw.

Phase II, subjects CC, GM, MP, FS. Measurements made at 1200 fsw on exposure day 4 showed that all four subjects had normal color vision. Visual fields were within normal limits (both eyes, all subjects) both at sea level and at 1200 fsw.

DISCUSSION

The tests employed provided objective information concerning visual function before and just after each stage of rapid compression to high pressure. Although these tests were not done during dynamic stages of compression, the subjects did perform various perceptual, memory, cognitive and psychomotor function tests during compression (Section E-10) which involved the visual system, including reading and eye-hand coordination. There was no indication that visual disturbances per se impeded the performance of these tests. Nor did any of the subjects report visual disturbances of any kind either during rapid compression or at stable pressures equivalent to depths of 800, 1200 and 1600 fsw.

While there were essentially no changes in visual acuity or accommodation throughout Phase I, changes in these

measures were noted in the more rapid pressurizations and the higher pressure of Phase II (Figs. 3 and 5). Changes in acuity observed in Phase II were associated only with compression from the surface, while changes in accommodation occurred during excursion to 1600 fsw and upon return to 1200 fsw. Although many of these changes occurred concomitantly with severe compression or hydrostatic pressure effects which may have indirectly influenced test reliability, some were associated only with minimal symptoms of discomfort. Deviations from control levels were greater in Phase II than in Phase I, coincident with the increased rate of compression, greater saturation pressure and increased severity of symptoms.

Most changes in acuity and accommodation were bilateral. Even minor fluctuations almost always affected both eyes in parallel. Such responses are consistent with direct hydrostatic effects on vision by a common pathway in the central nervous system. However, attentional distractions induced by symptoms could also cause bilateral changes in vision test results.

The observed changes in accommodation (usually less than 2 cm) are considered to be functionally insignificant. While the greatest lengthening (from an average of about 12.5 to 16 cm) was noted to be unusual by subject MP as he recorded it, he commented that he did not sense any change in vision at that time. The greatest change in acuity (from 20/20 to 20/25) is considered to be functionally insignificant.

SUMMARY

No subjective visual disturbances were reported in these exposures to compression and prolonged residence at stable high pressure. Complex tasks involving vision and eye-hand coordination were performed throughout, without interference due to visual difficulties.

Relatively small, transient, and functionally insignificant changes in visual accommodation and acuity were observed in Phase II, but none were observed in Phase I. Most but not all of these changes coincided with the greater severity of discomfort reported by the subjects in Phase II as compared to Phase I.

Color vision and peripheral visual fields were normal at the stable pressures of 800 and 1200 fsw.

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E-9. SPEECH GENERATION AND DISTORTION

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Initiation and articulation of speech involve complex processes, including the highest functions of the central nervous system, transmission of neuroelectric impulses, synaptic transmission and the coordinated actions of respiratory, laryngeal, mouth and facial muscles. Production of the speech signal involves an acoustic source-resonator function. Sources of sound include the vibratory action of the vocal folds as well as constrictions or closures above the level of the larynx. The resonators are the oral, nasal and pharyngeal cavities collectively called the vocal tract. The sound waves produced at or by the various sources interact with the cavities and tissues of the vocal tract and are formed into the sounds of speech by movements of the articulators before transmission.

One of the principal manifestations of compression and exposure to high pressure is increased muscle tremor (Section E-4) (1,16,19), which may be either central or peripheral (neuromuscular) in origin. In planning Predictive Studies IV, it was thought that effects of rapid compression and high hydrostatic pressures on neural and neuromuscular actions might have prominent expressions through interference with the precise functions involved in articulation of speech. Such effects would be different in mechanism from the physical alteration of speech when helium is breathed in place of nitrogen.

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Verbal communications are hindered when the sounds produced by correctly articulated speech are altered in compressed air atmospheres (4,13). Speech intelligibility is even further hindered when a helium-oxygen breathing mixture replaces air in high pressure operations (3,8,12,13, 15,17,18). Dense gases modify reception of sound as well as its generation by degrading the characteristics of the tympanic membrane through loading (18), thereby causing a reversible conductive hearing loss (6). Increased gas density distorts the frequency response and reduces the sensitivity of electroacoustic intercommunications equipment such as microphones, earphones and loudspeakers (11-13).

Modern helium speech processors, while highly useful are only partially effective in restoring the intelligibility of "helium speech" (8,17). Furthermore, when divers or experimental subjects speak to each other within a helium-oxygen filled pressurized compartment or to personnel outside the chamber at 1 ata, the speaker hears a distorted version of his own speech. Therefore an individual exposed to helium-oxygen and high ambient pressure for a prolonged period may attempt to adjust his own articulation to improve his speech intelligibility.

In Predictive Studies IV these several related factors were taken into account. The subjects' speech was recorded, both during operational intercommunications and while reading standard word lists designed to determine speech intelligibility, in order to evaluate acute effects of actual compression in a helium-oxygen environment as well as longer term effects of this exposure.

MATERIALS AND METHODS

OPERATIONAL INTERCOMMUNICATIONS

A multiple input, hands-free "party-line" system (7) was used for operational intercommunications between subjects, operations personnel and investigators. Personnel outside the chambers employed single-muff headsets with boom-mounted dynamic noise-cancelling microphones (Telex model CS-75; frequency response in air at 1 ata: 100-8000 Hz ± 5 dB). During compression the subjects used a dual-muff sound attenuating headphone (David Clark Co. model

10B-A) modified by the installation of a boom-microphone unit from the Telex CS-75. At stable pressures and when not engaged in experiments, the subjects used the lighter weight Telex CS-75 units. While breathing special gas mixtures during decompression from excursions, the subjects used oronasal masks fitted with the Telex microphones.

Speech originating from the subjects was processed by a helium speech processor (Marconi type 023). Speech originating outside the chambers bypassed the speech processor. All operational intercommunications were recorded on magnetic tape (Metrotech model 543) at 15/16 inch/second. Due to a malfunction in the speech processor, its "expansion" (depth) control could not be adjusted to optimize its operation for depths greater than approximately 1000 fsw.

SPEECH INTELLIGIBILITY RECORDING

Apparatus for recording word lists used for evaluation of speech intelligibility included condenser microphones (Bruel and Kjer [B & K] models 4134 [1/2 inch] and 4135 [1/4 inch]) with matching solid-state preamplifiers (B & K models 2618 and 2619). These microphones have smooth, extended frequency responses (in air at 1 ata: model 4134, 4.5 Hz to 20 kHz; model 4135, 4.5 Hz to 100 kHz). A helium-oxygen mixture at pressures up to 31 ata has been found not to change the shape of the frequency response of the model 4134 microphone over the range of 0.2 to 10 kHz (14).

Preamplifier output was connected via a through-bulkhead connector to a power supply and sound level meter (B & K models 2801 and 2203, respectively) and finally to a magnetic tape recorder (General Radio type 1525-A). It was impractical to improvise an acoustic booth enclosing both the subject and microphone to eliminate reverberation as has been described elsewhere (12). An alternative method for reverberation elimination was devised: A foamed plastic container, open at one end and lined at the closed end with a layer of open-cell foam rubber, was arranged to contain the microphone as shown in Fig. 1. With the subject holding the container at arm's length, reverberations were effectively eliminated.

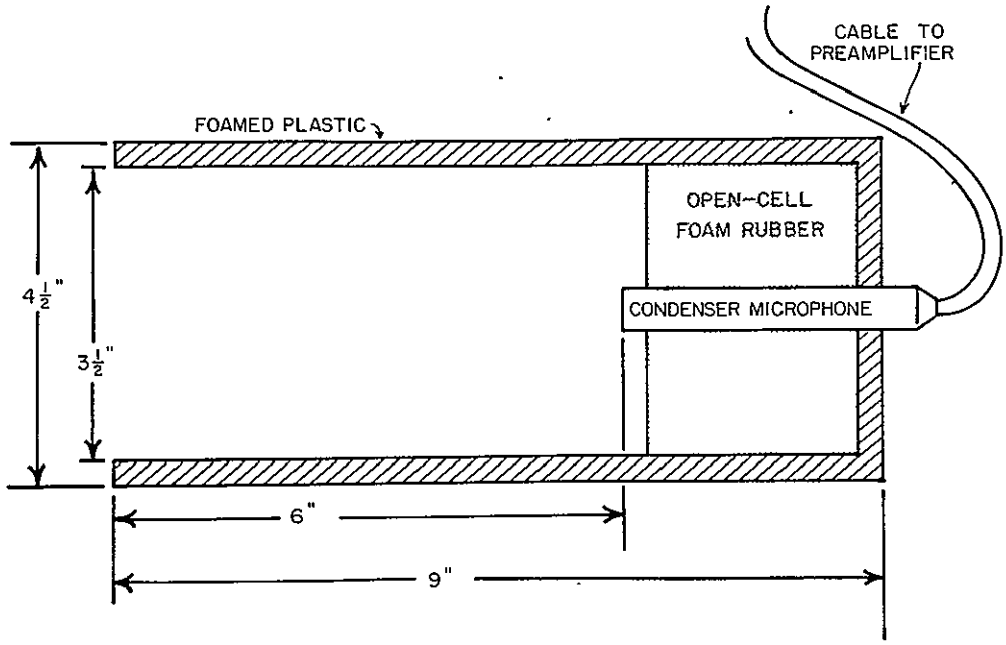

FIG.1. Microphone enclosure of foamed plastic adapted from a standard household insulating container.

EXPERIMENTAL PROCEDURES

Recordings for evaluation of speech intelligibility were made in Phase II with subjects CC, GM, MP and FS. There were two entirely different sets of circumstances during these recording sessions. At 1600 fsw recordings were made under hurried circumstances within 1 hour after 20-minute compressions from 1200 to 1600 fsw and immediately before decompression to 1200 fsw began. These recordings were made on exposure days 2 and 3 for CC and MP, and on day 3 for GM and FS. Other recordings were made in unhurried conditions, and at stable pressures of 1400 fsw (exposure day 11 for GM and FS; day 7 for CC and MP) and 1200 fsw (exposure days 4 and 8 for GM and FS; day 4 for CC and MP). Additional recordings were made at 1000, 860, 690, 560, 392 and 200 fsw during the slow decompression from saturation, and all were in a helium-oxygen breathing mixture except those at 1 ata, which were in air. The model 4134 microphone was used at 1200 and 1600 fsw; subsequently, it was replaced by the model 4135 microphone

for all recordings in helium-oxygen after an electrical failure occurred in the 4134's matching preamplifier.

Each subject was assigned an alternate form of the 50-word Griffiths' Rhyming Minimal Contrasts Test (9,11). Lists A-1, B-1, C-1 and D-1 (Table I) were assigned to

TABLE I. Word Lists Used for Speech Intelligibility Recordings[a]

List A-1	List B-1	List C-1	List D-1
1. SUD	SUM	SUB	SUN
2. FILL	FIG	FIN	FIZZ
3. BUST	JUST	RUST	GUST
4. HIP	RIP	TIP	DIP
5. LED	SHED	RED	WED
6. PEAK	PEAS	PEAL	PEACE
7. DONE	DUD	DUNG	DUB
8. TEN	PEN	DEN	HEN
9. DIG	DIN	DID	DIN
10. BAT	BATCH	BASH	BASS
11. TEETHE	TEER	TEASE	TEEL
12. WE'RE	WEAL	WEAVE	WEED
13. WILL	HILL	KILL	TILL
14. PICK	PIT	PIP	PIG
15. MAT	MAD	MATH	MAN
16. RENT	BENT	WENT	DENT
17. KICK	CHICK	THICK	PICK
18. TOP	HOP	POP	COP
19. BARK	DARK	MARK	LARK
20. ZIP	LIP	NIP	GYP
21. SAD	SAT	SAG	SACK
22. LAWS	LONG	LOG	LODGE
23. NEST	BEST	VEST	REST
24. PIN	SIN	TIN	WIN
25. TAB	TAN	TAM	TANG
26. THEE	DEE	LEE	KNEE
27. SHEEN	SHEAVE	SHEATHE	SHEATH
28. CUFF	CUB	CUT	CUP
29. LASH	LACK	LASS	LAUGH
30. SOLD	COLD	HOLD	TOLD
31. FEEL	REEL	SEAL	ZEAL
32. TOSS	TAJ	TONG	TALKS
33. SING	SIP	SIN	SIT
34. GALE	PALE	TALE	BALE
35. SHAME	GAME	CAME	SAME
36. PUP	PUFF	PUB	PUCK
37. DUMB	DUB	DUTH	DUFF
38. DIG	WIG	BIG	RIG
39. PASS	PATH	PACK	PAD
40. HATH	HASH	HALF	HAVE
41. PEEL	FEEL	EEL	HEEL
42. WIG	WITH	WIT	WITCH
43. VIE	THY	FIE	THIGH
44. FIN	TIN	SHIN	KIN
45. MAT	VAT	THAT	FAT
46. BEIGE	BASE	BAYED	BATHE
47. THIN	TIN	CHIN	SHIN
48. LEAVE	LIEGE	LEACH	LEASH
49. WAY	MAY	GAY	THEY
50. YORE	GORE	WORE	LORE

[a]List A-1, subject CC; list B-1, subject GM; list C-1, subject MP; list D-1, subject FS.

subjects CC, GM, MP and FS, respectively. Prior to the start of the recording sessions, the chamber was silenced by shutting down temperature control and carbon dioxide scrubber blowers and by temporarily discontinuing non-relevant communications. Under these conditions the chamber sound level at 1600 fsw, based on measurements during unmanned compressions for test purposes, was 60-70 dB (A scale). This is similar to the sound level measured elsewhere in a silenced chamber at pressure equivalents up to 1000 fsw (2). The sound level at the condenser microphone was further reduced by the shielding and sound absorption of its enclosure (Fig. 1). Each subject was instructed to count from one to ten while the recording level was adjusted. After stating his name and word list identification, he read the 50-word list, enunciating the phrase "say the word" before each word.

SPEECH INTELLIGIBILITY ANALYSIS

After the compression-pressure-decompression exposures were completed, the unprocessed word list recordings were played back from the tape recorder through the same helium speech processor employed for intercommunications and recorded as processed speech. The processor's expansion control was adjusted both for elimination of self-generated interference and for overall optimum performance at each "depth." The unprocessed and the processed speech recordings were evaluated for intelligibility by panels of listeners.

EVALUATION OF RESPONSES

Tape recordings of the subjects' speech were spliced to allow 3- to 5-second intervals between words; all words in the lists were spliced randomly to eliminate order effects. These tapes were then played to groups of 12-15 semi-trained listeners[3].

[3] University of Florida students were selected on the basis of 1) being native English speakers; 2) having normal hearing; and 3) being capable of performing the required listening tasks.

Before hearing the test tapes, listeners were exposed to a training/test tape which included: 50 words from CID Auditory Word List 3-A (Hirsh recording) recorded in a +10 dB S/N ratio of thermal noise (10); 25 words recorded in a helium-oxygen environment; 25 words from diver communication system recordings; and a 50-word CID Auditory Word List 4-A (10). The final 50 words constituted a screening test; only listeners who attained a score of at least 92% were used.

The listener was asked to write the word he heard as each was presented. The stimuli were the divers' word lists both directly recorded as unprocessed speech and as processed by the unscrambler. Listener responses were scored for the number of words correct; the mean percentage of correct words from each list as scored by the listeners was used as the intelligibility score.

RESULTS

OPERATIONAL INTERCOMMUNICATIONS

Effective operational intercommunications were maintained throughout the compression-pressure exposures, although they were hampered by reduced intelligibility and by a malfunction in the helium speech processor as follows: As the processor's expansion control setting was rotated clockwise, a position was reached at which it "popped" spontaneously and continually at a rapid rate. The control could be set at a lower "depth" setting, with only occasional self-generated interference.

None of the subjects had difficulty in the articulation of speech during the acute exposures of compression and high pressure. Reduced intelligibility appeared to be entirely due to the mechanical distortions resulting from the helium-oxygen-pressure environment.

INTELLIGIBILITY TESTS

The intelligibility scores are shown in Tables II and III and Figs. 2 and 3. Those scores which can be grouped according to depth and gas density are listed in Table II for computation of their means and standard errors

TABLE II. Speech Intelligibility Scores, Simulated Depth and Gas Density Aligned for Depth and Density (N = 4)

Exposure Day[a]	Depth (fsw)	Gas Density[b] (g/l)	Intelligibility Scores (% Correct) Subject CC	GM	MP	FS	Mean	±SEM
Unprocessed Speech								
3	1600	8.6	29.1	22.7	26.9	28.9	26.9	1.5
7;11	1400	7.6	27.1	31.1	32.4	38.1	32.2	2.3
4	1200	6.6	31.5	34.7	28.7	40.6	33.9	2.6
9;13	1000	5.8	25.9	38.7	33.4	42.0	35.0	3.5
10;14	860	5.2	26.9	43.0	26.7	31.1	31.9	3.8
11;15	690	4.3	34.6	42.3	28.6	36.0	35.4	2.8
12,16	560	3.6	29.9	47.7	35.0	38.7	37.8.	3.8
13;17	392	2.6	24.1	46.4	32.0	36.9	34.9	4.7
15;19	200	1.8	37.6	37.9	40.8	39.3	38.9	0.7
0	0	1.1	91.4	93.8	92.4	93.0	92.7	0.5
18;22	0	1.1	97.0	95.5	93.8	90.7	94.3	1.4
Processed Speech								
3	1600	8.6	44.9	40.7	52.3	49.6	46.9	2.6
7;11	1400	7.6	52.5	61.1	56.3	66.0	59.0	2.9
4	1200	6.6	57.3	49.1	56.4	55.6	54.6	1.9
9;13	1000	5.8	64.4	81.3	66.5	77.7	72.5	4.1
10;14	860	5.2	58.6	82.6	47.9	57.7	61.7	7.4
11;15	690	4.3	70.7	85.3	41.9	79.7	69.4	9.7
12;16	560	3.6	72.9	81.4	55.6	77.7	71.9	5.7
13;17	392	2.6	58.9	84.3	62.8	73.9	70.0	5.7
15;19	200	1.8	70.0	68.4	52.4	66.9	64.4	4.1

[a]The microphone enclosure was not used for the pre-exposure recordings at 0 fsw. When two exposure days are shown at the same depth, they refer to subject pairs CC and MP; GM and FS.

[b]Gas density is at 37°C.

TABLE III. Speech Intelligibility Scores Listed by Subject

Exposure Day	Depth (fsw)	Unprocessed Speech Score (% Correct)	Processed Speech Score (% Correct)	Unprocessed Speech Score (% Correct)	Processed Speech Score (% Correct)
		SUBJECT CC		SUBJECT MP	
0	0	91.4	-	92.4	-
2	1600	24.0	50.1	25.6	49.0
3	1600	29.1	44.9	26.9	52.3
4	1200	31.5	57.3	28.7	56.4
7	1400	27.1	52.5	32.4	56.3
9	1000	25.9	64.4	33.4	66.5
10	860	26.9	58.6	26.7	47.9
11	690	34.6	70.7	28.6	41.9
12	560	29.9	72.9	35.0	55.6
13	392	24.1	58.9	32.0	62.8
15	200	37.6	70.0	40.8	52.4
18	0	97.0	-	93.8	-
		SUBJECT GM		SUBJECT FS	
0	0	93.8	-	93.0	-
3	1600	22.7	40.7	28.9	49.6
4	1200	34.7	49.1	40.6	55.6
8	1200	21.7	56.5	24.6	63.5
11	1400	31.1	61.6	38.1	66.0
13	1000	38.7	81.3	42.0	77.7
14	860	43.0	82.6	31.1	57.7
15	690	42.3	85.3	36.0	79.7
16	560	47.7	81.4	38.7	77.7
17	392	46.4	84.3	36.9	73.9
19	200	37.9	68.4	39.3	66.9
22	0	95.5	-	90.7	-

of the mean. Where more than one measurement was made at the same depth but on different days on only one subject pair (at 1200 and 1600 fsw; see Table III), only those values obtained on the same exposure days are used in calculating mean values in Table II.

Mean intelligibility scores were 92.7% in air at 0 fsw before the helium-oxygen exposures began and 94.3% after the exposures were completed (Table II). This difference was consistent with the fact that the microphone enclosure was not used for the pre-exposure tests. A helium-oxygen mixture at 200 fsw reduced mean intelligibility scores of unprocessed speech to 38.9% and of processed speech to 64.4%. Both unprocessed and processed speech mean scores showed an overall decline from 200 to 1600 fsw, with irregular fluctuations (Fig. 2). There was less variability among the individual scores for the unprocessed speech than for the processed speech. Intelligibility scores of unprocessed helium speech obtained from tests elsewhere (12) are also shown in Fig. 2 for comparison with the test results of this Predictive Study.

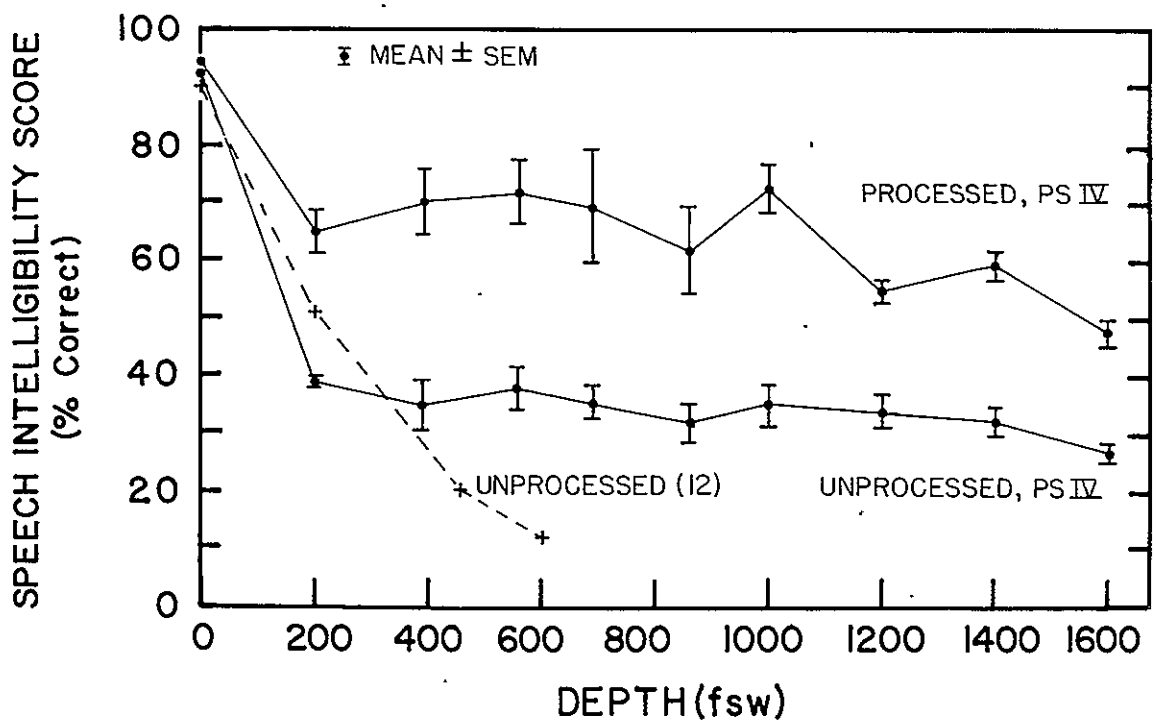

FIG. 2. Mean processed and unprocessed speech intelligibility scores as a function of simulated depth. Unprocessed scores from another study (12) are also shown. Scores at 0 fsw are in air, all others in helium-oxygen.

Individual results from Table III are shown in Fig. 3. Individual differences of the processed speech scores are greatest in the pressure range of the processor's greatest apparent effectiveness (200-1000 fsw; see also Table II and Fig. 2).

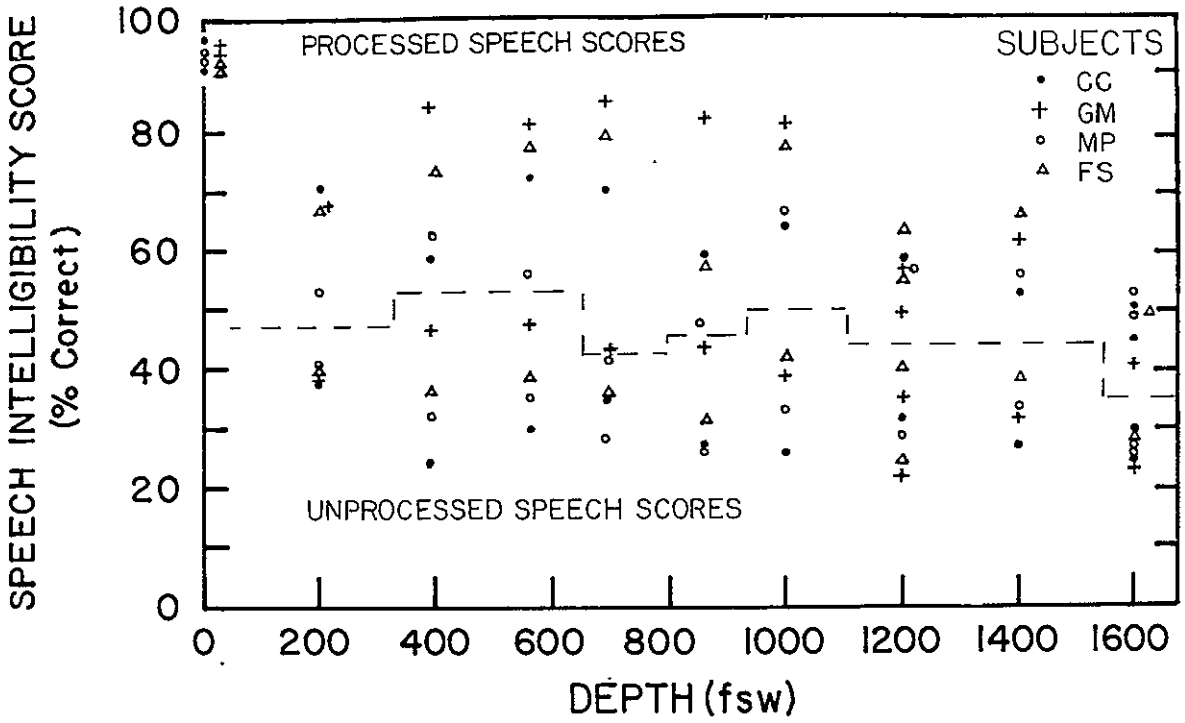

FIG. 3. Individual speech intelligibility scores as a function of simulated depth. Dashed line separates processed from unprocessed scores.

DISCUSSION

There were several reasons for obtaining speech recordings during Predictive Studies IV. They were necessary to evaluate whether acute effects of compression and pressure on the central nervous system or on neuromuscular function might adversely affect the articulation of speech. Furthermore, extended exposure to helium-oxygen environments might cause the individual to alter speech characteristics to compensate for distorted perceptions of his own speech.

Magnetic tape recordings of unprocessed helium speech in a group of four subjects at multiple simulated depths over a range from 0 fsw to the equivalent of 1600 fsw, have not previously been available. In addition to the analyses described in this report, the recordings obtained may be used for other purposes such as for analysis of the structural characteristics of helium speech as a function of helium density, for assistance in design and testing of helium speech processors, and for comparison of helium speech processor capabilities.

OPERATIONAL INTERCOMMUNICATIONS

Interpersonal speech communication originating inside pressure chambers is degraded by the interaction of the gas mixture under high ambient pressure in the vocal tract with the cavity walls and its resultant effect on the resonating output characteristics of the tract (12,13,18). The intelligibility of the speech is also further affected by the characteristics of the microphones used in the pressure chamber and the characteristics of speech reconstitution by electronic processors (8,17). The sound of helium expanding into the pressure chamber during rapid compression caused additional interference with the communications originating from the subjects.

However, the use of silencers inside the chambers to attenuate the sound generated by the expanding gas, the noise cancelling characteristics of the intercommunications microphones, and the relatively wide bandwidth of these microphones at 1 ata made it possible for the helium speech processor to render the subjects' speech sufficiently intelligible for operational and investigative purposes throughout Predictive Studies IV. Furthermore, the subjects had no apparent difficulties with articulation even during the acute stages of compression and exposure to high pressure. Tremulous or wavering speech, or other defects in the formulation of speech (which might have been due to compression or hydrostatic effects on the central nervous system or on neuromuscular control and coordination of muscle systems used in speech) were not observed at any time in either Phase I or Phase II.

INTELLIGIBILITY TESTS

The intelligibility test scores provide quantitative verification of the impressions obtained from listening to the operational speech. No difficulties in articulation were observed in the high quality recordings from the condenser microphone, either following 20-minute compressions to 1600 fsw or during the saturation decompression to 1 ata.

Unprocessed speech scores at 1 ata in air were similar to those of a previous study (12). However, the variation in intelligibility scores of the unprocessed speech as a function of simulated depth in Predictive Studies IV differed remarkably. Intelligibility scores in Predictive Studies IV decreased only slightly with increased pressure, in marked contrast to the earlier results (Fig. 2). This difference was probably due to the use in this study of extremely wide-bandwidth condenser microphones, the frequency response of which remained uniform well beyond the range of conventional microphones throughout the exposure pressure range (14). The beneficial effect of improved microphone characteristics on helium speech processor performance has previously been reported (8,17).

The small decrement of unprocessed speech intelligibility with increasing simulated depth, as evaluated from the recordings made with the condenser microphone, contrasts with the general past experience of listening to intercommunications speech at progressively greater depths. The latter situation resembles the data of Hollien, Thompson and Cannon (12) obtained with a conventional high quality dynamic microphone. However, both helium and pressure cause the frequency content of speech to rise (4,13,18) while increased pressure (density) progressively lowers the bandwidth of microphones (12-14). Only microphones which have exceptionally broad bandwidth at 1 ata and which are relatively insensitive to pressure are likely not to cause the familiar progressive degradation of speech intelligibility. Aside from this influence on the microphone system, increasing pressure itself does not appear to produce progressive degradation of speech characteristics (Fig. 2). This is consistent with theoretical computations of the effect of increased ambient pressure on voiced sounds; it was predicted to be unimportant at pressures greater than about 10 ata (4,5).

This finding is important to the design of helium speech intercommunication systems. Apparently, as much attention must be paid to the design of wide-bandwidth microphones which are insensitive to density as to the helium speech processors themselves.

The increased mean intelligibility scores of the processed speech as compared with the unprocessed scores were due to the reconstitution of helium speech by the electronic processor (Fig. 2). Individual processed scores (Fig. 3) varied widely, usually more than the unprocessed scores, due to the inability of the processor to cope with the variability in the characteristics of speech among individuals (12,18). Scores for GM and FS were usually higher than scores for CC and MP. The small decline in the scores at the higher pressures even without readjustment of the processor's expansion control is consistent with the observation in this study that increased pressure (density) has only a small effect on the characteristics of speech.

The exposures to helium-oxygen-pressure apparently did not cause the subjects to alter articulation noticeably in order to improve speech intelligibility to themselves or to each other. Intelligibility of the unprocessed speech as scored by the listening panels generally improved as exposure time increased, and scores in air at 1 ata at the end of the exposure period were no different than before the exposures began (Table II).

SUMMARY

Rapid compressions and acute exposures to pressure equivalents of 800, 1200 and 1600 fsw in a helium-oxygen breathing mixture did not interfere with speech formulation or articulation in four subjects. The intelligibility of unprocessed speech recorded in helium-oxygen atmospheres from 1600 to 200 fsw by wide-bandwidth condenser microphones was not substantially affected by altered ambient pressure (gas density). Characteristics of individual speech differed sufficiently that the intelligibility of processed speech varied widely among a small group of

individuals. Articulation of the four subjects apparently did not change during exposure to distorted perceptions of their own speech for up to 22 days.

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E-10. Perceptual, Memory, Cognitive and Psychomotor Functions

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It is generally agreed that rapid compression to high pressure with helium-oxygen can produce decrements in human performance, with indications that effects on psychomotor performance are most marked and that recovery may be prompt after compression is completed (4,6,7,16,29). Concurrent observations have included lowered alertness with electroencephalographic changes consistent with that state, fatigue, muscle tremor, fasciculations, and impaired coordination.

The performance experiments conducted in Predictive Studies IV were designed to survey systematically and quantitatively a broad range of perceptual, memory, cognitive and psychomotor effects of rapid compression to pressure equivalents of 400, 800, 1200 and 1600 fsw. Compression rates were selected to elicit, rather than prevent, definite symptoms and effects. Because these performance measurements were made at the same time as other studies of peripheral and central nervous system function and other physiological studies (22) (Sections D and E), the overall design of this study provided the means of comprehensively describing the functional state of individual subjects under different conditions of compression and hydrostatic pressure.

Measurements were made in some subjects both prior to and during periods of light exercise to assess effects of motor stimuli concurrent with compression-pressure effects on performance. Performance and physiological measurements were repeated in measurement modules (Section D) before, during and following changes in exposure conditions to permit

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tracking of the development of and recovery from effects of compression and hydrostatic pressure. Predictive Studies IV also provided an opportunity to assess the usefulness of a computer-controlled system for investigation of human performance under stress (19). It is anticipated that this performance measurement system may provide a means of overcoming limitations in laboratory-to-laboratory comparability of performance test results which currently exist because of the wide variety of tests now in use (3).

METHODS

TESTS, ABILITIES TESTED AND TEST SEQUENCES

Individual performance tests were selected to provide efficient and non-redundant assessment of a range of performance abilities. These tests and abilities are described in discussions concerning factor analytic performance experiments (14,17,19,20,21). From the wide range of human abilities which have been identified in such discussions, abilities were selected which were judged either to be related to effects of the "high pressure nervous syndrome" or to be prominently involved in performance tests included in previous studies of exposure to increased ambient pressure (1,2,4,12,16,29).

The 12 performance tests administered are listed in Table I. Each test was modified from its more conventional form (3,17,20,26,28) to adapt procedures and scoring to the performance test system (19), to reduce administration time and to permit many repetitions of a test to a subject.

Three test sequences, each of three minutes' duration, were composed from subgroups of the 12 performance tests (Table I). The psychomotor test sequence (one reaction time, two dexterity and two tracking tests) and the memory and cognitive test sequence (one cognitive and two memory tests) were administered to the rest subjects. The perceptual and psychomotor test sequence (two speed and two orientation tests) was administered to the exercise subjects.

The brief administration time of individual tests in close succession was required practically by the time limitations imposed by the overall measurement module system of

TABLE I. Design of Performance Experiments

Test Sequence	Test Name	Ability Assumed Tested	Test Time (sec)	Subject Assignment	
				Phase I	Phase II
Psychomotor	One-hand compensatory control test	Manual tracking	45		
	Key insertion test	Finger dexterity	20		
	Visual reaction time test	Reaction time	30	FS	FS
	Wrench and cylinder test	Manual dexterity	40		
	Two-hand compensatory coordination test	Multiple limb coordination	45		
Memory and cognitive	Word-number test	Associative memory	60		
	Arithmetic test	Number facility	60	LJ	MP
	Visual digit span test	Memory span	60		
Perceptual and psychomotor	Choice reaction time test	Response orientation	60		
	Number comparison test	Perceptual speed	45	WS CC	GM CC
	Grip speed test	Wrist-finger speed	15		
	Card rotations test	Spatial orientation	60		

tracking time-related changes (Section D). For purposes of performance testing, this three-minute group of tests is considered as a synthetic task (2). This mode of test administration was expected to maintain the motivation of the operationally experienced subjects involved in these studies. In addition, performance on the synthetic task (as opposed to scores for the individual tests) provides a measure of the skills and strategies required to perform complex operational procedures (14,16,18,19).

SUBJECT TRAINING AND ASSIGNMENT

The subjects were trained in each of the three performance test sequences. Initially, each test was performed separately without the time limitations of the actual exposures. Subsequently, groups of tests were practiced as three-minute sequences. Finally, the three-minute performance test sequences were combined with physiological tests in the major measurement modules (Section D). The measurement modules were administered in the dry chamber, both at sea level and during shallow dives, until test scores appeared to reach stable levels.

Each of six subjects was assigned to a two-man team, composed of one rest subject and one exercise subject. Table I describes the pattern according to which each subject performed one of the test sequences throughout one or both phases. In each team, the rest subject performed either the psychomotor test sequence or the memory and cognitive test sequence. The exercise subject performed the perceptual and psychomotor test sequence both prior to and during a six-minute period of light exercise on a bicycle ergometer. The temporal relationships within a measurement module between a performance test sequence and other measurements are shown in Section D.

APPARATUS

The performance testing apparatus was derived from the human test systems of PEMCON (26) and SINDBAD (28). The apparatus was redesigned at this Institute to permit on-line control of test administration and data acquisition and

off-line data analysis by a general purpose digital minicomputer. A report on the design criteria of this system has been presented elsewhere (19).

Two performance test stations were used, one for the rest subjects and another for the exercise subjects. At the rest subject station (Figs. 1 and 3), the subject sat in a chair facing a 5-1/2 inch diameter viewport, with a test console mounted on a slide-out table. Test display apparatus inside the pressure chamber included: a 23-cell display-response panel adapted by electronics and computer programming for direct computer control ; a computer-controlled 3-digit numeric display; and a rear projection screen which could be slid in front of the port. This test station could be set up in seconds for either the psychomotor test sequence or the memory and cognitive test sequence. Outside the chamber at the viewport, a computer-controlled, random-access projector and a nine-inch closed circuit television receiver were mounted on separate swing-away supports which allowed each to be presented for visual transmission through the port. The television receiver was used to present tracking tasks generated on the computer's video terminal; the 3-digit display was used to present the visual digit span test; the projector and display-response panel presented all other test stimuli.

STUDIES AT REST

The rest subject's responses, for transmission to the computer, were implemented by two force-operated hand control sticks for tracking tests, and by three response devices fabricated with magnetic cores for use with the display-response panel for all other tests (Fig. 1). Each cell of the display-response panel contained a lamp for display (illumination of selected cells), and a magnetically activated reed switch for response (closure of the reed switch when its cell was entered by a response device). The stylus was used for the memory and cognitive test sequences and for the visual reaction time test of the psychomotor test sequence. The wrench and cylinder device and the key insertion device were used for the other tests of the psychomotor test sequence in conjunction with a template which converted the round apertures of the 10 numeric cells on the display-response panel into alternating square and

round apertures. These apertures were sized so that round and square ends of the dexterity devices would fit snugly.

The "on" or "off" state of each lamp and the "open" or "closed" state of each reed switch of the display-response panel were, respectively, controlled by the computer and "read" by the computer. Illumination of a green cell indicated a test sequence was in progress; a red cell was illuminated at other times. A cell marked "RT" and a white cell were used for the visual reaction time test. Ten cells marked 0 to 9 were used by a subject to guide his order of responding or to indicate numeric answers to test items. A cell marked with an asterisk indicated that an answer to a test item had been completed.

STUDIES DURING EXERCISE

At the exercise subject station, the subject sat on a bicycle ergometer also facing a 5-1/2 inch diameter viewport (Fig. 5). The performance test apparatus at this station was limited by the requirement that this subject hold the bicycle ergometer handlebars while pedaling. Accordingly, exercise subjects' responses to test displays presented by a second projector and screen were by means of two grip-operated switches attached to the bicycle ergometer's handlebar grips. One of the four possible response combinations provided by the open and closed positions of the two switches (i.e., both switches closed) indicated the subject was ready for the next test within the test sequence or the next item within a test.

TEST PROCEDURES AND RESULTS

Each performance test sequence included different tests and was administered to different subjects (Table I). Therefore, each sequence is treated as a separate experiment in the following discussion, with a brief description of test procedures followed by the presentation of results. Additional details of procedures (19) and a brief summary of results (18) have been reported elsewhere. An appendix to this section provides in tabular form all of the test scores obtained (Appendix Tables 1-8); scores are also shown in graphic form in Figs. 2,4,6 and 7.

Scores for each test were based both upon counts of responses during the allotted test time and time characteristics of responses with respect to preceding stimuli or responses. The number of responses completed estimates production level; the number of responses correct or the number correct minus number incorrect estimates quality of performance; the number of errors estimates carefulness. The mean of inter-response times or mean reaction time estimates rate or speed of responding; the standard deviation of inter-response times or reaction times estimates stability in rate or speed of responding.

PSYCHOMOTOR TEST SEQUENCE: PROCEDURES

The reaction time test, the two dexterity tests and the two tracking tests which comprise this sequence are described in the order in which they were administered to subject FS in Phases I and II (Table I) at the test station as shown in Fig. 1.

One-Hand Compensatory Control Test

This test was designed to assess the ability of Manual Tracking, which is defined as precise, continuous adjustment of one or more axes of control to follow or to compensate for changes in a target's position, speed and/or acceleration. The subject attempted to keep a moving dot as close as possible to a stationary, centered reference mark. Uncompensated motion of the dot as generated by the computer and viewed on the closed circuit television screen was along a 4-1/2 inch straight line inclined at 45° of arc from the vertical. For a right-handed subject, the dot moved between the upper right and the lower left quadrants of the display, following a sinusoidal temporal pattern with a frequency of 25.5 cycles/minute. The subject's response was by means of the control stick on the side of his preferred hand.

The error score was computed and expressed separately for the horizontal and vertical axes. During 40 seconds of tracking, approximately 1300 samples of both x-axis and y-axis error voltages were stored in computer memory. Each x-y pair of voltages defined the extent to which the dot

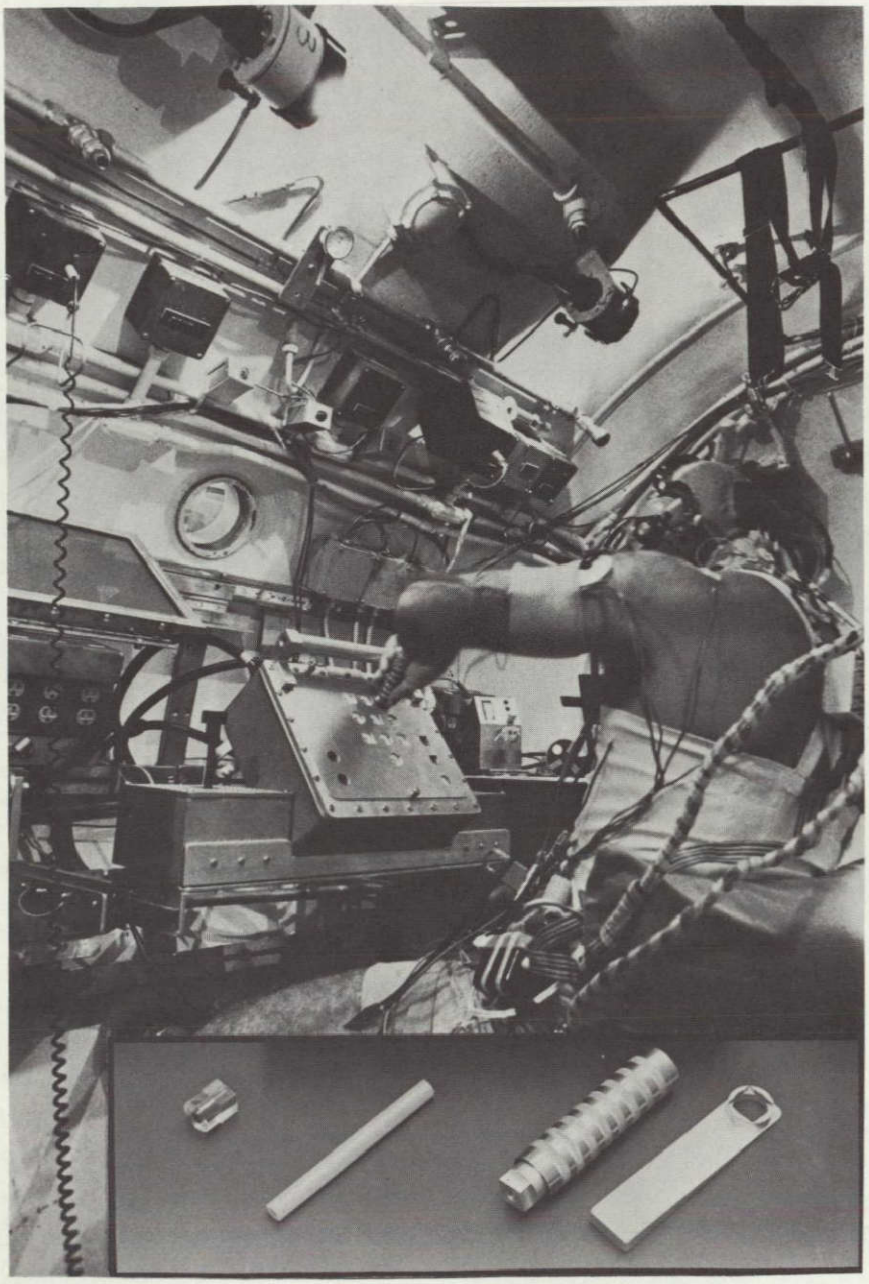

FIG. 1. The rest subject performance test station during administration of the psychomotor test sequence. The response devices used with the display-response panel are shown in the inset.

test time was calculated. This differs from the inverse of the mean reaction time to the extent that the time for a subject to start the first trial differs from zero.

Wrench and Cylinder Test

The wrench and cylinder test was included to assess the ability of Manual Dexterity--i.e., rapid, skillful manipulation of relatively large objects, using tools, with arm and hand movements. The subject used the wrench to expose alternately the round and square ends of the cylinder enclosed within a threaded sleeve (Fig. 1). The exposed end was used in the same way as the key insertion device in the test of finger dexterity.

Scores obtained for this test were the same as those for the key insertion test: the number of responses completed in each 40-second test period, the number of errors and the mean and the standard deviation of inter-response times. The score of mean inter-response time is emphasized here since it proved more sensitive for the 40-second test time than was the conventional score of number of responses completed.

Two-Hand Compensatory Coordination Test

This tracking test was included in the psychomotor test sequence to assess Multiple Limb Coordination--precise and continuous coordination of the simultaneous movements of two or more limbs to operate multiple controls in following or compensating for changes in a target's position, speed and/or acceleration. As with the one-hand tracking task, the subject attempted to keep the moving dot as close as possible to the center of the stationary reference mark.

The two tracking tasks were alike in all respects, including test time and scoring, except that in the two-hand compensatory coordination test the x-axis score represented left-hand tracking and only the horizontal component of the display dot's error position, whereas the y-axis score represented right-hand tracking and vertical dot position.

PSYCHOMOTOR TEST SEQUENCE: RESULTS

Test scores obtained during Phases I and II for subject FS (Table I) are presented graphically in Fig. 2. These must be related to the symptomatic and objective indices of compression effect described in Section E-1, with particular attention to the prominent general malaise experienced on first arrival at the 1200-fsw pressure in Phase II.

Reaction Time Test

Mean reaction time increased during the 80-minute compression from sea level to 800 fsw in Phase I and thereafter did not return to the control level. The test result at 1200 fsw on day 1 of this phase was not graphed because the subject produced only one response even after he was reminded that the test had begun.

Mean reaction time increased during the 50-minute compression to 800 fsw on the first day of Phase II. Performance in this test also decreased after reaching 800 and 1200 fsw, but it returned to the control level by the end of that day. It was at the control level at the start and end of days 2 and 3, although it increased transiently at the start of excursion-decompressions on those days.

Recovery from decrements was rapid and complete in Phase II, but in Phase I both the mean reaction time score and number of responses completed showed sustained decrements during exposure days 1 and 2. Considerable but very brief increases in mean and standard deviation of reaction time prior to the start of compression to 1200 fsw on the first day of each phase suggest that these scores are sensitive to anticipatory effects.

Dexterity Tests

Results for the key insertion test of Finger Dexterity show that performance decrements in number of responses completed did not persist throughout an exposure day except for the first day of Phase I and the first excursion from 1200 to 1600 fsw in Phase II. Decrements occurred in Phase I after compressions to 800 fsw and 1200 fsw, and in Phase II during and after compressions to 800, 1200 and 1600 fsw. The alternative scores appear to be closely associated with the number of responses completed, with the exception of the error score in Phase II. The number of errors

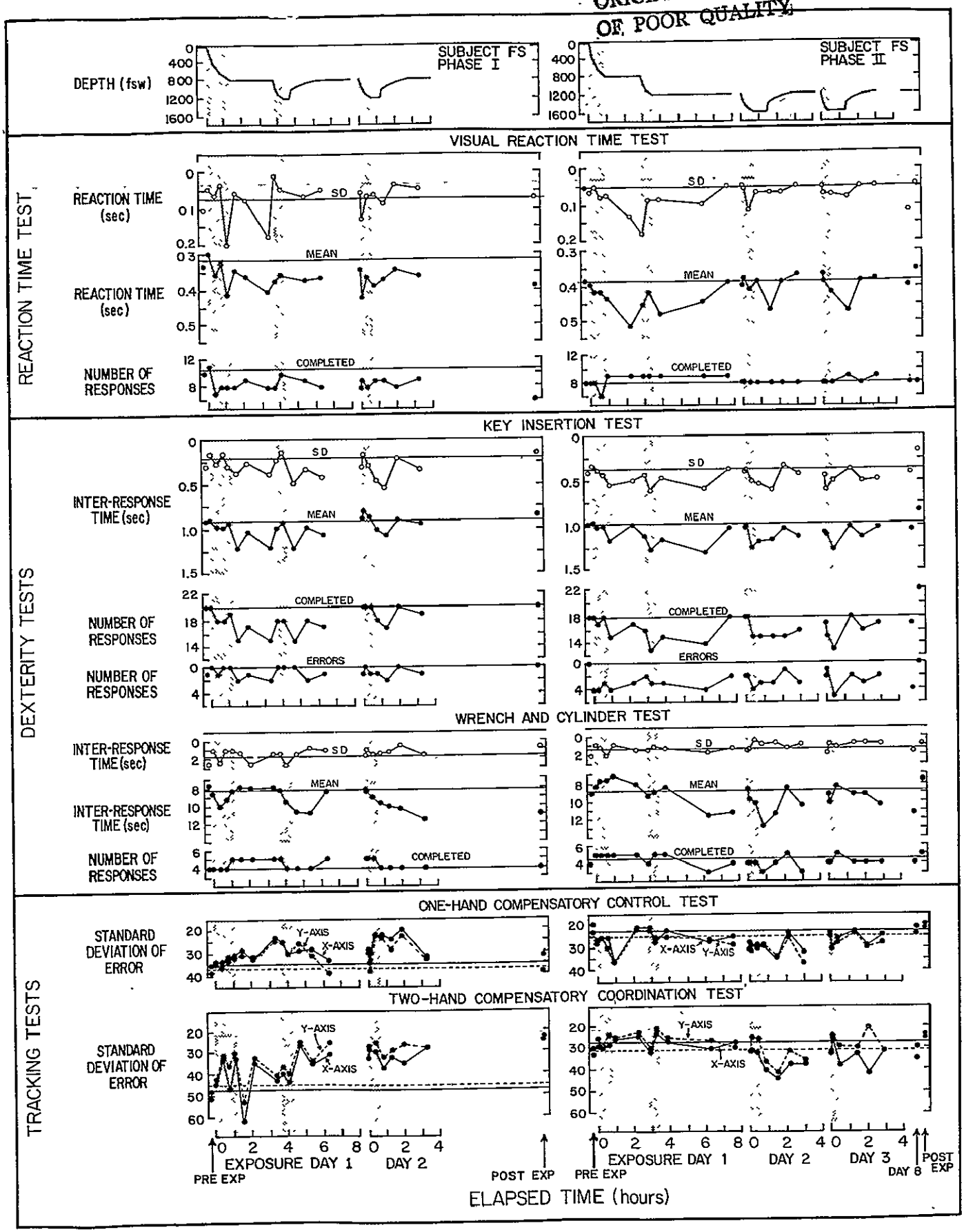

FIG. 2. Psychomotor test sequence results for subject FS in Phases I and II.

throughout that exposure phase are greater than those recorded in Phase I or in pre-exposure and post-exposure measurements.

For the wrench and cylinder test of Manual Dexterity, the number of responses completed in the 40-second test covered so narrow a range as to be insensitive to exposure conditions. In terms of the more sensitive score of mean inter-response time, results were not entirely consistent in that a decrement in performance occurred on the Phase I compression from 1 ata to 800 fsw but not during the faster compression to 800 fsw in Phase II. Mean inter-response time increased markedly during and after compression to 1200 fsw only in Phase I, and during only the first compression to 1600 fsw in Phase II. For the first exposure to a condition, the amount of decrement was apparently related to depth. Recovery appeared to be more rapid with successive exposures. Although recovery was often not complete at the end of a test day, the next exposure day started with performance at the control level.

Tracking Tests

Despite the training which preceded the exposures of Phase I, the scores in both tracking tests continued to improve throughout this phase, stabilizing at the improved level in Phase II. This overall learning trend in the one-hand compensatory control test of Manual Control was overcome in Phase I by an overall performance decrement at the end of each exposure day, perhaps related to fatigue. For the two-hand compensatory coordination test of Multiple Limb Coordination but not for the one-hand test of Manual Control, the learning trend in Phase I appeared to be interrupted by performance decrement following compression from sea level to 800 fsw and, perhaps, from 800 to 1200 fsw. In Phase II, there were decrements in the compensatory tracking scores for the One-Hand Compensatory Control test during compression from sea level to 800 fsw and for the Two-Hand Compensatory Coordination test during excursions from 1200 to 1600 fsw.

Throughout both phases and for both tracking tests, the x-axis and the y-axis error scores were usually closely associated. Exceptions occurred during excursions for the

test of Multiple Limb Coordination. During the second excursion in Phase I from 800 fsw to 1200 fsw and during both excursions in Phase II from 1200 fsw to 1600 fsw, the y-axis error score (vertical control with the right-hand joystick) was less affected than was the x-axis score (horizontal control with the left-hand joystick), but they returned to similar values following decompression to saturation pressures.

MEMORY AND COGNITIVE TEST SEQUENCE: PROCEDURES

The two memory tests and the one cognitive test which made up this performance test sequence (Table I and Fig. 3) are described below in the order administered.

Word-Number Test

This paired associates test was included to estimate the ability of Associative Memory, i.e., commission to memory by rote and recall of bits of material, regardless of complexity or meaningfulness. In each test administration the subject was given 30 seconds to memorize the six word-number pairs shown. In each word-number pair a different familiar one-syllable noun was paired with a two-digit number selected randomly from the numbers 10 through 99. When a second response slide carrying only the six words in a different order was shown, the subject used the stylus to indicate (by means of the display-response panel) the two-digit number which had been paired with each word. In Phase I the study slide was shown at the start of the three-minute test sequence and the response slide was shown two minutes later in the last 30 seconds of the sequence. Thus, the subject had to retain the word-number associations while solving arithmetic problems and recalling number series. This interference produced scores so low that the test was rendered insensitive. It was modified in Phase II by presenting the response slide immediately following the study slide.

The conventional score of the number of correct responses was supplemented with scores describing the rate of

FIG. 3. The rest subject performance test station during administration of the memory and cognitive test sequence.

producing the 12 required responses and the steadiness of responding, as the mean and the standard deviation of inter-response times, respectively.

Arithmetic Test

The Number Facility ability, requiring the rapid mental manipulation of familiar symbols according to simple and overlearned operations, was tested by having the subject solve as many simple addition, subtraction, multiplication and division problems as possible in 60 seconds. Problems were presented by slide projection, with five problems requiring one kind of operation on each slide. The subject indicated the numerical solution to each problem by means of the stylus and the display-response panel. Inter-response time was calculated as the time between two successive solutions.

The scores for this test included the conventional score of the number of correct responses made in the allotted time. In addition, the number of responses completed in the test time, the mean of inter-response times and the standard deviation of inter-response times were also computed.

Visual Digit Span Test

Relatively short-term memory was tested by means of the visual digit span test. The ability of Memory Span may be defined as accurate reproduction of recently presented material. Within each administration of this test, a trial began as a series of one-digit numerals ordered randomly were shown on the three-digit display at the rate of one numeral per second. In Phase I the series length increased from three to nine digits in two-digit steps. In Phase II the series length increased from four to seven digits in one-digit steps. This modification had little effect upon test difficulty and was introduced to avoid ending a 60-second test administration with the presentation of a long digit series to which the subject did not have an opportunity to respond. Each digit series was complete when the digital display showed all zeroes. The subject then attempted to

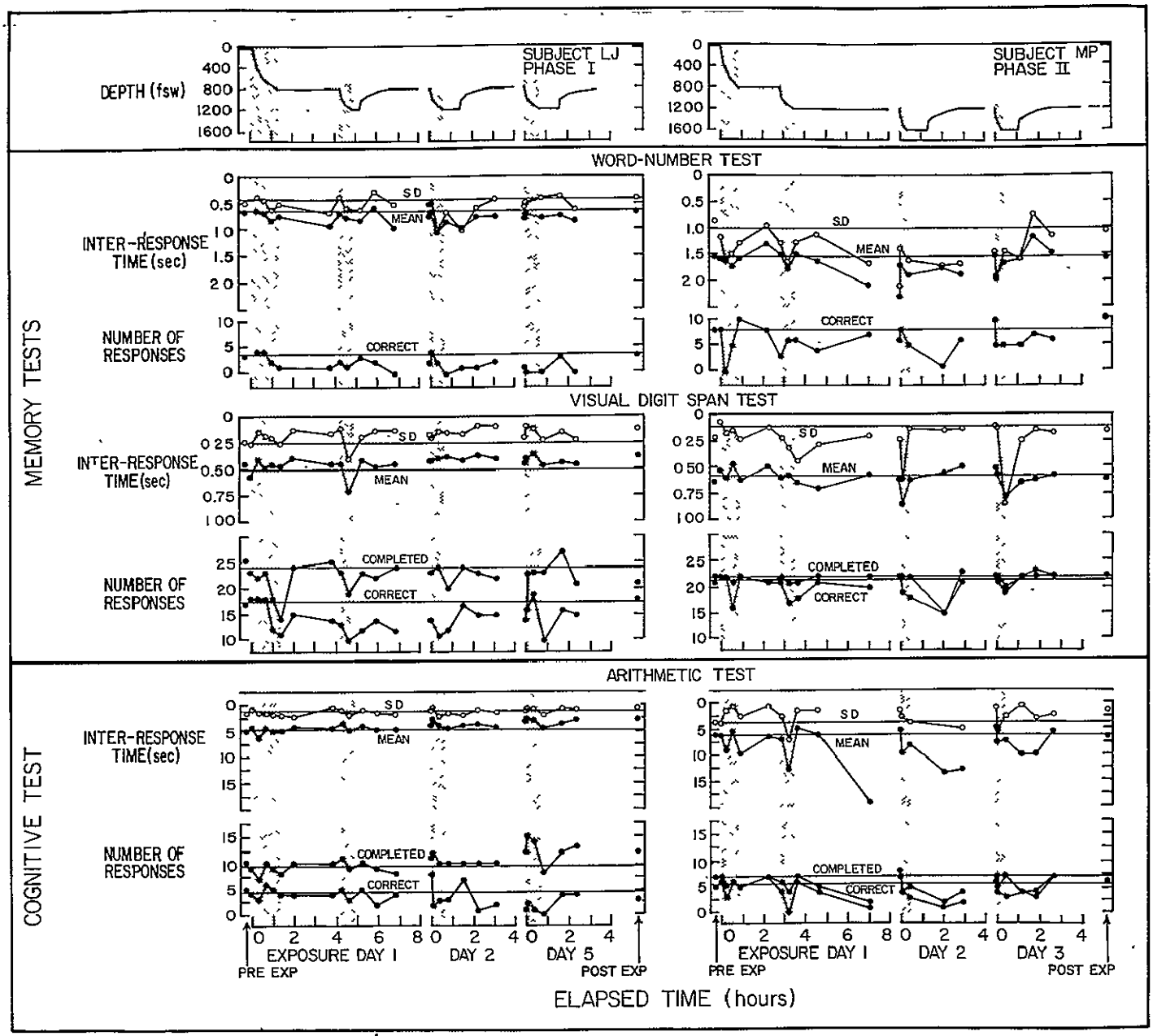

FIG. 4. Memory and cognitive test sequence results for subject LJ in Phase I and for subject MP in Phase II.

of inter-response time indicate similar relationships to exposure in Phase I, but in Phase II both mean and standard deviation of inter-response time increased during day 1, remained high on day 2, and returned to control levels by the end of day 3.

For the visual digit span test of Memory Span both subject LJ in Phase I and subject MP in Phase II usually showed performance decrements during or immediately after compression. For subject MP in Phase II the time required to recover from performance decrement increased with the first excursion to 1600 fsw as compared with compression to 1200 fsw, but recovery was more rapid for the second 1200-to-1600-fsw excursion than for the first. Similar relationships were not apparent in Phase I for LJ, who began and ended exposure days with the score of number of correct responses below the initial control level.

The two subjects also differed in the relationships between their scores based on counts of responses and scores based on inter-response times. For subject LJ the scores concerning number of responses vary with exposure condition throughout Phase I while his scores based on time characteristics of responding remain relatively constant throughout the excursions on exposure days 2 and 5. For subject MP, time-related scores follow a pattern similar to number-related scores throughout Phase II, with the exception that the standard deviation of inter-response times is more affected by compression than are other scores. For this subject the adaptation to successive excursions to 1600 fsw is evident in the scores of number of responses completed and number of responses correct but is not evident in the mean and the standard deviation of inter-response times.

Cognitive Test

All scores of the arithmetic test of Number Facility showed little change with exposure condition in Phase I. Although the score of number of correct responses did vary from the control level on days 2 and 5, the effects of compression rate, pressure and excursions were unclear because scores were not near the control level at the beginning of these exposure days. However, the pattern of the relationship between the number of correct responses and the number

of responses completed on day 5 is the same for this test as for the visual digit span test. Both tests suggest that the subject began this day at a lowered performance level and increased his response production in an apparent attempt to compensate for this lower level.

As for the other tests in this test sequence, the arithmetic test scores for subject MP in Phase II are clearer than are the scores for subject LJ in Phase I, because MP typically began test days with scores near their respective control levels. This subject demonstrated cognitive performance decrement during compressions from 800 fsw to 1200 fsw, and from 1200 fsw to 1600 fsw. Performance recovery appeared to be slower as pressure increased but to be more rapid after the second compression to 1600 fsw on day 3 than on the first such compression on day 2. Decrement late in day 1 while at 1200 fsw may have been due to fatigue.

PERCEPTUAL AND PSYCHOMOTOR TEST SEQUENCE: PROCEDURES

This performance test sequence was administered to exercise subjects at the test station illustrated in Fig. 5. The sequence (Table I) consisted of two speed tests (number comparison test and grip speed test) and two orientation tests (card rotations test and choice reaction time test). In each pair of tests, the first emphasizes perceptual function while the second emphasizes psychomotor function. In each measurement module, this sequence was first administered prior to exercise and again six minutes later during the second half of a six-minute period of light bicycle ergometer exercise. Each of the four tests which comprise this three-minute test sequence are described below in the order in which the tests were administered.

Choice Reaction Time Test

This test was developed to measure the ability of Response Orientation--rapid choice from two or more response alternatives of the response appropriate to each of two or more alternative stimulus situations. For each trial, one of three simple figures selected at random was presented to the subject after a delay which varied randomly from 1.00 to 3.00 seconds. Using the grip-operated switches, the

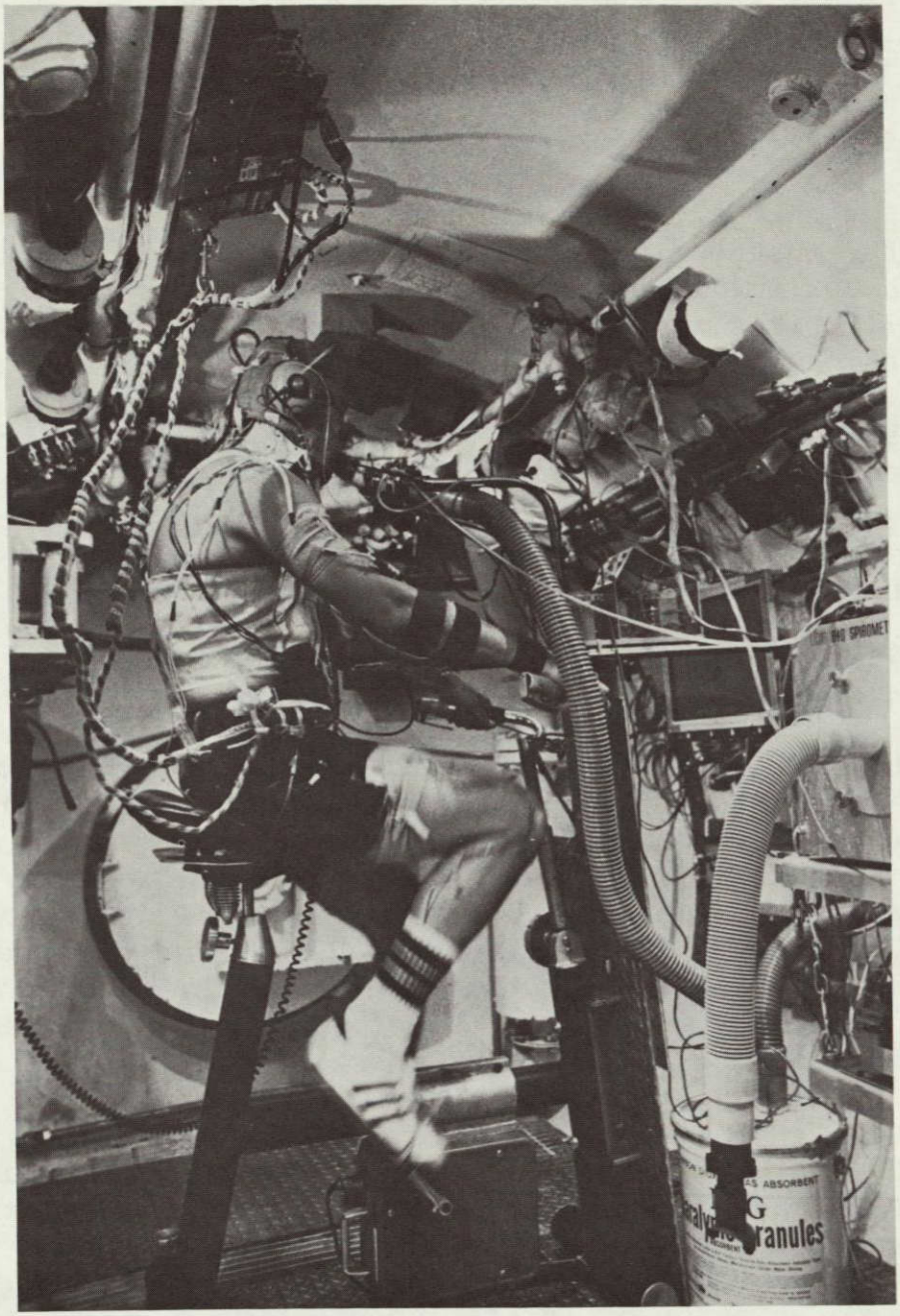

FIG. 5. The exercise subject performance test station during administration of the perceptual and psychomotor test sequence.

subject was instructed to respond as rapidly as possible with one of three possible responses associated with the presented figure. When the correct response was made, the stimulus figure was removed. When the subject indicated that he was ready for the next trial by closing both grip-operated switches, another trial began. Trials continued in this manner for 60 seconds.

For each trial, a reaction time score was calculated from the onset of the stimulus to the beginning of the subject's correct response. The mean of these reaction times for all trials completed in the test time was taken as the conventional score. The standard deviation of reaction times was calculated as an estimate of blocking. The number of responses completed and the number of incorrect responses made during the test time were also recorded.

Number Comparison Test

The number comparison test was administered to the exercise subjects as an estimate of the ability of Perceptual Speed--rapid identification, comparison or classification of familiar symbols according to simple criteria. Each slide presented to a subject carried six pairs of numbers. For each number pair, the subject was instructed to open the right-hand grip-operated switch if the two numbers were the same and to open the left-hand switch if the two numbers were different. The numbers varied only in one digit if they were intended to be different. Number length varied from 3 to 13 digits. The subject was also instructed to complete as many responses as possible in the 45 seconds allotted.

The more conventional score of the number of correct responses minus the number of incorrect responses was supplemented with scores of the number of responses completed, the number correct, mean inter-response time and standard deviation of inter-response time.

Grip Speed Test

To assess the ability of Wrist-Finger Speed (rapid repetition of simple wrist, hand and/or finger movements),

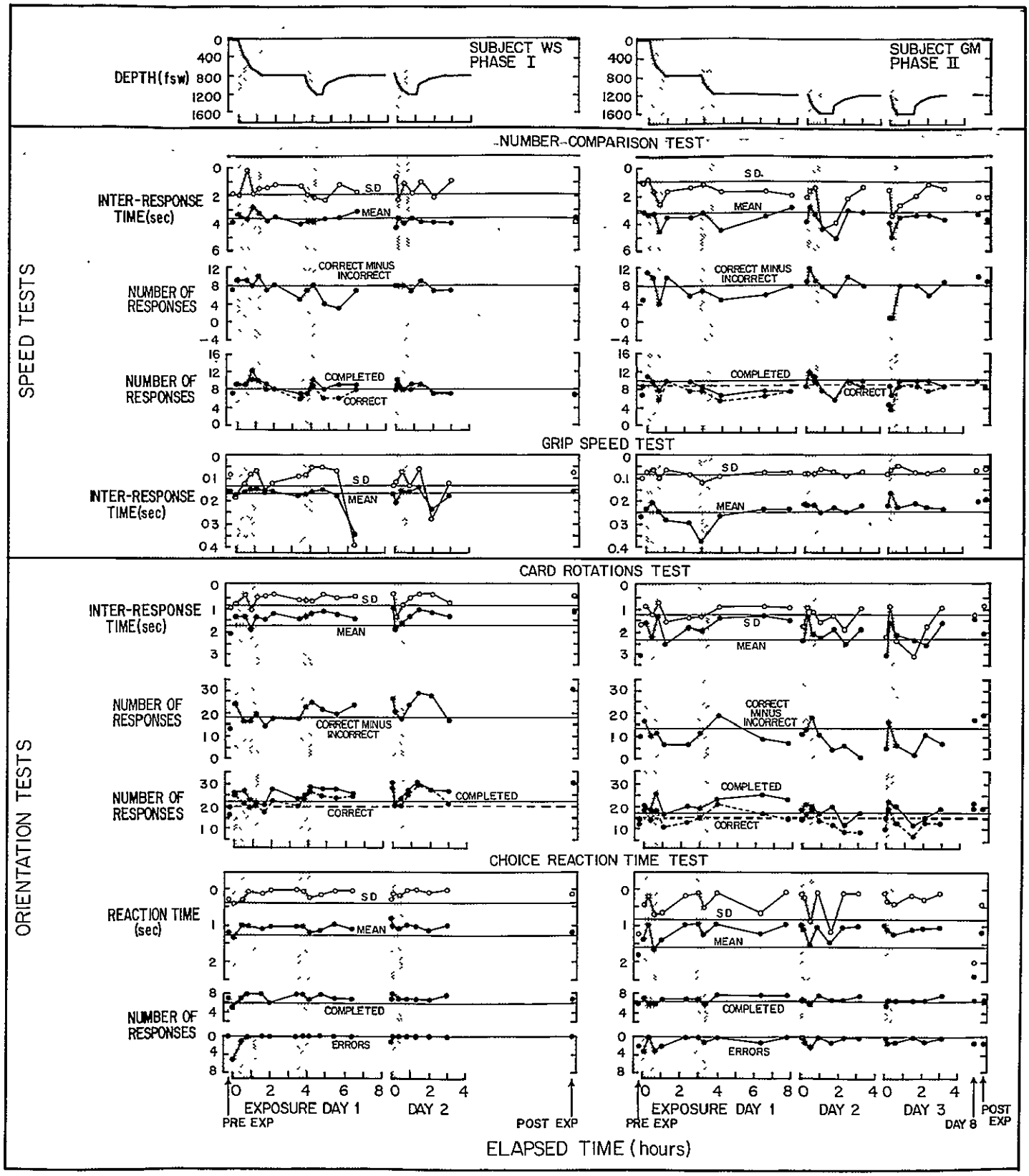

FIG. 6. Perceptual and psychomotor test sequence results for subject WS in Phase I and for subject GM in Phase II.

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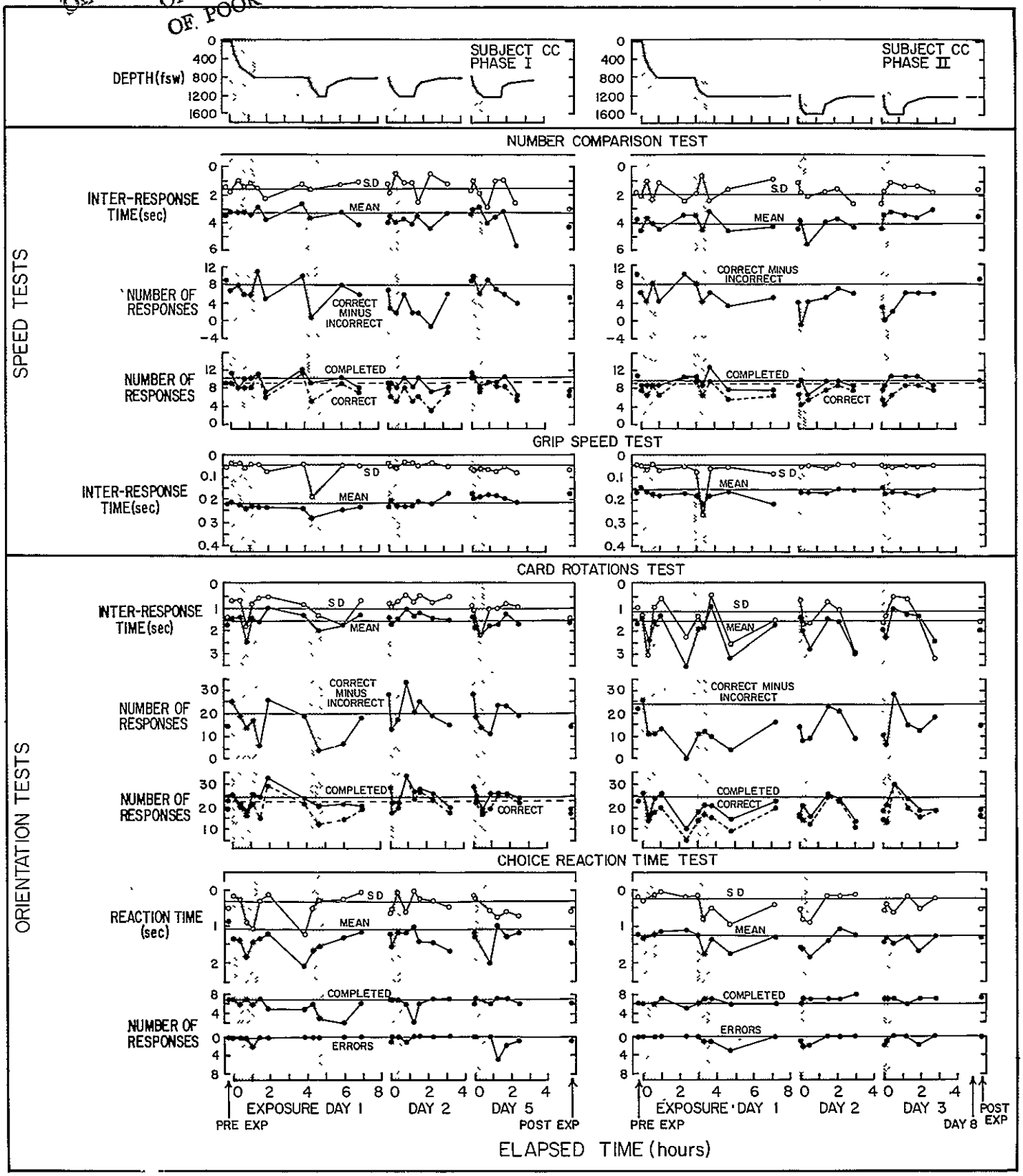

FIG. 7. Perceptual and psychomotor test sequence results for subject CC in Phases I and II.

Speed Tests

For the number comparison test of Perceptual Speed, compressions from 1 ata to 800 fsw were associated with performance decrement only to a minor extent (Figs. 6 and 7). Decrements were slightly greater during the 50-minute compressions in Phase II than in the 80-minute compressions of Phase I. Performance decrements of varying extent occurred in or after compressions to 1200 fsw usually with prompt recovery. Decrements also usually occurred in Phase II during or after compressions to 1600 fsw; recovery was usually less complete than in Phase I. Successive excursions over the same depth range appear to have progressively smaller effects upon these test scores. The scores of number of responses correct and of number correct minus incorrect (estimates of quality of performance) depart from their respective control values for subject GM at the beginning of day 3 in Phase II and for subject CC from the end of day 1 through day 3 of Phase II; other scores return to their respective control values at the beginning and the end of these exposure days. For all subjects and for all scores, sea level score values fluctuated over a wider range than was the case for other tests in this test sequence or in the other test sequences. Thus this test is probably lower in reliability than the other tests, or it may be more sensitive to variations in details of the experimental situation than the other tests.

Few performance decrements were shown in the grip speed test scores. Subject WS in Phase I performed at a relatively constant level except for a drop in performance at the end of day 1 after decompression from 1200 to 800 fsw and on day 2 during decompression. Subjects GM in Phase II and CC in Phases I and II performed this test at a relatively constant level except for transitory increases in the mean and the standard deviation of inter-response times during the compression from 800 to 1200 fsw on day 1.

Orientation Tests

The control levels for the card rotations test of Spatial Orientation may be biased by the pre-exposure scores which were low for all subjects when compared with scores obtained at 1 ata on day 1 of each phase. Subject CC's

post-dive test scores were also below the control level. Test scores for GM and CC in Phase II were low at the end of most exposure days, while subject CC's scores were low at the beginning of excursion days in Phase II as well. These variations make it difficult to interpret the effects of compression and pressure upon the card rotations test scores. Although these variations may be partially due to relatively low test reliability, some may be related to exposure. Generally, scores for this test were lower in Phase II than in Phase I. Within day 1, compression from sea level to 800 fsw was associated with performance decrements in all subjects. Decrements were considerably greater in Phase II than in Phase I. Subject CC had larger decrements and slower recovery in Phase II than did GM. On this day only subject CC had performance decrements during and after compression to 1200 fsw.

Response Orientation, as estimated by the mean reaction time score obtained in the choice reaction time test, appeared to be little affected by compression or pressure for subject WS in Phase I or for subject GM in Phase II. Subject CC demonstrated increased mean reaction times and increased scatter of reaction times--more so in Phase I than in Phase II. However, there were no consistent relationships between these performance decrements and compression or pressure. The score of number of errors remained constant at the zero level for subject WS but showed some relationship to compression and decompression for subjects GM and CC. This score reflects long-term memory of the very simple association between three alternative responses and three stimulus figures. The three subjects rank in the same order with respect to increased errors and increases in reaction time.

PERCEPTUAL AND PSYCHOMOTOR TEST SEQUENCE: RESULTS-- EFFECTS OF EXERCISE

In addition to the results discussed previously for the perceptual and psychomotor test sequence with the subjects at rest before exercise, measurements were made in each measurement module a second time during light exercise (Section E-14). Exercise was not performed during decompressions. The scores obtained while the subject was exercising are compared with the pre-exercise results in

Appendix Tables 5C (subject WS, Phase I), 6C (subject GM, Phase II), 7C (subject CC, Phase I) and 8C (subject CC, Phase II). Each value in the table is a difference between exercise and pre-exercise test scores, with the sign of the difference adjusted so that a plus sign indicates improved performance during exercise.

For measurements made at sea level (pre-exposure, first observation on day 1, and post-exposure) over all subjects and tests, scores obtained during light exercise are not systematically better or worse than scores obtained prior to exercise. However, when the mean and standard deviation of reaction time for the choice reaction time test are considered alone, performance is better with light exercise than before exercise. This pattern of comparisons also describes scores obtained at the beginning of each test day during each exposure. Because the choice reaction time test is most likely to be sensitive to the alertness of a subject in responding rapidly and correctly to environmental stimuli, these comparisons suggest that the activities preceding the test administration with light exercise and, possibly, the exercise itself may have served to alert the subject and, therefore, improve his test performance.

During compressions and exposures to high hydrostatic pressures only the score of mean inter-response time for the grip speed test was consistently affected by a repetition of the test sequence with exercise (subjects WS, GM, CC). This effect was more pronounced for subjects GM and CC than for WS. It should be noted that this effect of exercise upon the grip speed test may simply reflect an entrainment of grip speed performance by the rhythmic activity of operating the bicycle ergometer.

For all tests and all subjects during exposures, test scores concerning the temporal characteristics of performance were affected more than the other test scores by a second test administration with exercise, but the direction of the effect was not consistent from test to test or from subject to subject.

DISCUSSION

In selecting the perceptual, memory, cognitive and psychomotor performance tests administered in Predictive Studies IV, emphasis was placed on comprehensively describing the individual subject's change in performance abilities due to rapid compression and high pressure. Breadth of study was necessary since individuals can be expected to vary widely in their susceptibility to HPNS effects and to the manner in which effects are expressed (7,16,23,29). The compression circumstances selected for the overall purposes of the study were intended to induce demonstrable but not persistently incapacitating effects on most of the subjects (Section D).

SECONDARY STRESSES

Stresses secondary to compression and pressure may be expected to have nonspecific effects upon performance and to exaggerate primary effects. The symptoms of compression to high pressure (Section E-1) may distract a subject and therefore modify all abilities tested. The general fatigue of the prolonged experiment itself may have the same effect. Such fatigue which was sometimes reported by the subjects (Sections E-1 and E-5), as well as other residual effects of compression, may continue to affect performance at stable pressure after compression has been completed and adaptation to compression has occurred. In no studies has it been practical to accomplish full separation of such factors.

Other sources of potential discomfort and distraction have included increased noise during compression (Section E-9), ambient temperature which in the present studies was abruptly increased during compression from 1 ata to maintain thermal comfort (Section E-15), and some unwanted, residual temperature and humidity changes which occurred during excursion-compressions and decompressions (Section E-15).

PHYSIOLOGICAL STATE

The performance abilities of a subject are necessarily related to certain aspects of physiological status. Sporadic electroencephalographic changes consistent with lowered

states of alertness were observed during compression in the subjects (Section E-2). Other physiological changes reflecting central nervous system function in these performance studies included alterations in: visual evoked cortical responses (Section E-3), vestibular function and balance (Section E-6), and tremor and somatosensory evoked cortical responses (Section E-4). Relevant studies of auditory, visual and cardiac functions are discussed in Sections E-7, E-8, and E-13, respectively.

COMPRESSION VS. ABSOLUTE PRESSURE

The module system of repetitively testing the same subjects as time progressed made it possible to examine effects of stress on performance of individual subjects during actual compression and at stable pressure after compression was completed. Residual effects from compressions were probably present even after actual compressions ended and a stable, higher pressure was attained. Thus, while a complete separation of compression (change in pressure) and pressure effects may not be possible, transient changes during compression may be distinguished from sustained effects at a fixed pressure. The extent of recovery over the course of an exposure day and the performance at the start of a new day are relevant to the interpretation of the results.

Decrements in the scores of various performance tests were observed in the present study. Recovery occurred in some cases over a very brief time interval, such as during the compression which elicited the change. At other times, recovery took place over longer time periods, such as during the course of a day or overnight.

GENERAL ABILITIES

While the decrements signify reduction in specific performance abilities, the subjects were, with only a few temporary exceptions (Section E-1), able to carry out and complete a rigorous schedule of preparation for and conduct of experiment modules. During the days when performance measures were taken and decrements in specific test scores occurred, the subjects instrumented themselves for the complex series of experiments, served as assistant

investigators as well as subjects during the experiments, stowed the instrumentation gear and performed all housekeeping tasks within the pressure chambers.

From saturation at 1200 fsw they were compressed to 1600 fsw in 20 minutes and performed timed physical work requiring strength and dexterity on an oil wellhead underwater as effectively as during control experiments at 10 fsw (Section F). Thus, except for several periods during compressions from 1 ata when symptoms were most severe (Section E-1), the subjects were in general functionally capable even when test scores indicated marked decrements in specific aspects of performance.

COMPARISON OF PHASE I WITH PHASE II

Subjects FS and CC participated in Phases I and II. The exposures of Phase II were more severe than those of Phase I in terms of compression rate and pressures attained and therefore may be expected to produce greater effects on measures of performance in these subjects. However, since the less severe exposures were always scheduled first, adaptation to these initial exposures may have reduced the response to the subsequent, more severe exposures.

For the five tests administered to subject FS (Fig. 2), changes in performance on the first exposure days during compressions to 800 fsw and at 800 fsw were not clearly different between Phases I and II, despite the more rapid compression of Phase II. A comparison of excursions to 1200 fsw in Phase I and to 1600 fsw in Phase II shows that the key insertion test and the wrench and cylinder test gave similar results in both phases. Results of the visual reaction time test for excursions in both phases were also similar, with brief anticipatory decrements early in compression or decompression. The two tracking tasks are difficult to compare in the two phases because of prominent effects of learning and fatigue in Phase I; otherwise, the tracking scores were similar in the two phases.

Of the four tests administered to Subject CC (Fig. 7), only the choice reaction time test results differed in the two phases, with greater decrements during and after compression from 1 ata to 800 fsw and at 800 fsw in Phase when compared with Phase II. When the results during

excursions were compared, scores were similar in the two phases for both the grip speed test and the choice reaction time test. The number comparison and the card rotations tests demonstrated considerable scatter in scores obtained at 1 ata. This relatively low reliability, seen also in an earlier study (5), makes it difficult to compare perceptual test scores between the two separate studies, Phases I and II.

In summary, exposures in Phase I may have resulted in undefined adaptation, decreasing the effects of the more severe Phase II exposures. Any such adaptation is purely conjectural, with no supporting indications from the study.

EFFECTS OF SUCCESSIVE EXCURSIONS

In Phase I successive excursions from 800 to 1200 fsw on days 1, 2 and 5 can be compared for subjects LJ and CC, and successive excursions on days 1 and 2, for subjects FS and WS. For LJ and WS there were no consistent trends in performance level with repetition of excursion-compressions (Figs. 4 and 6). All of CC's scores showed improvement with successive excursions (Fig. 7). Subject FS showed adaptation in only the key insertion test (Fig. 2); of the five psychomotor tests administered to subject FS, this one is the most likely to be affected by tremor.

In Phase II successive excursions from 1200 to 1600 fsw on days 2 and 3 can be compared in subjects FS, MP, GM and CC. There was little apparent effect on the scores of the perceptual and psychomotor test sequence of repetition of excursion-compressions on those two days for subjects GM and CC (Figs. 6 and 7). All of subject MP's scores improved during the second excursion (Fig. 4), as did all of FS's scores, except the visual reaction time score (Fig. 2).

In general, the extent of adaptation in the performance capabilities tested due to successive exposures to excursions appears to vary with the subject and the ability tested.

EFFECTS OF COMPRESSION RATE IN EXCURSIONS

All excursion-compressions from 800 to 1200 fsw in Phase I were at the same rate and lasted 40 minutes (Section D). In Phase II the first compression from 1200 to 1600 fsw for GM and FS lasted 40 minutes; their second excursion-compression and all compressions over the same range for CC and MP were completed in 20 minutes. The increased excursion-compression rate on the second excursion did not result in greater deterioration in performance for subject GM and was associated with less decrement on four of the five tests administered to FS.

Adaptation to the increased pressure conditions may have masked any effect of the faster compression rate.

EFFECTS OF PRESSURE

Sustained effects of pressure on levels of performance would be demonstrated by a decrement which persisted from day to day. However, similar effects could result from other environmental stresses associated with the conditions of confinement.

Characteristically, most performance test scores in these studies were at or near control levels at the start of each exposure day, with a few exceptions as follows. For subject FS (Fig. 2), both tests involving coordination (the wrench and cylinder test and the two-hand compensatory coordination test) showed decrements at the end of days 2 and 3 and on day 8. For subject LJ (Fig. 4), all test scores were depressed at the end of day 2 and at the beginning of day 5. For subject CC in Phase II (Fig. 7), perceptual test results (the number comparison test and the card rotations test) were low at the end and beginning of test days.

Thus, there were few apparent sustained effects of pressure on the test scores. In some cases, scores during test days (and during pressure) improve; this may have been due to increased alertness due to testing, per se.

SCORES DURING EXCURSION-DECOMPRESSIONS

Marked decrements in test scores were sometimes observed during excursion-decompressions, particularly at the start of decompression. The novel, rapid decompressions (Section G-1) introduced in this Predictive Study or the activities associated with preparation for decompression may have been distracting to the subjects. Such transitory distractions may be indicated by the short-term decrements in mean reaction time at the beginning of decompression from 1600 fsw to 1200 fsw (Fig. 2).

EFFECTS OF EXERCISE

A major purpose in scheduling performance tests both before and during exercise related to the possibility that exercise might exacerbate effects of compression and pressure (Sections D, E-14), resulting in a performance decrement greater than at rest. Only the grip speed test showed such evidence of an interaction between exercise and performance, and even this result may represent an artifact of "lock-in" between gripping speed and pedaling rate. However, the possibility of such interactions deserves further study.

EFFECTS OF ABILITY TESTED AND COMPARISON WITH OTHER EXPERIMENTS

In comparing the results of Predictive Studies IV with those of other studies, it is necessary to consider differences in environmental conditions, in subjects and in test procedures (3,19). The results of these studies are compared with other recent data, (1,4,6,7,16,29) reviewed in the following section.

Perceptual Abilities

Perceptual function, as distinguished from cognitive function, has been studied infrequently during compression and pressure exposures. Different tests have led to different conclusions (29, p.404) concerning the speed of perception. The number comparison test of Perceptual Speed

showed a small but significant decrement in score at 500 fsw, reached at 15 feet/minute (25). The card rotations test of Spatial Orientation showed significant decrement in that study and also during a prolonged exposure to 1600 fsw, reached at 20 feet/hour (30).

In the present studies Perceptual Speed scores (the number comparison test) revealed decrements at 1200 fsw and 1600 fsw in subjects WS, GM and CC (Figs. 6 and 7). Decrements in Spatial Orientation (card rotations test) were shown at 800, 1200 and 1600 fsw (subjects GM and CC) (Figs. 6 and 7).

These two perceptual tests, of the twelve administered, appeared to be the lowest in test reliability. Low reliability was determined in an earlier evaluation of similar tests as well (5).

Memory Abilities

Memory abilities have not been found to be reduced by stresses of lesser rates of compression and lesser pressures in helium-oxygen, except for retention over relatively long time periods (29, p. 404) perhaps because of complicating factors such as anxiety and fatigue (10,11,25,30).

In the present studies subject LJ showed sustained decrements in Associative Memory test scores at pressure, without recovery from day to day; subject MP demonstrated decrements which were of short duration, with recovery from one day to another (Fig. 4). Brief decrements in MP's scores early in compressions and decompressions may have been due to anticipation, while fatigue was apparently a factor in LJ's scores on his exposure day 1.

Memory is also tested by the error score of the choice reaction time test. In this case more errors occurred in Phase II than in Phase I (Figs. 6 and 7).

In the visual digit span test of Memory Span (Fig. 4), both subjects demonstrated greater memory decrement with increased pressure, but there were different extents of decrement for the two subjects. Both recovered relatively rapidly.

Cognitive Abilities

Cognitive aspects of performance have been assessed in previous exposures to high helium-oxygen pressures, with mixed results (29 , p.404 ff). Tests of cognitive function, including the arithmetic test, were not significantly affected during saturation exposure at 1600 fsw after compressions very much slower than those in the present study (9,30). However, a number ordination test has shown a sustained decrement of up to 50% at 2001 fsw after multi-day, slow compression (15). At intermediate depths (to 1200 fsw) other studies have shown lesser decrement, with rapid recovery at stable pressure following the completion of compression but with considerable variation among subjects (6).

The results for the arithmetic test of Number Facility (Fig. 4) show considerable decrement in cognitive ability for subject LJ on exposure days 2 and 5 in Phase I and for MP on all three exposure days in Phase II.

Psychomotor Abilities

Psychomotor aspects of performance have been emphasized in earlier studies as well as in Predictive Studies IV because motor disturbances are known to be prominent signs of the "high pressure nervous syndrome" (6). Seven of the twelve performance tests administered in the present studies concern psychomotor function, including five tests in the psychomotor test sequence (Figs. 6 and 7).

In earlier studies, tapping and dotting tests of speed of response showed decrements of nearly 20 percent at depths of 1600 fsw (15). In the present study, using the grip speed test of Wrist-Finger Speed (Figs. 6,7), subject WS showed decrements only at the end of the first long exposure day and at the beginning of decompression on day 2. Subject CC showed evidence of blocking during compressions to 1200 fsw on the first day of both Phases I and II (Fig. 7). Both GM (Fig. 6) and CC (Fig. 7) produced almost constant scores except for moderate decrements during or immediately after their first compressions from 800 fsw to 1200 fsw.

While the grip speed test involves continuous rapid response, the reaction time test involves discrete response to a single stimulus. No performance decrement in the visual reaction time test score was observed at 800 fsw following compression at a rate of 91 feet/minute (8).

In the present studies short-term decrements immediately prior to or early in compressions and decompressions (Fig. 2) suggest anticipatory influences. Such decrements were greater in Phase II than in Phase I.

Choice reaction time tests of Response Orientation require not only rapid response to a stimulus but also choice of the appropriate response. Test results for this ability are more widely available in the literature than for the psychomotor abilities discussed in the preceding paragraphs. Little or no decrement in choice reaction time was found in exposures up to 1092 fsw following relatively high compression rates (25,27). Increased choice reaction times, typically of 10%-30% but of as much as about 25%, were observed during exposures up to 2001 fsw following slow compression over many days (15,24).

In Predictive Studies IV choice reaction time remained at or better than the control level for two subjects (WS and GM) (Fig. 6). For the third subject (CC) (Fig. 7) choice reaction time increased in Phases I and II, with greater effects in the former.

The abilities of Aiming, Finger Dexterity and Manual Dexterity have received considerable attention in earlier studies. These require rapid but precise positioning movements, rapid finger movements and rapid coordination of hand movements, respectively. In one experiment with a test involving mainly the Aiming ability, a maximum decrement of 16% was observed at 1000 fsw following compression at 16.7 feet/minute (13). Finger Dexterity has been measured with a variety of tests. In various experiments there has been no consistent relationship between the amount of decrement (ranging from 0% to approximately 35%) and hydrostatic pressure (up to 2001 fsw) or compression rate (ranging from 0.1 to 40 feet/minute) (6).

In the present studies, key insertion test scores assessing the Finger Dexterity ability deteriorated considerably in both phases (Fig. 2) as pressure was increased.

The ball-bearing test which has been administered in many previous studies, involves not only Finger Dexterity ability, but also Manual Dexterity ability. Test results have shown decrements in performance as great as 70% in compressions to pressures as great as 1500 fsw. However, performance decrements for this test were typically lower, in the range of 20%-50% in exposures to 1000 fsw. These decrements were related to compression rate and were maximal immediately after compression ended. The rate of recovery appeared to be slower than for other psychomotor abilities and to become slower as pressure increased (6).

The Manual Dexterity ability per se has been evaluated with a variety of tests at pressures to 2001 fsw. Decrements of 0%-60% have been observed, with no clear relationship between performance scores and pressure or compression rate. For example, scores with the wrench and cylinder test did not decrease in multi-day compressions up to 1600 fsw (30).

In the present studies performance on the wrench and cylinder test deteriorated during the rapid compression and exposure to pressure (Fig. 2). Fatigue may have been a more prominent factor in this test than in the other psychomotor tests since it is the more physically demanding.

Abilities assessed by means of precise tracking tests have not been widely used in compression-pressure exposures. In one experiment where a tracking test was administered, no decrement was observed during sustained exposure to 1600 fsw (30).

In Predictive Studies IV transitory loss of control and coordination was associated with rapid compression to 800 fsw. Additional decrements in control and coordination occurred during excursions from 1200 to 1600 fsw (Fig. 2). The learning effects, observed in these tracking tests throughout Phase I, are important in demonstrating that considerable learning can occur during exposure to rapid compression and high pressure.

SUMMARY

Measures of perceptual, memory, cognitive and psychomotor performance were obtained during compression, at stable high pressures following compression, during decompression from excursions, and at stable high pressures following excursion-decompressions. Results varied widely among subjects both with test administered and with exposure conditions; there were few consistent performance decrements. Recovery time from decrements varied from minutes to overnight.

The subjects were functionally competent even when test scores indicated marked performance decrements, except for several periods when signs and symptoms of compression and pressure were most severe.

No marked differences in performance test results were observed in the more severe exposures of Phase II as compared with Phase I in the two subjects who participated in both exposure phases. This lack of correlation between performance decrement and exposure severity may be due to adaptation during exposure to the less severe circumstances of Phase I which preceded Phase II.

Successive exposures to excursions from 800 fsw to 1200 fsw (Phase I) resulted in adaptation (progressive improvement in test scores) in one subject (CC) but no change in three subjects (LJ, WS, FS). Successive excursions from 1200 fsw to 1600 fsw (Phase II) resulted in adaptation in two subjects (MP, FS) and no change in two subjects (CC, GM).

There were essentially no evident sustained effects of high pressure on performance levels. Most test scores were at or near control levels at the start of each exposure day.

General physiological activation of exercise did not interact with compression-pressure effects. Performance scores during exercise were essentially the same as before exercise.

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APPENDIX TABLE 1. Psychomotor Test Sequence Scores for Subject FS in Phase I

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	REACTION TIME TEST			DEXTERITY TESTS							TRACKING TESTS			
			VISUAL REACTION TIME TEST			KEY INSERTION TEST				WRENCH AND CYLINDER TEST			ONE-HAND COMPENSATORY CONTROL TEST		TWO-HAND COMPENSATORY COORDINATION TEST	
			Reaction Time (sec)		Number of Responses	Number of Responses		Inter-response Time (sec)		Number of Responses	Inter-response Time (sec)		Standard Deviation of Errors			
			Mean	SD	Completed	Completed	Errors	Mean	SD	Completed	Mean	SD	x axis	y axis	x axis	y axis
Pre-exp.	0	-	0.33	0.10	10	20[b]	1[b]	0.91	0.30	5.33[b]	7.68	2.29	35.43	38.92	50.39	48.23
1	0	0	0.30	0.04	11	20	0	0.89	0.15	4	8.60	0.88	34.05	34.82	44.89	42.53
	410	0:23	0.35	0.06	7	18	1	0.97	0.27	4	9.93	2.18	34.06	36.28	34.02	32.06
	600	0:43	0.32	0.03	8	18	0	0.98	0.16	4	9.27	0.76	31.65	33.54	46.97	36.84
	703	1:03	0.41	0.20	8	19	0	0.93	0.29	5	8.22	0.77	31.16	32.28	33.63	31.07
	800	1:30	0.34	0.05	8	15	2	1.21	0.38	5	7.89	1.14	29.25	31.21	61.36	53.17
	800	2:06	0.36	0.07	9	17	1	1.03	0.26	5	8.00	2.31	32.90	31.76	35.34	32.89
	800	3:20	0.40	0.17	8	15	2	1.19	0.38	5	7.94	1.20	23.57	25.02	43.12	40.78
	820	3:42	0.37	0.01	8	18	0	0.99	0.23	5	8.24	1.12	25.25	25.17	40.13	36.93
	1105	4:02	0.36	0.04	10	18	0	0.93	0.13	4	9.50	2.47	30.32	30.44	43.89	40.10
	1025	4:42	-	-	-	15	0	1.21	0.49	4	10.63	1.32	29.06	25.95	27.65	26.19
	880	5:22	0.37	0.06	9	18	2	0.97	0.32	4	10.75	0.57	28.48	31.05	35.37	34.80
	800	6:18	0.36	0.04	8	17	1	1.06	0.42	5	8.37	0.89	33.50	39.33	31.47	26.27
	800	0	0.34	0.05	8	20	1	0.87	0.30	5	8.11	1.45	28.84	30.64	32.95	27.49
	820	0:01	0.42	0.12	9	20	0	0.80	0.16	5	8.45	0.82	34.79	38.49	29.00	29.05
	1105	0:21	0.36	0.06	8	20	1	0.87	0.29	5	9.06	1.37	22.50	22.10	30.34	25.99
	1200	0:46	0.39	0.06	9	18	1	1.00	0.45	4	9.78	1.19	22.39	23.78	37.23	32.60
	1025	1:16	0.37	0.08	9	17	2	1.07	0.55	4	10.08	1.07	24.72	28.87	32.62	29.26
	880	1.56	0.34	0.03	8	20	0	0.89	0.20	4	10.33	0.32	20.33	22.46	35.39	26.75
	800	2:52	0.36	0.04	9	19	1	0.94	0.34	4	11.52	1.37	32.10	33.35	27.96	28.22
Post-exp.	0	-	0.39	0.07	6	20	0	0.84	0.15	4	10.85	0.53	32.24	39.30	24.73	23.87

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

[b]In pre-exposure measurements for Subject FS test times were 30 sec for both the key insertion and wrench and cylinder tests. All other test times were 20 sec for the key insertion test and 40 sec for the wrench and cylinder test. These scores have been adjusted for this difference.

APPENDIX TABLE 2. Psychomotor Test Sequence Scores for Subject FS in Phase II

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	REACTION TIME TEST			DEXTERITY TESTS							TRACKING TESTS			
			VISUAL REACTION TIME TEST			KEY INSERTION TEST				WRENCH AND CYLINDER TEST			ONE-HAND COMPENSATORY CONTROL TEST		TWO-HAND COMPENSATORY COORDINATION TEST	
			Reaction Time (sec)		Number of Responses	Number of Responses		Inter-response Time (sec)		Number of Responses	Inter-response Time (sec)		Standard Deviation of Errors			
			Mean	SD	Completed	Completed	Errors	Mean	SD	Completed	Mean	SD	x axis	y axis	x axis	y axis
Pre-exp.	0	-	0.38	0.04	8	18	0	0.99	0.40	4	9.16	1.82	19.54	23.29	29.78	32.25
1	0	0	0.39	0.06	8	18	4	0.97	0.33	5	8.37	0.62	27.34	27.79	25.22	28.88
	417	0:14	0.41	0.04	8	17	4	1.01	0.39	5	7.62	1.00	24.79	26.23	28.89	29.49
	659	0:33	0.41	0.07	6	18	3	1.00	0.44	5	7.58	1.84	26.47	30.11	28.00	23.84
	800	0:56	0.43	0.07	9	15	4	1.17	0.55	5	7.15	0.64	36.53	37.06	25.58	25.02
	800	2:13	0.51	0.13	9	17	3	0.99	0.49	5	8.00	1.28	21.54	22.34	24.48	22.55
	880	2:54	0.45	0.18	9	16	2	1.13	0.43	4	9.29	1.33	21.72	23.29	31.54	29.66
	1115	3:13	0.41	0.08	9	13	3	1.28	0.60	5	8.92	0.93	28.34	26.19	23.37	20.86
	1200	3:51	0.48	0.08	9	15	3	1.15	0.45	5	8.43	1.20	23.09	25.82	27.01	24.84
	1200	6:15	0.44	0.09	9	14	4	1.30	0.58	3	11.61	1.63	28.24	27.13	29.91	26.23
	1200	7:38	0.39	0.04	9	18	2	1.02	0.37	4	11.28	1.10	25.81	29.34	27.15	29.44
2	1200	0	0.39	0.04	8	18	2	1.01	0.39	4	8.63	1.35	31.35	28.50	31.56	31.09
	1280	0:04	0.37	0.04	8	18	2	1.00	0.37	4	9.85	1.30	30.31	31.58	30.84	24.84
	1515	0:23	0.40	0.11	8	15	4	1.24	0.51	4	10.23	0.24	29.58	31.11	31.27	25.36
	1600	0:46	0.38	0.06	8	15	3	1.18	0.52	3	12.96	0.76	29.98	29.65	39.42	35.61
	1420	1:29	0.46	0.06	8	15	3	1.16	0.59	4	11.51	0.59	35.80	34.81	43.17	41.06
	1280	2:09	0.38	0.06	8	15	1	1.03	0.33	5	8.52	1.12	24.31	26.40	36.43	30.58
	1200	3:00	0.36	0.04	8	16	3	1.12	0.42	3	10.52	0.65	32.48	37.72	36.66	34.78
3	1200	0	0.36	0.04	8	17	2	1.07	0.43	4	9.41	1.67	24.12	25.54	32.02	31.48
	1320	0:03	0.38	0.06	8	15	1	1.09	0.59	4	10.22	0.75	31.29	31.42	25.87	24.40
	1600	0:26	0.41	0.06	8	13	5	1.26	0.49	5	8.39	1.07	27.03	29.03	37.45	29.02
	1420	1:22	0.47	0.07	9	18	2	1.00	0.36	4	9.28	0.66	23.71	24.51	31.98	29.25
	1285	2:01	0.38	0.04	8	16	3	1.12	0.49	4	9.26	0.59	29.98	30.83	40.62	20.56
	1200	2:52	0.38	0.04	9	17	2	1.02	0.48	4	10.49	0.63	25.01	28.44	30.88	31.20
8	1200	-	0.39	0.11	8	17	4	1.04	0.40	4	11.43	1.45	21.73	24.60	34.20	29.05
Post-exp.	0	-	0.35	0.04	8	22	0	0.82	0.15	5	7.76	0.76	20.91	22.36	24.74	24.00

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 3. Memory and Cognitive Test Sequence Scores for Subject LJ in Phase I

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	MEMORY TESTS								COGNITIVE TEST			
			WORD-NUMBER TEST			VISUAL DIGIT SPAN TEST				ARITHMETIC TEST			
			Number of Responses	Inter-response Time (sec)		Number of Responses		Inter-response Time (sec)		Number of Responses		Inter-response Time (sec)	
			Correct	Mean	SD	Correct	Completed	Mean	SD	Correct	Completed	Mean	SD
Pre-exp.	0	-	3	0.65	0.47	17	25	0.44	0.25	5	10	5.02	1.55
1	0	0	-	-	-	18	23	0.57	0.27	4	9	4.44	0.98
	411	0:21	4	0.62	0.36	18	22	0.41	0.15	3	7	6.34	1.48
	610	0:41	4	0.66	0.40	18	23	0.48	0.19	6	10	4.45	1.76
	708	1:01	2	0.80	0.62	12	18	0.46	0.21	5	9	5.03	1.95
	800	1:24	1	0.73	0.49	11	14	0.48	0.26	4	8	5.13	1.84
	800	2:00	-	-	-	15	24	0.40	0.13	4	10	4.27	2.24
	800	3:47	1	0.91	0.66	14	25	0.45	0.16	4	10	4.44	0.58
	840	4:15	2	0.66	0.34	13	23	0.45	0.12	5	11	3.73	1.08
	1118	4:35	1	0.75	0.58	10	19	0.73	0.42	3	9	4.93	2.13
	1025	5:14	3	0.79	0.61	12	23	0.42	0.19	5	10	4.14	1.12
	885	5:54	2	0.55	0.25	14	22	0.47	0.13	2	9	4.86	1.70
	800	6:50	0	0.94	0.51	12	24	0.45	0.13	4	8	4.81	1.95
2	800	0	2	0.72	0.50	14	23	0.42	0.17	8	11	3.95	1.17
	820	0:01	4	0.64	0.49	14	23	0.41	0.20	2	12	2.78	0.70
	1105	0:21	2	1.03	1.00	11	24	0.40	0.14	3	10	4.14	2.37
	1200	0:47	0	0.83	0.65	12	20	0.39	0.16	3	10	4.64	1.59
	1025	1:31	1	0.95	0.97	17	24	0.42	0.16	7	10	4.03	2.13
	875	2:14	1	0.73	0.56	15	23	0.37	0.08	1	10	3.74	1.02
	800	3:07	2	0.73	0.40	15	22	0.40	0.09	2	10	4.32	1.63
5	800	0	1	0.77	0.55	14	16	0.44	0.19	1	12	3.32	0.92
	820	0:01	0	0.70	0.47	16	23	0.40	0.08	2	15	2.77	0.76
	1105	0:21	-	-	-	19	23	0.36	0.12	1	14	2.99	0.78
	1200	0:47	0	0.75	0.38	10	23	0.45	0.22	0	8	4.59	1.97
	1020	1:41	3	0.71	0.35	16	27	0.44	0.15	4	12	3.48	0.72
	910	2:21	0	0.82	0.61	15	21	0.46	0.22	4	13	2.92	1.05
Post-exp.	0	-	3	0.65	0.39	18	21	0.38	0.12	3	12	2.69	0.63

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 4. Memory and Cognitive Test Sequence Scores for Subject MP in Phase II

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	MEMORY TESTS								COGNITIVE TEST			
			WORD-NUMBER TEST			VISUAL DIGIT SPAN TEST				ARITHMETIC TEST			
			Number of Responses	Inter-response Time (sec)		Number of Responses		Inter-response Time (sec)		Number of Responses		Inter-response Time (sec)	
			Correct	Mean	SD	Correct	Completed	Mean	SD	Correct	Completed	Mean	SD
Pre-exp.	0	-	8	1.52	0.83	21	22	0.63	0.21	5	7	5.98	3.73
1	0	0	8	1.55	1.13	22	22	0.53	0.06	6	7	6.18	3.87
	450	0:14	0	1.57	1.58	22	22	0.60	0.17	3	5	8.92	1.62
	680	0:33	5	1.70	1.47	16	21	0.48	0.15	6	6	5.51	0.75
	800	0:54	10	1.55	1.25	22	22	0.61	0.23	5	5	9.76	2.63
	800	2:15	8	1.27	0.92	21	21	0.50	0.11	7	7	6.32	0.57
	880	2:54	3	1.48	1.25	21	22	0.59	0.22	4	6	7.13	2.65
	1115	3:13	6	1.75	1.64	17	21	0.58	0.32	0	4	12.56	7.19
	1200	3:37	6	1.48	1.24	18	21	0.64	0.45	6	7	4.94	1.55
	1200	4:38	4	1.60	1.10	21	22	0.70	0.28	4	5	6.12	1.49
	1200	7:03	7	2.05	1.64	20	22	0.57	0.20	1	2	19.00	-
2	1200	0	6	2.28	2.06	22	22	0.61	0.22	7	8	5.31	1.58
	1320	0:03	8	1.68	1.34	19	22	0.85	0.61	4	4	9.60	2.77
	1600	0:26	5	1.88	1.60	18	22	0.61	0.13	3	5	8.03	3.91
	1410	1:24	-	-	-	-	-	-	-	-	-	-	-
	1280	2:02	1	1.75	1.71	15	15	0.55	0.14	1	2	13.30	-
	1200	2:53	6	1.87	1.67	21	23	0.49	0.13	2	4	12.80	4.99
3	1200	0	10	1.51	1.43	22	22	0.50	0.11	6	7	4.73	0.97
	1320	0:03	5	1.91	1.94	21	22	0.56	0.13	4	5	7.73	5.66
	1600	0:26	5	1.64	1.43	19	20	0.77	0.84	3	7	7.29	2.77
	1420	1:11	5	1.56	1.58	22	22	0.64	0.25	4	4	9.88	0.64
	1285	1:50	7	1.14	0.72	22	23	0.61	0.14	3	4	9.83	3.08
	1200	2:41	6	1.46	1.14	22	22	0.58	0.18	7	7	5.62	2.36
Post-exp.	0	-	10	1.56	1.05	22	22	0.60	0.15	6	6	6.52	1.53

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 5A. Perceptual and Psychomotor Test Sequence Scores for Subject WS in Phase I. Measurements Made with Subject at Rest.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	7	7	7	3.92	1.86	0.15	0.09	13	16	19	2.11	0.97	1.21	0.34	7	0
1	0	0	9	9	9	3.35	1.98	0.17	0.18	23	24	25	1.37	0.81	1.33	0.44	5	5
	448	0:27	9	9	9	3.72	0.16	0.16	0.12	16	21	26	1.38	0.43	1.01	0.32	7	1
	625	0:47	8	10	12	2.87	1.84	0.15	0.08	16	19	22	1.90	1.09	1.04	0.13	8	0
	723	1:07	10	10	10	3.21	1.44	0.14	0.07	19	20	21	1.39	0.52	-	-	-	-
	800	1:34	7	8	9	3.79	1.39	0.16	0.15	14	17	20	1.52	0.49	1.11	0.17	8	0
	800	1:59	8	8	8	3.53	1.19	0.16	0.12	17	22	27	1.25	0.40	1.05	0.07	6	0
	800	3:24	5	6	7	4.05	1.22	0.18	0.09	17	20	23	1.47	0.66	1.04	0.05	8	0
	900	3:46	7	7	7	3.91	1.94	0.17	0.08	22	23	24	1.39	0.66	1.06	0.10	8	0
	1125	4:06	8	9	10	3.91	2.08	0.16	0.05	24	26	28	1.22	0.68	1.20	0.28	7	0
	1000	4:46	4	6	8	3.71	2.32	0.15	0.05	21	24	27	1.15	0.40	1.16	0.21	8	0
	870	5:26	3	6	9	3.60	1.14	0.17	0.06	19	23	27	1.28	0.57	0.99	0.08	7	0
	800	6:23	7	8	9	3.08	1.68	0.35	0.39	23	24	25	1.45	0.49	1.11	0.08	7	0
2	800	0	8	8	8	4.26	0.61	0.17	0.13	26	28	30	1.01	0.38	0.87	0.33	7	1
	900	0:05	8	9	10	3.61	2.30	0.21	0.12	20	20	20	1.89	1.43	1.03	0.17	8	0
	1125	0:25	8	8	8	4.04	1.11	0.16	0.07	17	20	23	1.67	0.87	1.10	0.21	7	0
	1200	0:50	7	8	9	3.64	1.82	0.16	0.13	23	25	27	1.37	0.56	1.01	0.08	7	0
	1005	1:20	9	9	9	3.86	0.97	0.14	0.06	28	29	30	1.06	0.39	1.04	0.07	7	0
	870	2:00	7	7	7	3.92	2.12	0.24	0.28	27	27	27	1.17	0.40	1.14	0.13	7	0
	800	2:57	7	7	7	3.98	0.89	0.18	0.12	16	21	26	1.36	0.77	1.02	0.06	8	0
Post-exp.	0	-	7	7	7	3.59	3.85	0.15	0.08	30	30	30	1.09	0.43	1.17	0.19	7	0

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 5B. Perceptual and Psychomotor Test Sequence Scores for Subject WS in Phase I. Measurements Made While Subject Performing Light Exercise.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	8	8	8	4.46	1.58	0.13	0.05	18	19	20	2.22	1.05	1.05	0.12	8	0
1	0	0	8	8	8	4.50	1.48	0.15	0.08	19	23	27	1.32	0.54	0.67	0.28	6	7
	504	0.33	8	8	8	4.06	1.01	0.17	0.12	14	19	24	1.53	0.43	1.05	0.07	7	0
	659	0:53	7	8	9	3.77	1.03	0.18	0.11	9	17	25	1.51	0.50	1.02	0.12	8	0
	752	1·13	8	8	8	4.20	1.79	0.17	0.07	11	16	21	1.86	0.83	1.05	0.08	8	0
	800	1:40	8	8	8	4.68	1.32	0.16	0.08	16	20	24	1.64	1.06	1.04	0.09	8	0
	800	2:15	8	8	8	4.07	1.12	0.17	0.07	17	21	25	1.57	0.58	1.05	0.12	8	0
	800	3:30	6	7	8	3.33	1.18	0.16	0.06	23	24	25	1.39	0.58	1.07	0.09	8	0
	1010	3:52	8	8	8	4.54	0.65	0.16	0.08	21	23	25	1.36	0.65	1.00	0.05	8	0
	1155	4:12	7	8	9	3.93	1.16	0.18	0.07	17	21	25	1.39	0.59	1.01	0.14	8	0
2	800	0	4	6	8	3.72	1.15	0.17	0.07	15	20	25	1.41	0.64	1.05	0.16	8	0
	1010	0:11	6	7	8	3.53	2.23	0.19	0.09	23	24	25	1.35	0.37	0.99	0.06	7	0
	1155	0:31	6	7	8	4.47	3.07	0.22	0.14	25	28	31	1.13	0.40	1.05	0.05	8	0
	1200	0:56	9	9	9	4.00	1.31	0.20	0.12	26	26	26	1.41	0.51	1.03	0.10	8	0
Post-exp.	0	-	9	9	9	3.71	1.11	0.17	0.06	23	24	25	1.56	0.48	1.01	0.09	8	0

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 5C. Effect of Exercise on Perceptual and Psychomotor Test Sequence Scores for Subject WS in Phase I[a]

Exposure Day	Depth Range (fsw)	Elapsed Time[b] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	+1	+1	+1	-0.54	+0.28	+0.02	+0.04	+ 5	+3	+1	-0.11	-0.08	+0.16	+0.22	+1	0
1	0	0	-1	-1	-1	-1.15	+0.50	+0.02	+0.10	- 4	-1	+2	+0.05	+0.27	+0.66	+0.16	+1	-2
	448-504	0:27	-1	-1	-1	-0.34	-0.85	-0.01	0.00	- 2	-2	-2	-0.15	0.00	-0.04	+0.25	0	+1
	625-659	0:47	-1	-2	-3	-0.90	+0.81	-0.03	-0.03	- 7	-2	+3	+0.39	+0.59	+0.02	+0.01	0	0
	723-752	1:07	-2	-2	-2	-0.99	-0.35	-0.03	0.00	- 8	-4	0	-0.47	-0.31	-	-	-	-
	800	1:34	+1	0	-1	-0.89	+0.07	0.00	+0.07	+ 2	+3	+4	-0.12	-0.57	+0.07	+0.08	0	0
	800	1:59	0	0	0	-0.54	+0.07	-0.01	+0.05	0	-1	-2	-0.32	-0.18	0.00	-0.05	+2	0
	800	3:24	+1	+1	+1	+0.72	+0.04	+0.02	+0.03	+ 6	+4	+2	+0.08	+0.08	-0.03	-0.04	0	0
	900-1010	3:46	+1	+1	+1	-0.63	+1.29	+0.01	0.00	- 1	0	+1	+0.03	+0.01	+0.06	+0.05	0	0
	1125-1155	4:06	-1	-1	-1	-0.02	+0.92	-0.02	-0.02	- 7	-5	-3	-0.17	+0.09	+0.19	+0.14	+1	0
2	800	0	-4	-2	0	+0.54	-0.54	0.00	+0.06	-11	-8	-5	-0.40	-0.26	-0.18	+0.17	+1	+1
	900-1010	0:05	-2	-2	-2	+0.08	+0.07	+0.02	+0.03	+ 3	+4	+5	+0.54	+1.06	+0.04	+0.11	-1	0
	1125-1155	0:25	-2	-1	0	-0.43	-1.96	-0.06	-0.07	+ 8	+8	+8	+0.54	+0.47	+0.05	+0.16	+1	0
	1200	0:50	+2	+1	0	-0.36	+0.51	-0.04	+0.01	+ 3	+1	-1	-0.04	+0.05	-0.02	-0.02	+1	0
Post-exp.	0	-	+2	+2	+2	-0.12	+2.74	-0.02	+0.02	- 7	-6	-5	-0.47	-0.05	+0.16	+0.10	+1	0

[a]Each entry is the absolute value of the difference between the value measured with the subject at rest and the value measured while the subject performed light exercise. The symbol (+) indicates that performance improved with exercise; the symbol (-) indicates that the subject's performance was poorer during exercise than while at rest.

[b]Elapsed times are those for the rest measurement periods given in part A of this table; the exercise periods occur six minutes later. Depth ranges are based on the depth given for rest (part A) and exercise (part B).

APPENDIX TABLE 6A. Perceptual and Psychomotor Test Sequence Scores for Subject GM in Phase II. Measurements Made with Subject at Rest.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	5	7	9	3.16	0.99	0.27	0.10	10	12	14	3.09	1.75	1.77	1.20	6	2
1	0	0	11	11	11	3.35	0.77	0.23	0.07	16	18	20	1.68	0.96	1.35	0.41	7	3
	484	0:18	10	10	10	3.25	1.63	0.21	0.06	10	14	18	2.25	1.34	0.95	0.16	6	0
	689	0:37	4	6	8	4.54	2.59	0.25	0.10	11	18	25	1.43	0.78	1.60	0.67	6	3
	800	1:00	10	10	10	3.49	1.60	0.28	0.06	6	11	16	2.57	1.62	1.36	0.64	7	2
	800	2:17	6	8	10	3.52	1.34	0.30	0.09	6	13	20	1.85	1.44	0.94	0.17	7	0
	960	2:58	7	8	9	3.19	1.09	0.37	0.12	11	15	19	1.99	1.41	0.90	0.08	7	0
	1135	3:17	-	-	-	-	-	-	-	-	-	-	-	-	1.18	0.49	6	1
	1200	3:55	5	6	7	4.46	1.67	0.26	0.09	19	21	23	1.43	0.96	0.91	0.08	8	0
	1200	6:19	6	7	8	3.44	1.62	0.23	0.07	9	17	25	1.35	0.94	1.17	0.63	8	1
	1200	7:42	8	8	8	2.20	1.88	0.23	0.07	7	15	23	1.52	0.97	0.91	0.06	8	0
2	1200	0	9	9	9	3.85	2.02	0.21	0.07	11	15	19	2.38	1.77	0.94	0.12	7	0
	1360	0:08	12	12	12	2.77	1.61	0.22	0.07	13	17	21	1.36	0.94	1.05	0.18	7	1
	1535	0:27	9	10	11	3.28	1.38	0.21	0.08	18	19	20	2.12	1.13	1.47	0.84	6	2
	1600	0:50	8	8	8	4.37	4.39	0.25	0.06	11	14	17	2.30	1.63	0.99	0.08	8	0
	1400	1:33	6	6	6	5.09	3.93	0.23	0.07	4	12	20	1.91	1.35	1.41	1.13	7	1
	1270	2:13	10	10	10	3.03	2.09	0.24	0.09	6	9	12	2.53	1.92	1.00	0.09	7	0
	1200	3:04	8	9	10	3.15	1.30	0.22	0.07	1	9	17	1.90	1.00	0.94	0.10	8	0
3	1200	0	1	5	9	3.93	1.47	0.22	0.08	5	10	15	3.03	2.24	0.94	0.08	6	0
	1440	0:07	1	4	7	4.99	3.42	0.16	0.06	16	19	22	1.61	0.88	1.06	0.32	7	1
	1600	0:30	8	9	10	3.54	2.63	0.22	0.04	6	13	20	2.13	2.37	1.18	0.37	7	1
	1400	1:26	8	9	10	3.39	1.91	0.21	0.07	2	7	12	2.36	3.06	1.07	0.14	7	0
	1275	2:05	6	8	10	3.38	1.08	0.22	0.08	11	13	15	2.59	1.79	1.03	0.24	7	1
	1200	2:56	9	9	9	3.66	1.38	0.23	0.06	7	13	19	1.64	0.93	1.01	0.09	8	0
8	1200	-	10	10	10	3.29	2.07	0.20	0.07	17	19	21	1.46	1.28	1.92	2.31	7	1
Post-exp.	0	-	9	9	9	3.71	2.10	0.19	0.06	19	19	19	2.06	0.91	1.12	0.38	7	1

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 6B. Perceptual and Psychomotor Test Sequence Scores for Subject GM in Phase II. Measurements Made While Subject Performing Light Exercise.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	7	8	9	3.57	1.53	0.29	0.12	3	8	13	3.04	0.62	1.19	0.59	8	1
1	0	0	10	10	10	3.43	1.70	0.22	0.08	20	20	20	2.11	1.03	1.05	0.33	7	1
	584	0:24	4	6	8	3.48	1.60	0.24	0.06	4	10	16	2.60	1.42	1.17	0.72	8	1
	733	0:43	6	7	8	4.92	1.75	0.29	0.08	10	14	18	2.09	1.30	1.38	0.62	7	3
	800	1:06	8	8	8	4.51	1.08	0.29	0.08	7	11	15	2.89	1.26	0.94	0.13	8	0
	800	2:23	9	9	9	2.49	1.70	0.23	0.08	13	17	21	1.71	0.77	1.11	0.18	8	2
	1040	3:04	5	6	7	3.81	2.76	0.28	0.10	7	16	25	1.48	0.92	1.36	0.60	7	1
	1165	3:23	2	4	6	2.95	1.77	0.22	0.08	-1	4	9	4.23	4.05	0.92	0.07	6	0
	1200	4:01	9	9	9	4.22	5.03	0.23	0.08	12	16	20	2.08	1.80	0.93	0.12	8	0
	1200	6:25	10	10	10	3.20	1.80	0.32	0.10	14	18	22	1.65	1.22	2.54	2.53	6	5
	1200	7:48	8	9	10	3.31	1.77	0.28	0.10	-6	7	20	2.41	2.02	1.83	1.86	7	4
2	1200	0	11	11	11	2.74	2.08	0.23	0.09	-1	8	17	2.07	1.70	0.93	0.07	7	0
	1440	0:14	9	10	11	2.80	1.52	0.25	0.07	5	10	15	2.86	3.46	0.98	0.11	8	0
	1565	0:33	5	7	9	2.38	1.92	0.30	0.12	5	12	19	2.23	1.31	1.45	0.62	7	3
	1600	0:56	10	10	10	3.05	1.52	0.27	0.08	2	9	16	2.53	1.75	1.00	0.04	7	0
3	1200	0	7	8	9	3.05	1.73	0.23	0.08	12	16	20	2.08	1.47	1.09	0.25	7	0
	1530	0:13	8	8	8	3.70	2.68	0.21	0.08	7	11	15	2.96	3.74	1.08	0.37	7	1
	1600	0:36	7	9	11	3.13	0.89	0.27	0.09	12	16	20	1.96	2.54	1.08	0.32	7	1
8	1200	-	8	9	10	3.19	1.04	0.26	0.07	18	19	20	1.96	1.06	0.90	0.11	7	0
Post-exp.	0	-	7	8	9	2.69	1.09	0.18	0.08	16	16	16	2.46	1.57	1.03	0.37	7	1

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 6C. Effect of Exercise on Perceptual and Psychomotor Test Sequence Scores for Subject GM in Phase II[a]

Exposure Day	Depth Range (fsw)	Elapsed Time[b] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Completed	Errors
Pre-exp.	0	-	+2	+1	0	-0.41	-0.54	-0.02	-0.02	- 7	-4	-1	+0.05	+1.13	+0.58	+0.61	+2	+1
1	0	0	-1	-1	-1	-0.08	-0.93	+0.01	-0.01	+ 4	+2	0	-0.43	-0.07	+0.30	+0.08	0	+2
	484-584	0:18	-6	-4	-2	-0.23	+0.03	-0.03	0.00	- 6	-4	-2	-0.35	-0.08	-0.22	-0.56	+2	-1
	689-733	0:37	+2	+1	0	-0.38	+0.84	-0.04	+0.02	- 1	-4	-7	-0.66	-0.52	+0.22	+0.05	+1	0
	800	1:00	-2	-2	-2	-1.02	+0.52	-0.01	-0.02	+ 1	0	-1	-0.32	+0.36	+0.42	+0.51	+1	+2
	800	2:17	+3	+1	-1	+1.03	-0.36	+0.07	+0.01	+ 7	+4	+1	+0.14	+0.67	-0.17	-0.01	+1	-2
	960-1040	2:58	-2	-2	-2	-0.62	-1.67	+0.09	+0.02	- 4	+1	+6	+0.51	+0.49	-0.46	-0.52	0	-1
	1135-1165	3:17	-	-	-	-	-	-	-	-	-	-	-	-	+0.26	+0.42	0	+1
	1200	3:55	+4	+3	+2	+0.24	-3.36	+0.03	+0.01	- 7	-5	-3	-0.65	-0.84	-0.02	-0.04	0	0
	1200	6:19	+4	+3	+2	+0.24	-0.18	-0.09	-0.03	+ 5	+1	-3	-0.30	-0.28	-1.37	-1.90	-2	-4
	1200	7:42	0	+1	+2	-1.11	+0.11	-0.05	-0.03	-13	-8	-3	-0.89	-1.05	-0.92	-1.80	+1	-4
2	1200	0	+2	+2	+2	+1.11	-0.06	-0.02	-0.02	-12	-7	-2	+0.31	+0.07	+0.01	+0.05	0	0
	1360-1440	0:08	-3	-2	-1	-0.03	+0.09	-0.03	0.00	- 8	-7	-6	-1.50	-2.52	+0.07	+0.07	+1	+1
	1535-1565	0:27	-4	-3	-2	+0.90	-0.54	-0.09	-0.04	-13	-7	-1	-0.11	-0.18	+0.02	+0.22	+1	-1
	1600	0:50	+2	+2	+2	+1.32	+2.87	-0.02	-0.02	- 9	-5	-1	-0.23	-0.12	-0.01	+0.04	-1	0
3	1200	0	+6	+3	0	+0.88	-0.26	-0.01	0.00	+ 7	+6	+5	+0.95	+0.77	-0.15	-0.17	+1	0
	1440-1530	0:07	+7	+4	+1	+1.29	+0.74	-0.05	-0.02	- 9	-8	-7	-1.35	-2.86	-0.02	-0.05	0	0
	1600	0:30	-1	0	+1	+0.41	+1.74	-0.05	-0.05	+ 6	+3	0	+0.17	-0.17	+0.10	+0.05	0	0
8	1200	-	-2	-1	0	+0.10	+1.03	-0.06	0.00	+ 1	0	-1	-0.50	+0.22	+1.02	+2.20	0	+1
Post-exp.	0	-	-2	-1	0	+1.02	+1.01	+0.01	-0.02	- 3	-3	-3	-0.40	-0.66	+0.09	+0.01	0	0

[a]Each entry is the absolute value of the difference between the value measured with the subject at rest and the value measured while the subject performed light exercise. The symbol (+) indicates that performance improved with exercise; the symbol (-) indicates that the subject's performance was poorer during exercise than while at rest.

[b]Elapsed times are those for the rest measurement periods given in part A of this table; the exercise periods occur six minutes later. Depth ranges are based on the depth given for rest (part A) and exercise (part B).

APPENDIX TABLE 7A. Perceptual and Psychomotor Test Sequence Scores for Subject CC in Phase I. Measurements Made with Subject at Rest.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Completed	Errors
Pre-exp.	0	-	9	9	9	3.37	1.31	0.21	0.05	15	19	23	1.66	1.35	0.84	0.49	7	0
1	0	0	7	9	11	3.16	1.68	0.20	0.04	25	25	25	1.40	0.66	1.32	0.17	7	0
	454	0:25	8	8	8	3.12	0.92	0.22	0.03	19	20	21	1.36	0.63	1.35	0.27	6	0
	629	0:45	6	8	10	3:18	1.31	0.23	0.05	14	16	18	2.43	1.80	1.78	0.86	7	0
	727	1:05	6	8	10	3.34	1.09	0.23	0.04	17	21	25	1.40	0.77	1.42	1.05	6	2
	800	1:28	11	11	11	2.79	1.40	0.23	0.04	6	15	24	1.55	0.56	1.33	0.31	7	0
	800	1:54	5	6	7	3.70	2.18	0.23	0.07	26	29	32	0.99	0.48	1.19	0.12	5	0
	800	3:52	10	11	12	2.58	1.13	0.24	0.04	19	21	23	1.27	0.81	2.08	1.21	5	0
	920	4:19	1	5	9	3.54	1.52	0.27	0.18	-	-	-	-	-	1.65	0.52	6	0
	1136	4:39	-	-	-	-	-	-	-	4	12	20	1.95	1.32	1.54	0.32	3	0
	875	5:58	8	9	10	3.16	1.18	0.24	0.04	7	14	21	1.72	1.71	1.30	0.28	2	0
	800	6:55	6	7	8	4.08	1.00	0.23	0.05	18	19	20	1.27	0.65	1.15	0.09	6	0
2	800	0	7	8	9	3.88	1.17	0.22	0.04	28	28	28	1.38	0.78	1.19	0.65	7	1
	900	0:05	3	6	9	3.51	1.82	0.20	0.05	13	17	21	1.68	0.90	1.56	0.54	7	0
	1125	0:25	2	5	8	3.88	0.33	0.22	0.06	17	19	21	1.44	0.70	1.16	0.08	7	0
	1200	0:51	6	8	10	3.68	1.06	0.22	0.03	33	33	33	1.03	0.42	1.17	0.61	6	1
	1200	1:17	2	5	8	4.05	1.03	0.22	0.03	20	23	26	1.30	0.69	1.02	0.04	2	0
	1005	1:35	2	6	10	3.45	2.51	0.21	0.05	25	26	27	1.19	0.42	1.42	0.26	6	0
	865	2:18	-1	3	7	4.29	0.45	0.21	0.04	19	22	25	1.42	0.71	1.44	0.31	7	0
	800	3:12	6	7	8	3.28	1.17	0.17	0.05	15	17	19	1.50	0.48	1.67	0.48	7	0
5	800	0	9	10	11	3.32	1.68	0.17	0.06	28	28	28	1.35	0.88	1.17	0.19	6	0
	900	0:05	10	10	10	2.99	0.95	0.19	0.07	18	21	24	1.82	1.05	1.29	0.26	7	0
	1125	0:25	6	7	8	2.79	1.82	0.18	0.06	14	16	18	2.16	2.18			-	-
	1200	0:51	9	9	9	3.99	2.83	0.17	0.06	11	18	25	1.75	1.03	2.00	0.57	6	0
	1200	1:17	7	8	9	3.58	0.96	0.18	0.07	23	24	25	1.69	0.98	1.00	0.77	7	5
	1001	1:45	6	8	10	3.16	0.87	0.19	0.05	23	24	25	1.25	0.81	1.28	0.59	7	2
	904	2:25	4	5	6	5.67	2.79	0.21	0.08	19	21	23	1.65	0.93	1.17	0.73	6	1
Post-exp.	0	-	5	6	7	4.28	3.02	0.17	0.06	14	16	18	1.61	1.47	1.47	0.59	6	1

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 7B. Perceptual and Psychomotor Test Sequence Scores for Subject CC in Phase I. Measurements Made While Subject Performing Light Exercise.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	5	6	7	4.07	1.33	0.21	0.06	7	13	19	2.26	2.00	1.14	0.55	6	0
1	0	0	5	6	7	4.13	2.70	0.21	0.05	19	20	21	1.68	0.90	1.71	0.55	7	1
	517	0:31	4	6	8	4.74	1.21	0.24	0.05	23	24	25	1.34	0.52	1.41	0.45	7	0
	659	0:51	3	6	9	3.89	2.05	0.22	0.06	19	22	25	1.41	0.91	1.54	1.30	7	2
	757	1:11	9	9	9	3.81	0.82	0.22	0.05	14	19	24	1.47	0.79	1.36	0.59	7	1
	800	1:34	3	7	11	3.29	1.90	0.28	0.08	19	22	25	1.40	0.64	1.57	0.41	7	0
	800	2:10	8	9	10	3.05	1.88	0.23	0.06	7	14	21	2.10	1.73	1.50	0.52	7	0
	800	3:58	7	8	9	3.80	1.18	0.26	0.05	23	26	29	1.25	0.78	1.26	0.17	8	0
	1020	4:25	10	10	10	2.93	1.23	0.30	0.09	16	21	26	1.44	0.77	1.61	0.59	7	0
	1164	4:45	-	-	-	-	-	-	-	-	-	-	-	-	2.44	1.65	5	0
2	800	0	8	8	8	3.88	0.99	0.21	0.05	15	20	25	1.28	0.57	1.22	0.60	7	1
	1010	0:11	2	5	8	3.84	3.84	0.24	0.05	17	21	25	1.51	0.77	1.03	0.38	7	1
	1155	0:31	8	9	10	2.73	1.77	0.25	0.05	17	23	29	1.18	0.56	1.40	0.44	7	0
	1200	0:57	6	8	10	3.56	1.15	0.23	0.06	20	23	26	1.29	0.44	1.54	0.66	7	0
5	800	0	10	11	12	2.81	1.67	0.20	0.06	25	26	27	1.29	0.82	1.50	0.62	7	0
	1010	0:11	7	8	9	3.63	0.93	0.20	0.08	23	23	23	1.56	0.68	1.24	0.63	7	1
	1155	0:31	-	-	-	-	-	0.19	0.06	18	22	26	1.47	0.93	1.22	0.23	8	0
	1200	0:57	9	9	9	3.45	1.88	0.24	0.07	25	26	27	1.18	0.49	1.11	0.14	7	1
Post-exp.	0	-	10	10	10	3.45	1.66	0.16	0.06	23	24	25	1.72	0.98	1.17	0.26	7	0

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 7C. Effect of Exercise on Perceptual and Psychomotor Test Sequence Scores for Subject CC in Phase I[a]

Exposure Day	Depth Range (fsw)	Elapsed Time[b] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Completed	Errors
Pre-exp.	0	-	-4	-3	0	-0.70	-0.02	0.00	-0.01	- 8	- 6	- 4	-0.60	-0.65	-0.30	-0.06	-1	0
1	0	0	-2	-3	-4	-0.97	-1.02	0.00	-0.01	- 6	- 5	- 4	-0.28	-0.24	-0.39	-0.38	0	-1
	454-517	0:25	-4	-2	0	-1.62	-0.29	-0.02	-0.02	+ 4	+ 4	+ 4	+0.02	+0.11	-0.06	-0.18	+1	0
	629-659	0:45	-3	-2	-1	-0.71	-0.74	+0.01	-0.01	+ 5	+ 6	+ 7	+1.02	0.89	+0.24	-0.44	0	-2
	727-757	1:05	+3	+1	-1	-0.47	+0.27	+0.01	-0.01	- 3	- 2	- 1	-0.07	-0.02	+0.06	+0.46	+1	+1
	800	1:28	-8	-4	0	-0.50	-0.50	-0.05	-0.04	+13	+ 7	+ 1	+0.15	-0.08	-0.24	-0.10	0	0
	800	1:54	+3	+3	+3	+0.65	+0.30	0.00	0.01	-19	-15	-11	-1.11	-1.25	-0.31	-0.40	+2	0
	800	3:52	-3	-3	-3	-1.22	-0.05	-0.02	-0.01	+ 4	+ 5	+ 6	+0.02	+0.03	+0.82	+1.04	+3	0
	920-1020	4:19	+9	+5	+1	+0.61	+0.29	-0.03	+0.09	-	-	-	-	-	+0.04	-0.07	+1	0
	1136-1164	4:39	-	-	-	-	-	-	-	-	-	-	-	-	-0.90	-1.33	+2	0
2	800	0	+1	0	-1	-0.00	+0.18	+0.01	-0.01	-13	- 8	- 3	+0.10	+0.21	-0.03	+0.05	0	0
	900-1010	0:05	-1	-1	-1	-0.33	-2.02	-0.04	0.00	+ 4	+ 4	+ 4	+0.17	+0.13	+0.53	+0.16	0	-1
	1125-1155	0:25	+6	+4	+2	+1.15	-1.44	-0.03	+0.01	0	+ 4	+ 8	+0.26	+0.14	-0.24	-0.36	0	0
	1200	0:51	0	0	0	+0.12	-0.09	-0.01	-0.03	-13	-10	- 7	-0.26	-0.02	-0.37	-0.05	+1	+1
5	800	0	+1	+1	+1	+0.51	+0.01	-0.03	0.00	- 3	- 2	- 1	+0.06	+0.06	-0.33	-0.43	+1	0
	900-1010	0:05	-3	-2	-1	-0.64	+0.02	-0.01	-0.01	+ 5	+ 2	- 1	+0.26	+0.37	+0.05	-0.37	0	-1
	1125-1155	0:25	-	-	-	-	-	-0.01	0.00	+ 4	+ 6	+ 8	+0.69	+1.25	-	-	-	-
	1200	0:51	0	0	0	+0.54	+0.95	-0.07	-0.01	+14	+ 8	+ 2	+0.57	+0.54	+0.89	+0.43	+1	-1
Post-exp.	0	-	+5	+4	+3	+0.83	+1.36	+0.01	0.00	+ 9	+ 8	+ 7	-0.11	+0.49	+0.30	+0.33	+1	+1

[a]Each entry is the absolute value of the difference between the value measured with the subject at rest and the value measured while the subject performed light exercise. The symbol (+) indicates that performance improved with exercise; the symbol (-) indicates that the subject's performance was poorer during exercise than while at rest.

[b]Elapsed times are those for the rest measurement periods given in part A of this table; the exercise periods occur six minutes later. Depth ranges are based on the depth given for rest (part A) and exercise (part B).

APPENDIX TABLE 8A. Perceptual and Psychomotor Test Sequence Scores for Subject CC in Phase II. Measurements Made with Subject at Rest.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME REST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	10	10	10	3.43	1.57	0.16	0.05	22	22	22	1.71	1.03	1.21	0.21	6	0
1	0	0	6	7	8	4.20	1.81	0.15	0.05	25	25	25	1.49	1.36	1.31	0.30	6	0
	484	0:18	4	6	8	3.34	0.70	0.16	0.07	11	13	15	2.43	3.07	-	-	-	-
	696	0:37	8	8	8	3.72	2.08	0.17	0.04	11	17	23	1.63	1.02	1.21	0.17	6	0
	800	0:58	4	6	8	4.15	0.83	0.18	0.06	13	19	25	1.36	0.65	1.14	0.07	7	0
	800	2:19	10	10	10	3.16	2.21	0.17	0.05	0	5	10	3.52	2.31	1.07	0.20	5	0
	960	2:58	8	9	10	3.18	1.59	0.18	0.08	11	14	17	1.91	1.38	1.24	0.18	6	0
	1135	3:17	4	6	8	4.22	0.32	0.22	0.26	12	16	20	1.88	1.90	1.75	0.81	7	1
	1200	3:41	6	9	12	2.88	2.16	0.18	0.06	10	15	20	0.97	0.48	1.33	0.51	7	1
	1200	4:42	3	5	7	4.25	1.27	0.16	0.06	4	9	14	3.18	2.59	1.73	0.94	6	3
	1200	7:07	5	6	7	3.97	0.60	0.22	0.09	16	19	22	1.74	1.56	1.28	0.43	6	0
2	1200	0	4	6	8	4.09	0.84	-	-	14	15	16	1.42	0.70	1.57	0.57	6	1
	1440	0:07	-1	4	9	3.58	1.55	0.17	0.06	8	14	20	2.00	1.75	1.58	0.80	7	2
	1600	0:30	4	5	6	5.29	1.86	0.17	0.05	9	12	15	2.81	1.70	1.83	0.88	7	2
	1390	1:28	5	7	9	3.67	1.52	0.17	0.06	23	24	25	1.48	0.76	1.40	0.18	7	0
	1270	2:06	7	8	9	3:43	1.29	0.15	0.05	21	22	23	1.63	1.08	1.06	0.18	7	0
	1200	2:57	6	7	8	4.10	2.36	0.16	0.05	9	11	13	2.94	2.99	1.23	0.16	8	0
3	1200	0	3	5	7	4.17	2.38	0.14	0.05	10	14	18	1.93	1.67	1.40	0.55	7	2
	1440	0:07	0	4	8	3.20	1.52	0.17	0.06	6	13	20	2.25	1.41	1.31	0.40	7	1
	1600	0:30	2	6	10	3.00	0.81	0.16	0.06	28	29	30	1.05	0.53	1.47	0.61	7	0
	1400	1:15	6	8	10	3.21	1.13	0.17	0.05	15	19	23	1.29	0.62	1.29	0.19	6	0
	1275	1:54	6	8	10	3.40	1.09	0.18	0.06	12	15	18	1.40	1.37	1.64	0.52	7	2
	1200	2:45	6	7	8	3.81	1.57	0.16	0.05	18	18	18	2.48	3.18	1.24	0.23	7	0
Post-exp.	0	-	9	9	9	3.31	1.32	-	-	14	16	18	1.97	1.63	1.30	0.53	7	0

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 8B. Perceptual and Psychomotor Test Sequence Scores for Subject CC in Phase II. Measurements Made While Subject Performing Light Exercise.

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Completed	Mean	SD	Mean	SD	Completed	Errors
Pre-exp.	0	-	10	10	10	2.87	1.78	0.17	0.06	24	24	24	1.52	0.97	1.15	0.16	7	0
1	0	0	7	9	11	2.86	0.80	0.18	0.08	6	10	14	3.41	3.73	1.09	0.15	7	0
	584	0:24	9	10	11	3.03	2.08	0.16	0.05	14	16	18	1.50	1.48	2.03	1.02	7	0
	744	0:43	9	10	11	2.69	1.30	0.18	0.06	14	17	20	2.07	1.64	1.63	1.25	7	0
	800	1:04	6	8	10	3.33	2.27	0.18	0.05	21	23	25	1.28	0.60	1.22	0.17	8	0
	800	2:25	9	9	9	3.25	1.13	0.18	0.04	10	16	22	1.84	1.67	1.27	0.38	7	1
	1040	3:04	5	7	9	3.62	1.32	0.23	0.11	21	23	25	1.47	0.91	1.57	0.61	7	1
	1165	3:23	6	8	10	3.35	0.84	0.19	0.09	10	16	22	1.67	1.17	1.37	0.54	7	1
	1200	3:47	5	8	11	2.89	0.82	0.25	0.26	12	17	22	2.01	1.78	1.52	0.42	7	3
	1200	4:48	7	9	11	3.21	1.33	0.19	0.08	12	17	22	1.75	1.03	1.50	0.64	7	0
	1200	7:13	7	8	9	2.77	1.38	0.20	0.06	11	18	25	1.61	1.29	1.25	0.22	8	0
2	1200	0	8	8	8	2.84	2.21	0.19	0.06	20	21	22	1.50	1.01	1.44	0.46	7	0
	1530	0:13	9	10	11	2.89	1.89	0.18	0.05	11	16	21	1.41	1.09	1.62	0.53	7	1
	1600	0:36	3	5	7	4.39	4.73	0.21	0.06	14	19	24	1.52	0.71	2.02	1.11	6	3
3	1200	0	8	8	8	2.95	1.51	0.19	0.06	29	29	29	1.04	0.46	1.16	0.13	7	0
	1530	0:13	9	10	11	2.98	1.79	0.18	0.08	26	28	30	1.19	0.55	1.53	0.75	8	1
	1600	0:36	2	6	10	3.40	1.73	0.23	0.08	27	31	35	0.91	0.43	2.01	0.70	7	4
Post-exp.	0	-	6	9	12	2.79	1.15	-	-	16	18	20	2.22	1.20	1.19	0.41	8	1

[a]Elapsed times are calculated from start of compression on each day as zero time to one minute into the three-minute measurement periods. Depths correspond to these times.

APPENDIX TABLE 8C. Effect of Exercise on Perceptual and Psychomotor Test Sequence Scores for Subject CC in Phase II[a]

Exposure Day	Depth Range (fsw)	Elapsed Time[b] (hr:min)	SPEED TESTS							ORIENTATION TESTS								
			NUMBER COMPARISON TEST					GRIP SPEED TEST		CARD ROTATIONS TEST					CHOICE REACTION TIME TEST			
			Number of Responses			Inter-response Time (sec)		Inter-response Time (sec)		Number of Responses			Inter-response Time (sec)		Reaction Time (sec)		Number of Responses	
			Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Correct minus Incorrect	Correct	Com-pleted	Mean	SD	Mean	SD	Com-pleted	Errors
Pre-exp.	0	-	0	0	0	+0.56	-0.21	-0.01	-0.01	+ 2	+ 2	+ 2	+0.19	+0.06	+0.06	+0.05	+1	0
1	0	0	+ 1	+2	+3	+1.34	+1.01	-0.03	-0.03	-19	-15	-11	-1.92	-2.37	+0.22	+0.15	+1	0
	484-584	0:18	+ 5	+4	+3	+0.31	-1.38	0.00	+0.02	+ 3	+ 3	+ 3	+0.93	+1.59	-	-	-	-
	696-744	0:37	+ 1	+2	+3	+1.03	+0.78	-0.01	-0.02	+ 3	0	- 3	-0.44	-0.62	-0.42	-1.08	+1	0
	800	0:58	+ 2	+2	+2	+0.82	-1.44	0.00	+0.01	+ 8	+ 4	0	+0.08	+0.05	-0.08	-0.10	+1	0
	800	2:19	- 1	-1	-1	-0.09	+1.08	-0.01	+0.01	+10	+11	+12	+1.68	+0.64	-0.20	-0.18	+2	-1
	960-1040	2:58	- 3	-2	-1	-0.44	+0.27	-0.05	-0.03	+10	+ 9	+ 8	+0.44	+0.47	-0.33	-0.43	+1	-1
	1135-1165	3:17	+ 2	+2	+2	+0.87	-0.52	+0.03	+0.17	- 2	0	+ 2	+0.21	+0.73	+0.38	+0.27	0	0
	1200	3:41	- 1	-1	-1	-0.01	+1.34	-0.07	-0.20	+ 2	+ 2	+ 2	-1.04	-1.30	-0.19	+0.09	0	-2
	1200	4:42	+ 4	+4	+4	+1.04	-0.06	-0.03	-0.02	+ 8	+ 8	+ 8	+1.43	+1.56	+0.23	+0.30	+1	+3
	1200	7:07	+ 2	+2	+2	+1.20	-0.78	+0.02	+0.03	- 5	- 1	+ 3	+0.13	+0.27	+0.03	+0.21	+2	0
2	1200	0	+ 4	+2	0	+1.25	-1.37	-	-	+ 6	+ 6	+ 6	-0.08	-0.31	+0.13	+0.11	+1	+1
	1440-1530	0:07	+10	+6	+2	+0.69	-0.34	-0.01	+0.01	+ 3	+ 2	+ 1	+0.59	+0.66	-0.04	+0.27	0	+1
	1600	0:30	- 1	0	+1	+0.90	-2.87	-0.04	-0.01	+ 5	+ 7	+ 9	+1.29	+0.99	-0.19	-0.23	-1	-1
3	1200	0	+ 5	+3	+1	+1.22	+0.87	-0.05	-0.01	+19	+15	+11	+0.89	+1.21	+0.24	+0.42	0	+2
	1440-1530	0:07	+ 9	+6	+3	+0.22	-0.27	-0.01	-0.02	+20	+15	+10	+1.06	+0.86	-0.22	-0.35	+1	0
	1600	0:30	0	0	0	-0.40	-0.92	-0.07	-0.02	- 1	+ 2	+ 5	+0.14	+0.10	-0.54	-0.09	0	-4
Post-exp.	0	-	- 3	0	+3	+0.52	+0.17	-	-	+ 2	+ 2	+ 2	-0.25	+0.43	+0.11	+0.12	+1	-1

[a]Each entry is the absolute value of the difference between the value measured with the subject at rest and the value measured while the subject performed light exercise. The symbol (+) indicates that performance improved with exercise; the symbol (-) indicates that the subject's performance was poorer during exercise than while at rest.

[b]Elapsed times are those for the rest measurement periods given in part A of this table; the exercise periods occur six minutes later. Depth ranges are based on the depth given for rest (part A) and exercise (part B).

E-11. SLEEP ELECTROENCEPHALOGRAPHIC PATTERNS

R.H. Wilcox[1], J.W. Spencer[1], F. Russo[1] and C.J. Lambertsen[2]

The inclusion of electroencephalographic recording during sleep as part of the overall Predictive Studies IV program provided an opportunity to examine brain electrical activity in a normal function critical to sustained well-being and even to survival at high ambient pressures. These measurements are relevant to high pressure exposures because the slower EEG frequencies characteristic of deep sleep (3) are consistently observed in awake individuals under conditions of pressurization (7); it is therefore important to investigate possible enhancement of pressure effects by sleep. Furthermore, the apparent restorative qualities of sleep and its extensive physiological influences upon the central nervous system make its study generally pertinent to any stressful situation.

The first systematic monitoring of sleep patterns in diving was accomplished during a 60-day, open-sea, nitrogen saturation at 2.2 ata (ONR Tektite I-University of Pennsylvania Predictive Studies II [11]), where no significant changes, except slightly increased total sleep time, occurred (9). Sleep studies were subsequently conducted during the U.S. Navy 1000-foot helium saturation dive at Duke University (1968) (18) and during early stages of decompression from a 2001-foot COMEX dive (1972) (12). The data from these records were not published. A study at 300 meters in a slightly hyperoxic helium-oxygen atmosphere showed the awakening stages of sleep (stages 1 and 2) to be increased, while stage 4 sleep was decreased (16).

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More recently the Behavioral Sciences Department of NMRI has monitored sleep in a total of five diver-subjects during three helium-oxygen high pressure exposures (1,13). Standardized sleep scoring showed significant decreases in stages 3 and 4 sleep in all five divers at or near 1000 fsw, as compared with their own controls at low pressures. However, sleep patterns at 1600 fsw in two of these divers were similar to control recordings obtained at 40 fsw during decompression (13). The observed decrease in stages 3 and 4 sleep at 1000 fsw and its apparent recovery at 1600 fsw should especially be noted because delta sleep (sum of stages 3 and 4) contains the slowest wave frequencies and appears to serve an important restorative function in comparison with other sleep stages (19). It was therefore necessary to obtain additional information within the depth range of 1000-1600 fsw.

METHODS

Brain electrical activity during sleep was recorded at sea level in subjects CC and MP for the three nights prior to compression to 1200 fsw in Phase II, and for the next three nights at that depth. The availability of three consecutive nights for sleep EEG monitoring, both at sea level and at 1200 fsw, provided important opportunity over previous studies which were limited to single-night recordings.

ELECTRODE APPLICATION

Execution of the sleep EEG study was facilitated by extensive recording of waking EEGs from the same subjects, who became proficient in the application of the electrodes. The scalp was prepared by shaving areas about 3 cm in diameter to conform to the International 10-20 System of EEG positions. The three primary locations used during the sleep studies were left frontal (F_3), right frontal (F_4), and left occipital (O_1). In addition to these areas, six other placements were used: left and right mastoid electrodes to serve as referents for the scalp placements; left and right electro-oculographic (EOG) electrodes lateral to each eyelid to monitor rapid eye movement (REM)

sleep; and left and right electromyographic (EMG) electrodes along each side of the jaw to provide a gross indication of body movement.

All electrodes were affixed with electrode cream (Grass Instruments EC2) after each site was cleaned with dilute soap solution. Biopotential Skin Electrodes (Beckman Instruments, Inc.) were applied at the three scalp sites, but their bulk prevented use at the other positions. Cup-shaped disc electrodes (Grass Instruments Model E5, gold-plated) were used in the mastoid and EOG placements, with Telectrodes (Johnson & Johnson disposable electrodes) in the EMG positions. Sleep EEG electrode units were locked in and out of the chamber independently of the waking EEG electrode systems.

EEG RECORDING AND ANALYSIS

Inside the chamber the subjects slept in adjacent bunks which allowed both men to use a single EEG electrode junction box. Records from both were obtained simultaneously on a single, 16-channel EEG recorder (Grass Model 6), using 5 channels for each record: two monopolar frontal derivations, with each frontal electrode run against the opposite mastoid reference; a monopolar occipital derivation, with the occipital electrode run against the opposite mastoid; and the two bipolar combinations of EOG and EMG information.

All sleep records were analyzed independently by two raters using the standardized method for scoring human sleep stages (15), after which discrepancies were resolved by consensus scoring. In addition, subjects filled out questionnaires to provide normative sleep data, and sleep logs were completed on the morning following each run to provide subjective assessment of sleep quality.

RESULTS

NIGHTLY SLEEP PERCENTAGES

Table I and Fig. 1 give nightly percentages of each sleep stage relative to total recording time from the

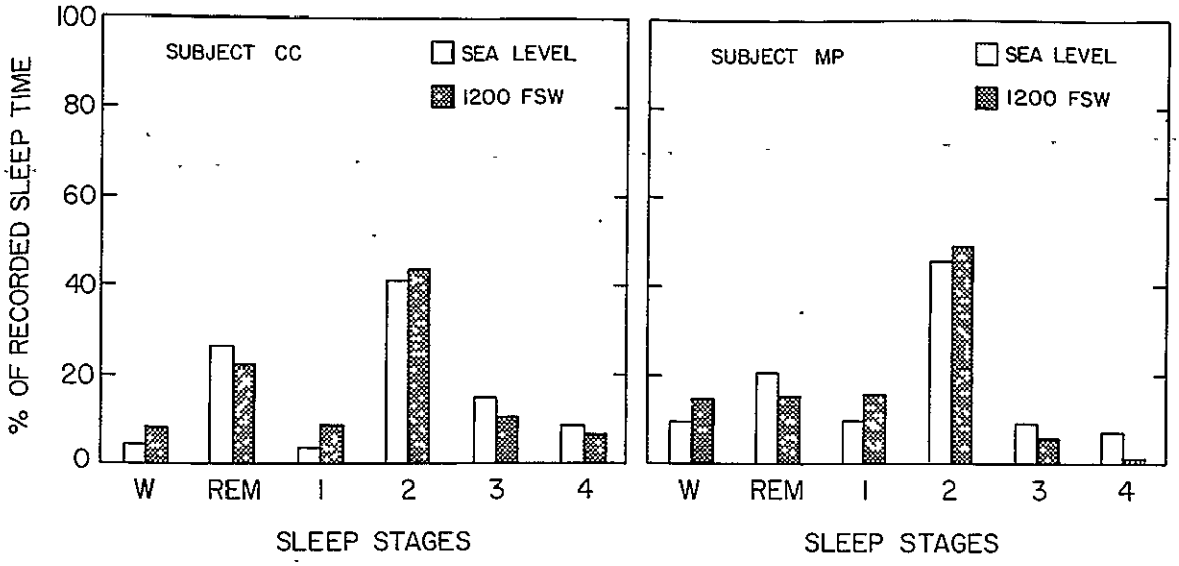

FIG. 1. Sleep stages at sea level and at 1200 fsw for subjects CC and MP. Each bar in the histogram represents the average percentage for three consecutive nights.

Sleep stages are defined as follows: W, awake. Stage 1, drowsiness; predominant low voltage, fast activity; no spindles. Stage 2, spindles, with a low voltage background. Stage 3, between 20% and 50% of record dominated by slow waves of 100 μV or more. Stage 4, at least half of record dominated by slow waves of 100 μV or more. Stage REM, rapid eye movement sleep; high arousal threshold; low voltage, desynchronized cortical EEG similar to stage 1.

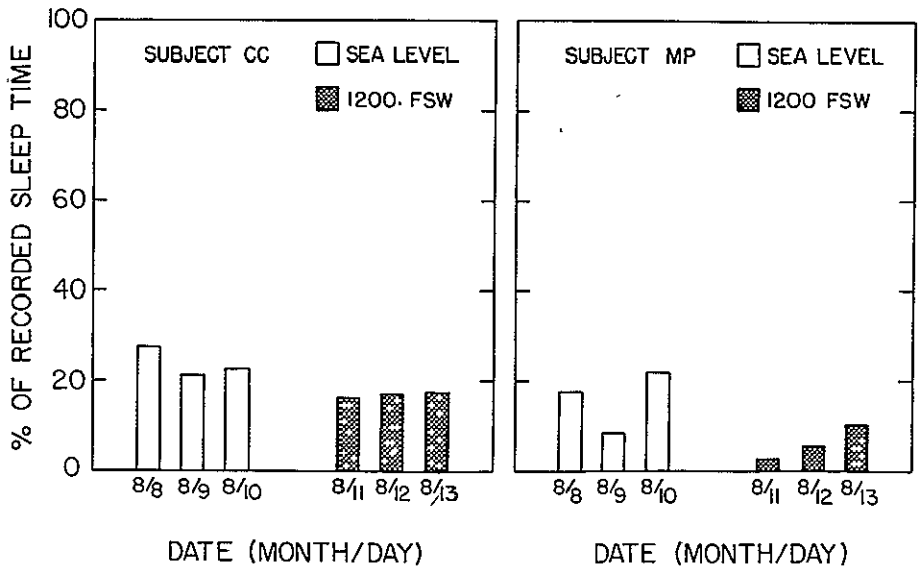

FIG. 2. Delta sleep percentages for subjects CC and MP at sea level for three consecutive nights and at 1200 fsw for three consecutive nights.

TABLE I. Relative Amounts of Sleep Stages at Sea Level and at 1200 fsw[a]

	SUBJECT CC							SUBJECT MP						
	Sleep Stage							Sleep Stage						
Date	W	REM	1	2	3	4	Delta[b]	W	REM	1	2	3	4	Delta[b]
	Control (Normobaric)							Control (Normobaric)						
8/08	1.7	28.6	4.0	38.4	17.0	10.1	27.1	3.2	21.2	12.3	46.0	11.4	5.9	17.3
8/09	2.3	27.6	3.5	45.7	12.3	8.6	20.9	12.5	20.4	7.9	51.2	6.5	1.5	8.0
8/10	11.2	23.7	3.7	39.0	16.0	6.4	22.4	13.3	18.9	7.4	38.6	8.4	13.3	21.7
Avg.	5.1	26.6	3.7	41.0	15.1	8.4	23.5	9.7	20.2	9.2	45.3	8.8	6.9	15.7
	Experimental (1200 fsw)							Experimental (1200 fsw)						
8/11	2.8	17.3	6.5	57.4	10.1	5.8	15.9	7.6	12.2	14.3	63.5	2.2	0.1	2.3
8/12	10.7	25.2	9.9	37.2	10.0	7.0	17.0	15.2	19.2	16.6	43.4	5.4	0.1	5.5
8/13	10.7	25.7	10.0	35.9	11.0	6.7	17.7	22.2	13.5	15.8	38.2	8.2	2.1	10.3
Avg.	8.1	22.7	8.8	43.5	10.4	6.5	16.9	15.0	15.0	15.6	48.4	5.3	0.8	6.0

[a]Numbers represent nightly percentages of each sleep stage relative to total recording time from the point the lights were extinguished.

[b]Delta sleep is the sum of stages 3 and 4.

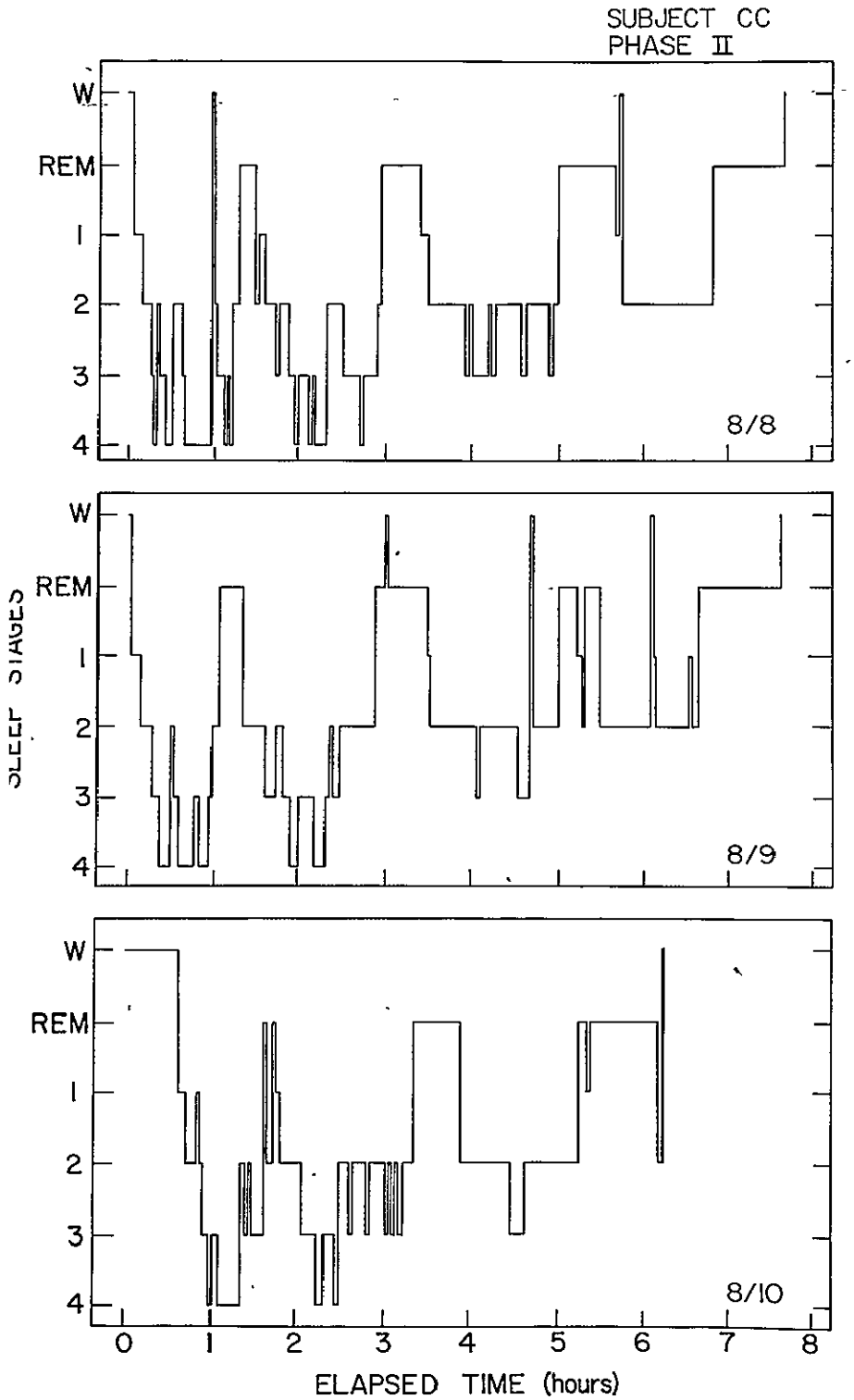

FIG. 3. Sleep profiles at sea level; subject CC, three consecutive nights.

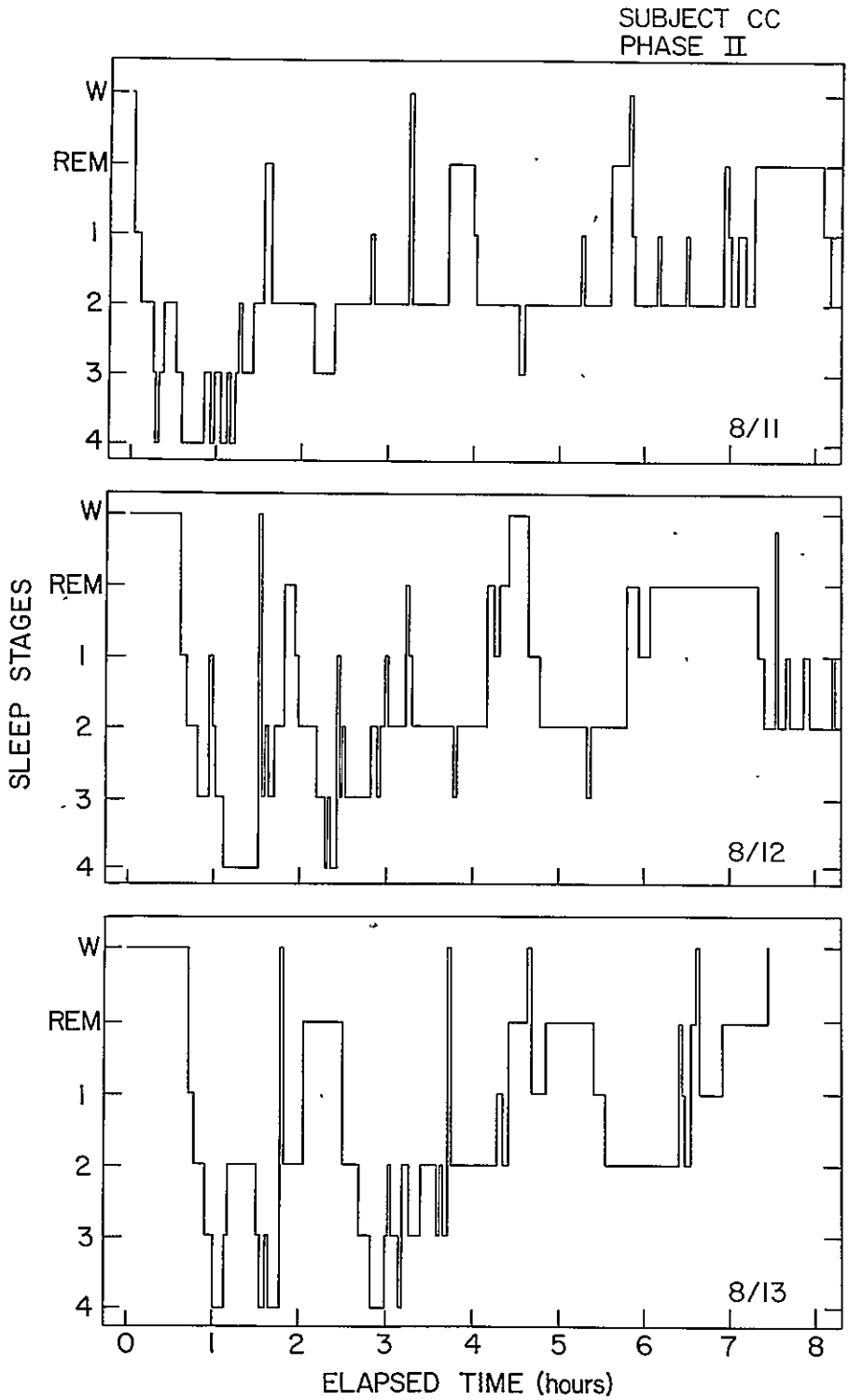

FIG. 4. Sleep profiles at 1200 fsw; subject CC, three consecutive nights.

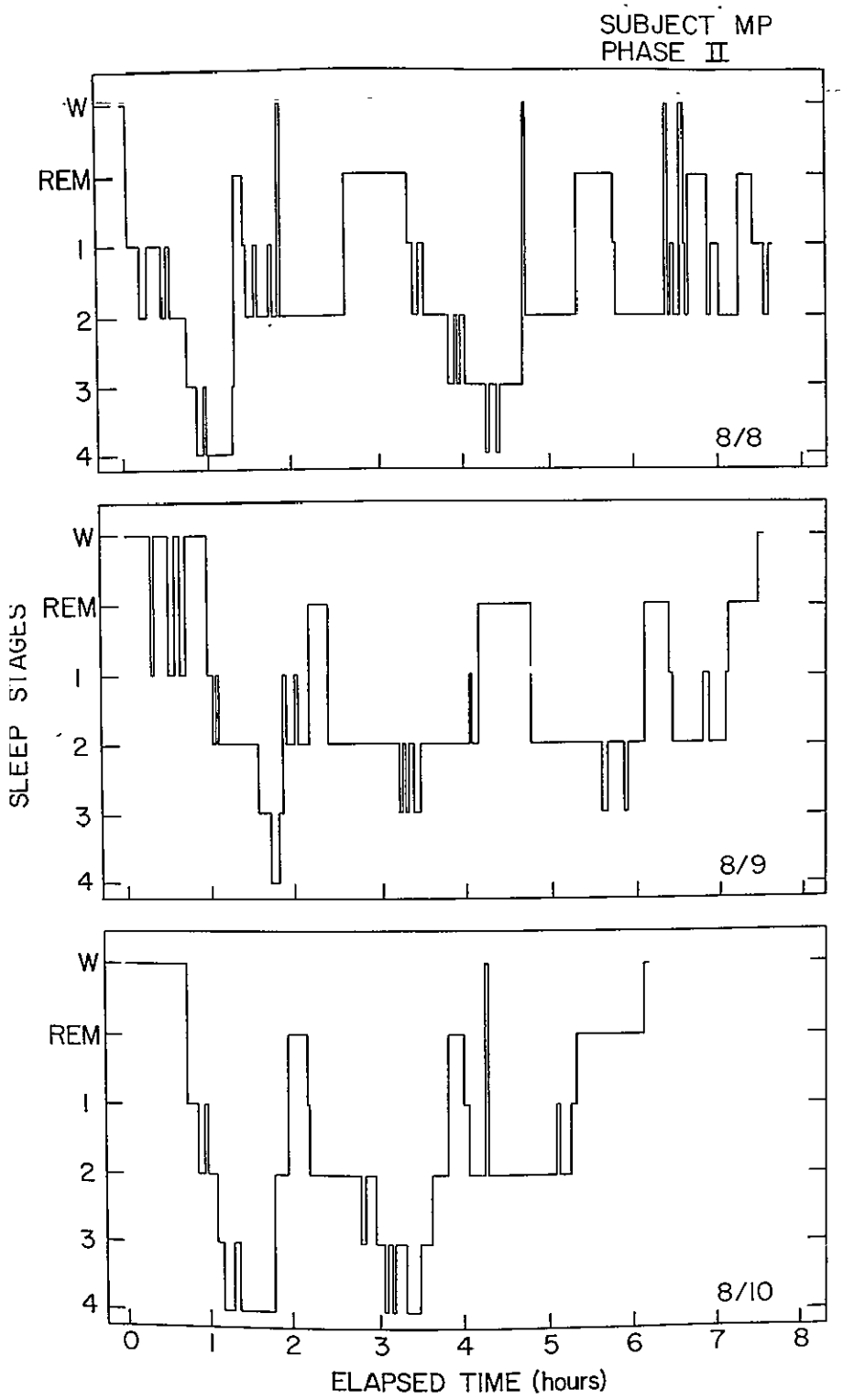

FIG. 5. Sleep profiles at sea level; subject MP, three consecutive nights.

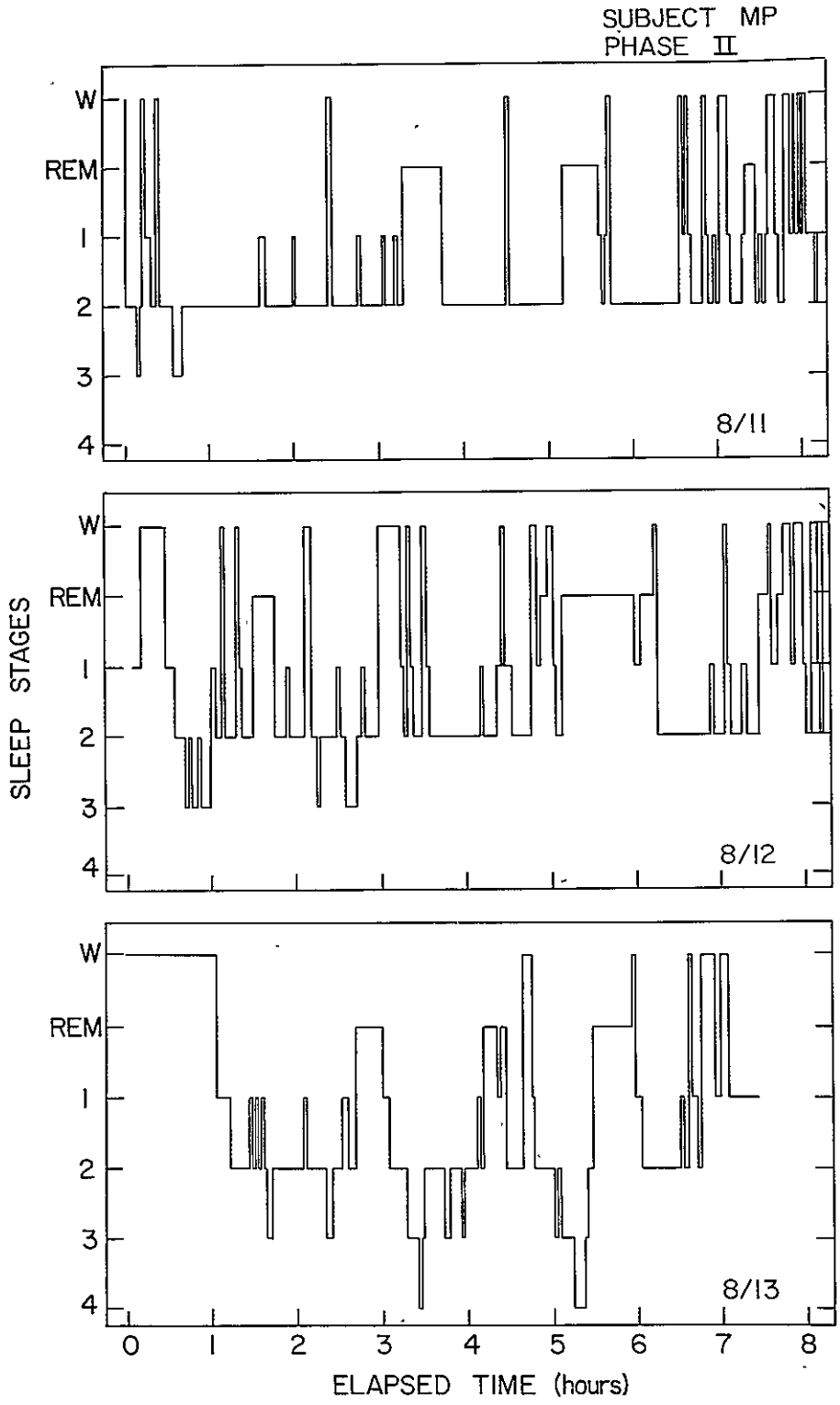

FIG. 6. Sleep profiles at 1200 fsw; subject MP, three consecutive nights.

point the lights were extinguished; Fig. 2 shows delta sleep percentages on consecutive nights. REM sleep percentages of both subjects were lower at 1200 fsw than at sea level, while percentages of waking time and stages 1 and 2 were somewhat higher.

Stages 3 and 4 sleep decreased for both subjects, with the largest change occurring in stage 4 for subject MP. On two of three experimental nights he showed virtually no stage 4 sleep and averaged 0.8% of total sleep time for the three nights, whereas his baseline stage 4 went as high as 13.3% for an average of 6.9%. Subject MP's stage 3 sleep also decreased from 8.8% to 5.3%. The stage 4 sleep decrease of subject CC was not as marked as that of MP, decreasing from 8.4% to 6.5%. His stage 3 sleep averaged 15.1% at sea level, 10.4% at 1200 fsw.

Subject MP's delta sleep was higher for two of his three control nights than for any night at 1200 fsw (15.7% average at sea level and 6.0% at 1200 fsw). Delta sleep (Table I, Fig. 2) for subject CC exceeded 20% on all three baseline nights, while his highest delta percentage at 1200 fsw was 17.7%. He averaged 23.5% delta sleep at sea level and 16.9% at 1200 fsw. Both CC and MP showed their lowest percentages of delta sleep during their first night at 1200 fsw and increasing percentages the two nights thereafter.

SLEEP PROFILES

Sleep stages plotted at two-minute intervals for each subject-night are shown in Figs. 3-6. Despite fluctuations in the total amounts of time accumulated in the various stages of sleep, the basic periodicity of the sleep patterns was little affected at 1200 fsw. This was especially true of CC; his rather abrupt early cycling into delta sleep on baseline nights at 1 ata (Fig. 3) occurred during the experimental nights at 1200 fsw as well (Fig. 4). His REM sleep patterns were also altered little at depth, except for an extremely long REM period during the second experimental night. His increased waking time at 1200 fsw was accompanied by longer initial onsets to sleep.

Although the sleep profiles for MP during the experimental period (Fig. 6) are not as stable--primarily because of the increased incidence and duration of waking episodes--the underlying periodicity, especially as mirrored in REM cycling, is still apparent. His descent into stage 4 sleep after the fifth hour on the third experimental night is somewhat unusual, but he does show some late delta sleep on the first and second nights of the baseline condition as well (Fig. 5).

SUBJECTIVE SLEEP LOGS

The subjective sleep logs supplement some aspects of the electrophysiological data. CC generally rated his three experimental nights of sleep as less sound than his baseline nights; this reflects the decrease in delta sleep and the increase in waking time. The only night to which he gave the maximum restfulness rating was the first control night, during which the incidence and duration of stage 4 sleep was highest. MP also reported his soundest sleep during the baseline period, particularly the second and third nights when awakenings were fewest. However, he claimed that his most restful night of sleep was the second experimental night when awakenings were extensive.

DISCUSSION

The Predictive Studies IV program provided the first opportunity to obtain sleep records over six consecutive nights which were equally divided into an initial normobaric control period and a later experimental period at 1200 fsw. Results show that delta sleep percentages decreased for both subjects CC and MP (Fig. 2) at 1200 fsw; this agrees with the single-night data obtained at 870-1000 fsw in previous exposures (1,13). Availability of the recordings at normal atmospheric pressure allows comparison with the circumstances of increased hydrostatic pressure and respiration of a helium-oxygen gas mixture, experimental variables not present during the control period. However, these were not in fact the only conditions altered, since differences in daytime activity levels and the psychological effects of being in a hazardous environment must be considered part of the overall change.

INDIVIDUAL SUBJECT CHARACTERISTICS

The normal sleep patterns of CC and MP were quite different. CC fell asleep almost immediately, descended quickly into stages 3 and 4 sleep, had few awakenings during the night and was difficult to arouse in the morning. This subject adjusted well to the recording conditions, since all three control nights showed similar sleep stage percentages, except for the long initial sleep onset on the third night, which immediately preceded compression to 1200 fsw. This suggests CC was bothered little by such things as the electrode array or the unfamiliar bunk arrangement.

On the other hand, MP had a delayed sleep onset, descended slowly and transiently into stages 3 and 4, spent more than half of the night in the light stages of 1 and 2, and generally awakened spontaneously in the morning. As might be expected, his first control night was atypical in that he awakened often in apparent discomfort, while his last two control nights were characterized by longer initial latencies to sleep, but fewer subsequent arousals.

Such marked individual differences emphasize the importance of having each subject serve as his own control. While both subjects showed similar decreases in their delta sleep percentages in the experimental condition, CC (the deep sleeper) actually accumulated a greater proportion of delta sleep at 1200 fsw than MP (the light sleeper) had at sea level.

INTERRELATIONSHIPS OF STAGES 3 AND 4 SLEEP

In contrast to previous sleep records at increased pressures, stage 4 sleep was present in both subjects on all nights when recordings were made. In fact, for each subject, at least one of his three experimental nights contained a larger percentage of stage 4 sleep than the lowest percentage of his three control nights. A similar inversion was found for MP's stage 3 sleep. However, average percentages of stages 3 and 4 sleep for the three-night periods of recording are consistently lower at 1200 fsw (Fig. 1). Combining stages 3 and 4 into the composite category of delta sleep shows a similar pattern for individual nights of recording (Fig. 2, Table I). With one

exception in MP, the percentage of delta sleep for each of the experimental nights at increased pressure is lower than that for any of the control nights. This similarity between the patterns of stage 3 and stage 4 sleep reduction at 1200 fsw indicates an absolute loss of delta sleep, rather than merely a redistribution of delta activity during the experimental condition.

Another interesting aspect of the delta sleep percentages was their progressive recovery toward normal over the three days at 1200 fsw for both subjects (Fig. 2, Table I). This apparent adaptation was particularly prominent in MP, who had 2.3%, 5.5% and 10.3% delta sleep for the three consecutive nights at pressure. The full extent of recovery is not known because sleep recordings were not made on subsequent nights.

EFFECTS ON OTHER SLEEP STAGES

Stages 3 and 4 sleep were not the only stages to be affected by exposure to increased pressures. Both subjects showed decrements in REM sleep at 1200 fsw as compared to their own normobaric controls, with parallel increments in stages 1, 2 and waking time (Fig. 1). The general association of delta sleep reductions with increased waking time and greater proportions of the drowsy stages of sleep could indicate that experimental conditions affected the overall quality of sleep rather than stages 3 and 4 specifically.

However, the association of delta sleep reductions with increased waking times does not occur consistently on a night-by-night basis. For CC (Fig. 2, Table I) the high percentage of delta sleep on his first control night was accompanied by a low amount of waking time, but his other two control nights and three experimental nights showed high variability of waking time in association with rather uniform quantities of delta sleep.

The general sleep pattern of MP is more inconsistent (Fig. 2, Table I). His highest percentage of delta sleep on the third control night was accompanied by his highest percentage of wakefulness during normobaric recording. In addition, his progressively increasing percentages of

delta sleep during the first three nights at 1200 fsw were also directly paralleled by increasing percentages of wakefulness. Therefore sleep patterns for individual nights are consistent with the specific effect of experimental conditions on delta sleep as opposed to a general effect on the quality of sleep.

COMPARISON WITH OTHER STUDIES

Table II summarizes data obtained from seven subjects in four different studies, including the two "sleep" subjects in Phase II of this program, Predictive Studies IV. Delta sleep percentages recorded at sea level and shallow depths are compared with similar recordings in the same subjects at depths ranging from 870 to 1600 fsw. In three of these studies (1,13) sleep patterns were recorded at 870 to 1000 fsw, and control records were obtained only subsequently, at 40 to 116 fsw, during decompression. The delta sleep percentages at 1200 fsw for the two men in the present study do not greatly differ from percentages obtained at 870 to 1000 fsw in previous studies. Similarly, there is no indication that the controls differed depending upon whether they were obtained at normobaric pressure breathing room air, or at increased pressure breathing helium-oxygen. The one major difference which is consistently seen is that the delta sleep percentages at ambient pressures equivalent to 870, 1000 and 1200 fsw are lower than their corresponding controls.

In contrast to the general agreement shown at these depths, sleep patterns monitored over a single night at 1600 fsw approached normal delta sleep percentages (13), rather than deviating further from the normal, as was expected. There is no explanation for this discrepancy; the single-night recordings at the highest pressure were the first for the subjects and were obtained under difficult experimental conditions. Such situations usually reduce delta sleep.

FACTORS CONTRIBUTING TO SLEEP PATTERN CHANGES AT HIGH PRESSURE

It is recognized that, in addition to increased hydrostatic pressure and the helium-oxygen breathing mixture,

general factors such as discomfort, the long and grueling experiment day, communication difficulty, and crowded physical conditions of the entrapment contributed to the stress and fatigue that were integral parts of the 1200-foot working exposure. Indeed such factors alone often result in significant reductions in stage 4 sleep (2). Long-term sleep experiments conducted at the South Pole (10) have shown that an exotic environment may have a similar effect. On the other hand, sleep EEG monitoring in three Skylab astronauts showed that the proportion of delta sleep during entrapment in a large spacecraft was nearly doubled in one and little changed in the other two (5).

While stresses were greater at the higher ambient pressure, the control recordings in Predictive Studies IV were taken during the final period of intensive preparation for the pressure exposures, on the three nights immediately preceding the compression. Therefore subjects CC and MP were never entirely removed from highly stressful, unusual conditions. If effects of helium or hydrostatic pressure are induced by the compressions employed they must be considered as contributing to the composite of all influences. At present they are not separable.

It has been reported that helium has excitatory effects on the nervous system of animals (4,14) and humans (17). EEG monitoring during three-hour naps in nine men who breathed either normoxic helium or normoxic nitrogen showed that the helium mixture was associated with a significant reduction in stage 4 sleep and an increased waking time, which together are consistent with a generalized arousal effect (17). Regardless of its effect upon the quality of sleep, however, helium per se does not appear to impair higher central nervous system functions. It is clearly not a depressant at the pressure studied here, since in the prior program (Predictive Studies III) it was found that complex mental functions were normal during prolonged exposure to 1200 fsw (6,8).

The data summarized in Table II (1,13) indicate that increased hydrostatic pressure probably contributes significantly to the observed reductions in delta sleep percentages at 870-1200 fsw, although there is no basis upon which to conjecture an actual mechanism. At present, the persistence or the rate and degree of adaptation during continuous sojourn at high pressure are not known.

TABLE II. Effect of Hydrostatic Pressure on Delta Sleep

Dive	Reference	Subject	Baseline		Increased Pressure[a]	
			Depth (fsw)	Delta Sleep (%)	Depth (fsw)	Delta Sleep (%)
U.S. Navy-Duke University, 1973	1	GB	54-116	15.3	870	4.7
U.S. Navy Experimental Diving Unit, 1973	13	PM	54-116	18.3	1000	3.8
		JR		26.7		15.0
U.S. Navy-Taylor, 1973	13	LR	40	23.7	1000	2.0
		CW		19.7		11.2
		LR			1600	11.8
		CW				13.7
University of Pennsylvania[b], 1975	This study	CC	0	23.5	1200	16.9
		MP		15.7		6.0

[a]Wilcoxon signed-rank test indicates that delta sleep percentages at pressures equivalent to 870-1200 fsw are significantly smaller ($p = 0.01$ for one-tail test) than those at sea level or shallow depths.

[b]Percentages are averages of three consecutive nights of sleep recording at these conditions.

It is likely that adaptation does occur. Since there is now no basis upon which to conjecture the mechanism of a hydrostatic pressure effect upon sleep, the pressure range over which the effect occurs as well as the rate and extent of any adaptation should be established to help clarify its origin. Related effects of hydrostatic pressure, such as the pressure-induced increment of slow brain wave activity in awake man and depression of cortical activity in animals (7), should also be explored in future experiments.

Acknowledgment

The authors express their gratitude to R. Gelfand for his extensive assistance in adaptation of the recording systems to the conditions of the experiments.

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E-12. ACUTE EFFECTS OF HYDROSTATIC PRESSURE ON PULMONARY FUNCTION

R.E. Peterson[1], C.J. Lambertsen[1], J.M. Clark[1] and R. Gelfand[1]

The results of this Institute's Predictive Studies III experiments, which compared effects of helium, nitrogen and neon as respiratory gases, indicated that pulmonary ventilatory functions were adequate not only at rest, but also during mild and moderate exercise even with gas densities to 25 g/l (equivalent to helium-oxygen breathing at 5000 fsw) (6). Measurement conditions in Predictive Studies III were, for reasons of experiment design, generated by administering gas mixtures of controlled density at actual ambient pressures to 1200 fsw (37 ata).

The results of the Predictive Studies III exposures are in sharp contradistinction to reports of incapacitating dyspnea during an exposure to helium-oxygen at 50 ata (pressure equivalent of 1600 fsw). In that study (18), subjects reported dyspnea during exercise at a lower work level and for shorter durations than were successfully completed in this laboratory at the helium-oxygen density equivalent of 5000 fsw (6).

In addition, other studies have reported greater decrements in ventilatory capacity with acute increases in respiratory gas density than were found in the more severe exposures of Predictive Studies III (5,9,12,13,17,20-22).

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It was considered that these two variances from the findings in Predictive Studies III might be related to 1) failures at 50 ata due to neuromuscular influences of the greater ambient hydrostatic pressure; and 2) progressive adaptations of pulmonary function during prolonged exposure to increased ambient pressures (6).

For these reasons, two major objectives of the present study related to pulmonary function were as follows: 1) investigation of acute effects of rapid compression to high pressure; and 2) tracking of changes during sustained exposure to high hydrostatic pressure. Therefore, pulmonary functions were measured immediately after completion of rapid compressions from 1 ata to 800 fsw, 1200 fsw and 1600 fsw, and also at intervals during the hours following arrival at the stable pressures of 800 fsw and 1200 fsw. This allowed study of the extent of changes in performance with time at depth and of the physiological bases for such changes. It was anticipated that the discomforts which accompany rapid compression to high pressure might exacerbate those pulmonary function decrements which are known to be due to increased respiratory gas density. It was therefore also part of the plan to correlate psychomotor effects of rapid compression (Section E-10) with any development and resolution of pulmonary mechanical deficits.

METHODS

EXPERIMENT CONDITIONS

The circumstances for pulmonary function measurements made in Phases I (at 0-800-1200-800 fsw) and II (0-800-1200-1600-1200 fsw) of this study are indicated in Tables I-IV. Measurements were made before and after each step in compression as well as after each excursion-decompression. Additional measurements in Phase II were made at 1200 fsw and 1400 fsw and during the course of the saturation-decompression. The Phase II measurements can be divided into two groups: 1) those made immediately after compression to new depths (including acute effects of compression); and 2) those made after several days at 1200 fsw, at 1400 fsw and during the saturation-decompressions (after the acute effects of compression had dissipated). The latter

group of measurements, presumed to be affected only by the inspired gas density, provided the necessary baseline against which to evaluate the immediate effects of compression.

SUBJECTIVE OBSERVATIONS CONCERNING PULMONARY FUNCTION

Section E-1 summarizes the symptoms and overt manifestations which resulted from the compression and pressure exposures. Comments pertaining to respiratory fatigue or dyspnea here are derived from the more complete summary in that section.

PHYSIOLOGICAL MEASUREMENTS

Experimental maneuvers were performed to evaluate three aspects of pulmonary mechanical performance:

Maximum breathing capacity. Estimated by measurement of forced vital capacity (FVC) and maximum voluntary ventilation (MVV), the maximum breathing capacity is indicative of short-term pulmonary function and exercise capability as limited by ventilatory capacity. The FVC maneuver is performed by inspiring fully and then expiring as forcefully as possible. The resulting peak instantaneous expiratory flow rate is the maximum expiratory flow rate ($\dot{V}_{E_{max}}$). Peak expiratory rather than peak inspiratory flow rate limits maximum voluntary ventilation and maximum breathing capacity when flow resistance increases in dense atmospheres (21). The MVV maneuver is performed by breathing cyclically as deeply and as rapidly as possible for a short period of time (in this case, 15 seconds). The resultant gas exchange rate is considered the upper boundary of ventilatory capability. Transpulmonary pressure was measured at the same time as MVV (in the more severe circumstances of Phase II only) to serve as an index of the subjects' inspiratory-expiratory efforts. This effort index (the peak-to-peak difference between average maximum inspiratory and expiratory pressures) was used to evaluate motivation- and motor-related effects on the test results.

Respiratory muscle strength index. This was measured (Phase II only) as the airway pressure at the mouth generated by voluntary maximum inspiratory and expiratory efforts

against the airway closed by an electrically activated shutter. Repeated measurements at a lung volume of approximately 40% were averaged. This index of respiratory muscle strength was obtained for correlation with other tests (MVV, $\dot{V}_{Emax}$) which are also related to muscle strength and coordination, and for comparison with results of other experiments which prominently involved strength or coordination (Sections E-5 and E-10).

Vital capacity. Vital capacity (VC) is the maximum volume of gas which a subject can inspire (or expire) in a single breath after a maximum expiration (or inspiration). This measure of lung volume has been found to increase during sustained exposure to high pressure (high respired gas density) (4,23).

APPARATUS

For the physiological studies cited, the physical parameters measured were ventilatory volume and instantaneous gas flow (Phases I and II) and transpulmonary pressure and mouth pressure (Phase II only). Volume and flow measurements were made with a "dry seal" spirometer (Ohio Medical model 840), calibrated for volume with a one-liter "standard" syringe. Flow calibrations were made by integrating the flow signal from a similar injection of gas and determining the coefficient necessary to make the integrated volume equal the injected volume. Pressure measurements were made with a differential pressure transducer (Statham P23Dd), calibrated with a water manometer. Transpulmonary pressure was monitored with an esophageal balloon attached to the transducer by seven feet of polyethylene tubing (i.d. = 0.047 inches). Pressure, flow and volume signals were sampled, digitized and stored on magnetic disk cartridges by a PDP-12 digital computer which was later used to process the data and prepare it for analysis.

SUBJECTS

Pulmonary function was studied in the "exercise subjects" (CC and WS in Phase I, CC and GM in Phase II). Planned measurements were completed by all subjects except WS who, due to an episode of decompression sickness, did not make excursions during the latter part of Phase I.

RESULTS

SUBJECTIVE OBSERVATIONS

During several of the exercise periods in the initial day of Phase II, subject CC made comments which were similar to previous reports of respiratory distress at depths of 1300 fsw or deeper (1, 18). These comments, which referred to the periods of bicycle ergometer exercise (Section E-14), rather than the pulmonary function tests, included: "not getting enough oxygen," "breathing too fast," "overbreathing the system," and "much resistance in the system." These statements were the strongest immediately after the initial compression from 0 to 1200 fsw, but were also made before and during the 1200- to 1600-fsw excursions on each of the next two days. Tendency to syncope or inability to continue during exercise were not mentioned at any time. Comments during the excursions were largely qualifications of the statements made during the initial compression; no indications of difficulty occurred at the stable saturation pressures.

The level of exercise (see Section E-14) was modest compared with the severe ergometer exercise tolerated by the subjects of Predictive Studies III while breathing more dense gases at 1200 fsw (6).

PULMONARY FUNCTION MEASUREMENTS

Values obtained from the pulmonary function measurements in Phases I and II are summarized in Tables I-IV.

Phase I

As expected, $\dot{V}_{E_{max}}$ and MVV were reduced in both subjects by the increased density of the inspired gas at 800 fsw and 1200 fsw (Figs. 1 and 2). Subject CC substantially increased his $\dot{V}_{E_{max}}$ and MVV toward normal at 800 fsw

TABLE I. Pulmonary Function During Exposures to Pressures Equivalent to 800 and 1200 fsw (Subject CC, Phase I)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Density (g/l)	MVV (l/min)	$\dot{V}_{E_{max}}$ (l/sec)	VC (l)
1	0	0	1.14	168	8.3	5.7
	800	1:54	5.12	77	4.1	-
	800	3:54	5.04	75	3.8	5.9
	800	7:00	4.71	78	4.0	5.8
2	800	0	4.53	94	5.6	6.0
	1200	1:08	6.44	79	4.5	-
	800	3:20	4.42	86	5.7	5.8
5	800	0	4.54	92	5.8	6.1
	1200	1:10	6.43	89	5.1	6.2
Post-exp.	0	-	1.15	174	8.2	6.1

TABLE II. Pulmonary Function During Exposures to Pressures Equivalent to 800 and 1200 fsw (Subject WS, Phase I)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Density (g/l)	MVV (l/min)	$\dot{V}_{E_{max}}$ (l/sec)	VC (l)
1	0	0	1.15	192	11.1	6.6
	800	2:03	5.07	94	4.6	6.5
	800	3:27	5.07	92	5.2	6.3
	800	6:27	4.69	98	5.2	6.3
2	800	0	4.70	94	5.1	6.5
	800	3:04	4.54	106	5.6	6.3
Post-exp.	0	-	1.15	181	10.7	6.0

TABLE III. Pulmonary Function During Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw and During Decompression (Subject CC, Phase II)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Density (g/l)	MVV (l/min)	MVV Effort Index (cm H_2O)	$\dot{V}_{E_{max}}$ (l/sec)	$P_{E_{max}}$ (cm H_2O)	$P_{I_{max}}$ (cm H_2O)	VC (l)
1	0	0	1.14	146	44	8.5	73.9	72.4	5.8
	800	1:15	5.06	78	17	3.8	58.2	49.0	5.7
	800	2:34	5.05	70	15	3.6	71.7	51.6	5.3
	1200	4:04	6.87	49	11	4.0	56.1	38.5	5.2
	1200	5:02	6.87	46	7	2.7	-	-	5.0
	1200	7:29	6.87	66	12	3.6	45.6	42.5	5.3
2	1200	0	6.61	69	30	3.7	71.7	65.5	5.8
	1600	0:47	8.55	62	16	3.1	54.8	34.2	5.7
	1200	3:16	6.53	74	-	4.2	87.5	50.1	5.6
3	1200	0	6.60	73	25	4.2	82.6	69.4	6.0
	1600	0:45	8.50	60	29	4.2	106.9	65.8	5.9
	1200	3:20	6.40	60	-	3.8	111.7	65.5	5.8
4	1200	-	6.60	72	30	3.7	155.3	75.5	6.0
7	1400	-	7.62	81	-	4.7	-	-	-
8	1200	-	6.92	77	-	4.1	102.5	94.6	6.2
9	1059	-	5.98	78	-	4.7	-	-	5.9
10	850	-	5.11	97	-	5.5	-	-	6.3
11	696	-	4.32	97	-	5.3	-	-	6.3
12	552	-	3.58	116	-	5.9	-	-	6.3
13	394	-	2.76	113	-	6.2	-	-	6.0
15	200	-	1.77	153	-	7.9	-	-	6.0
Post-exp.	0	-	1.15	155	49	8.1	-	-	5.6

TABLE IV. Pulmonary Function During Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw and During Decompression (Subject GM, Phase II)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Density (g/l)	MVV (l/min)	MVV Effort Index (cm H_2O)	$\dot{V}_{E_{max}}$ (l/sec)	$P_{E_{max}}$ (cm H_2O)	$P_{I_{max}}$ (cm H_2O)	VC (l)
1	0	0	1.14	160	30	11.2	189.2	111.4	5.4
	800	1:16	5.07	88	28	5.0	237.2	110.6	5.9
	800	2:32	5.06	108	30	6.3	220.3	115.5	5.8
	1200	5:14	6.87	72	-	4.2	214.5	101.8	6.0
	1200	6:38	6.88	71	-	4.2	201.8	101.1	5.9
	1200	7:58	6.88	80	-	4.7	195.7	82.3	5.8
2	1200	0	6.60	82	29	5.0	205.0	107.9	6.0
	1600	1:05	8.50	80	25	4.8	219.8	87.2	6.0
	1200	3:19	6.50	77	-	4.6	210.1	119.0	6.0
3	1200	0	6.59	84	24	4.8	208.5	119.2	6.0
	1600	0:45	8.51	74	24	4.5	209.9	118.0	6.2
	1200	2:39	6.49	78	-	5.3	165.1	95.5	6.2
4	1200	-	6.59	78	22	5.4	169.9	93.2	6.2
8	1200	-	6.60	85	-	5.0	198.2	91.1	6.4
11	1400	-	7.62	88	-	5.0	-	-	-
12	1200	-	6.92	83	-	4.9	-	-	6.5
13	1059	-	5.89	90	-	5.6	-	-	6.6
14	849	-	5.11	101	-	6.2	-	-	6.5
15	694	-	4.31	104	-	6.0	-	-	6.5
16	550	-	3.56	112	-	7.0	-	-	6.5
17	397	-	2.78	136	-	7.9	-	-	6.6
19	200	-	1.77	162	-	9.7	-	-	6.6
Post-exp.	0	-	1.15	169	52	9.8	150.8	110.5	6.1

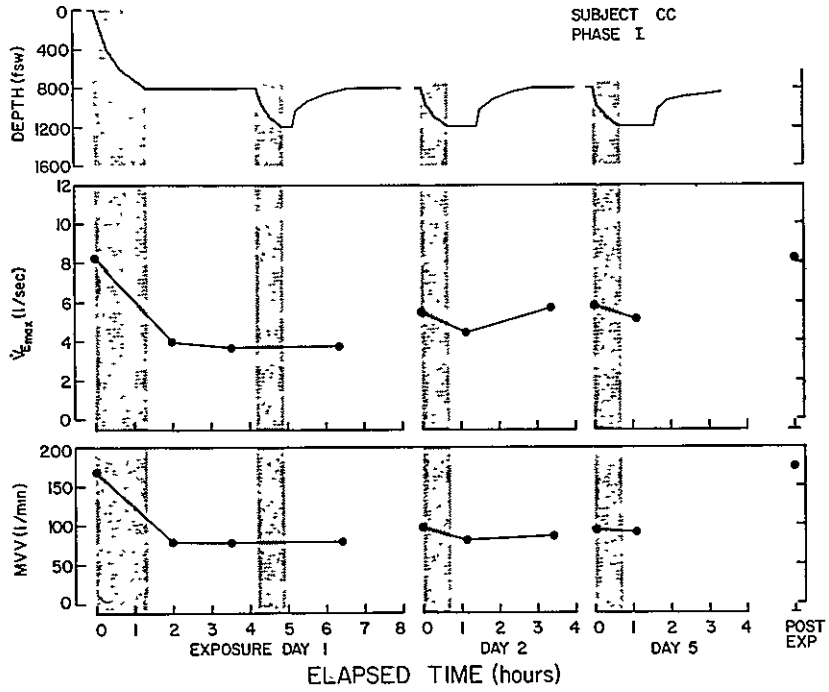

FIG. 1. Effects of compression-pressure-time exposures on maximum breathing capacity measurements.

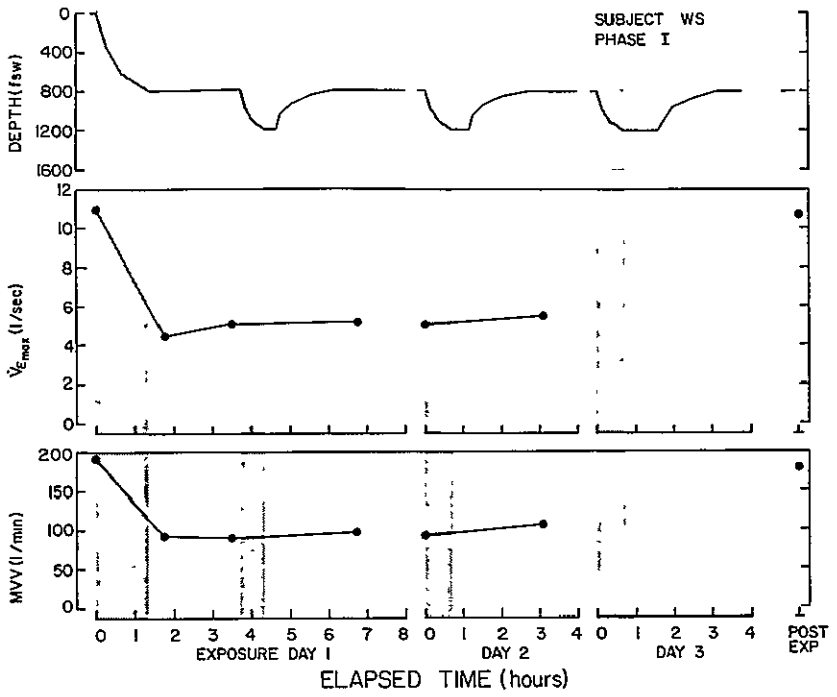

FIG. 2. Effects of compression-pressure-time exposures on maximum breathing capacity measurements.

over exposure days 1 to 2, and at 1200 fsw from day 2 to day 5 (Table I). He also had a small increase in VC (nearly 6%) over the course of the exposure while WS showed little change.

Phase II

Ventilation volume. Measurements of $\dot{V}E_{max}$ and MVV made after several days at 1200 fsw and during the decompression from saturation pressure are plotted against breathing gas density in Figs. 3 and 4, respectively. Performance decreased with increased density as expected from previous studies generally, and Predictive Studies III in particular. The curves on the graphs are power functions (2,14,20) calculated as the best fit to the data by a least squares criterion. The equations for these curves are also shown.

Since MVV and $\dot{V}E_{max}$ are functions of breathing gas density, this variable must be eliminated to examine the data for acute effects of compression and increased hydrostatic pressure. This was accomplished by using the continuous curves defined by the power functions which relate MVV and $\dot{V}E_{max}$ in prolonged exposure to increased gas density as reference levels for pulmonary performance. Actual measurements of $\dot{V}E_{max}$ and MVV obtained during the acute exposures to compression and pressure are expressed as deviations from this performance reference for each appropriate breathing gas density and are plotted against exposure duration in Figs. 5 and 6 for subjects CC and GM, respectively. "Respiratory efforts" during the MVV maneuvers (differences between average maximum inspiratory and expiratory pressures) are also shown.

Most MVV and $\dot{V}E_{max}$ performances of both subjects were somewhat below the density reference levels, particularly following initial compression from the surface to 1200 fsw. Subject CC's respiratory effort decreased markedly on exposure day 1 and, while it recovered substantially on succeeding days, it remained below his established capability for the first four days of the pressure exposure. GM's

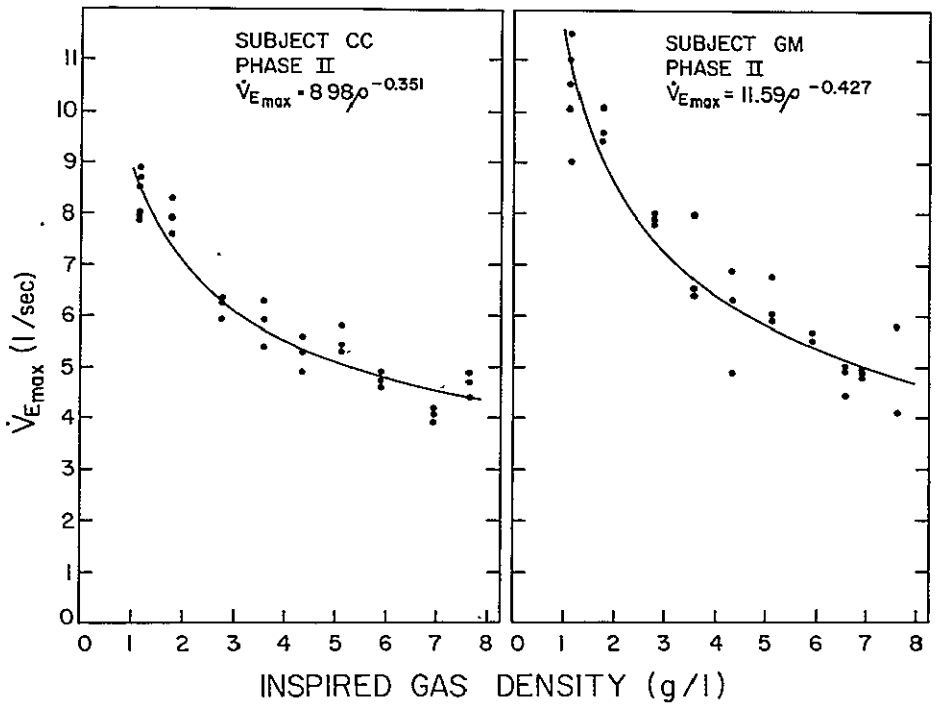

FIG. 3. Maximum expiratory flow rate as a function of inspired gas density measured after several days at high pressure and during decompression.

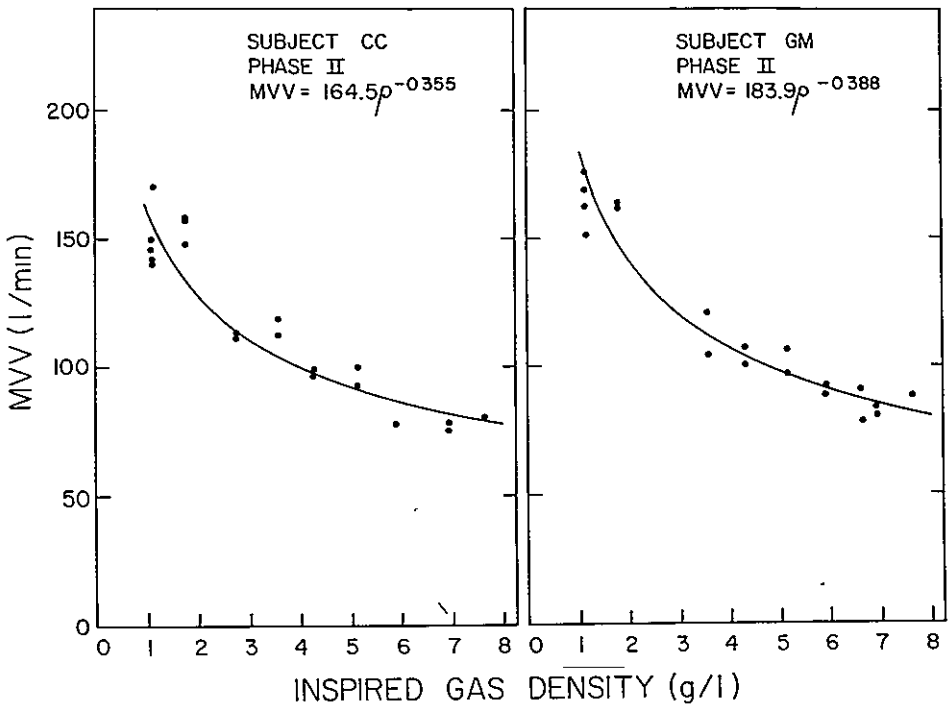

FIG. 4. Maximum voluntary ventilation as a function of inspired gas density measured after several days at high pressure and during decompression.

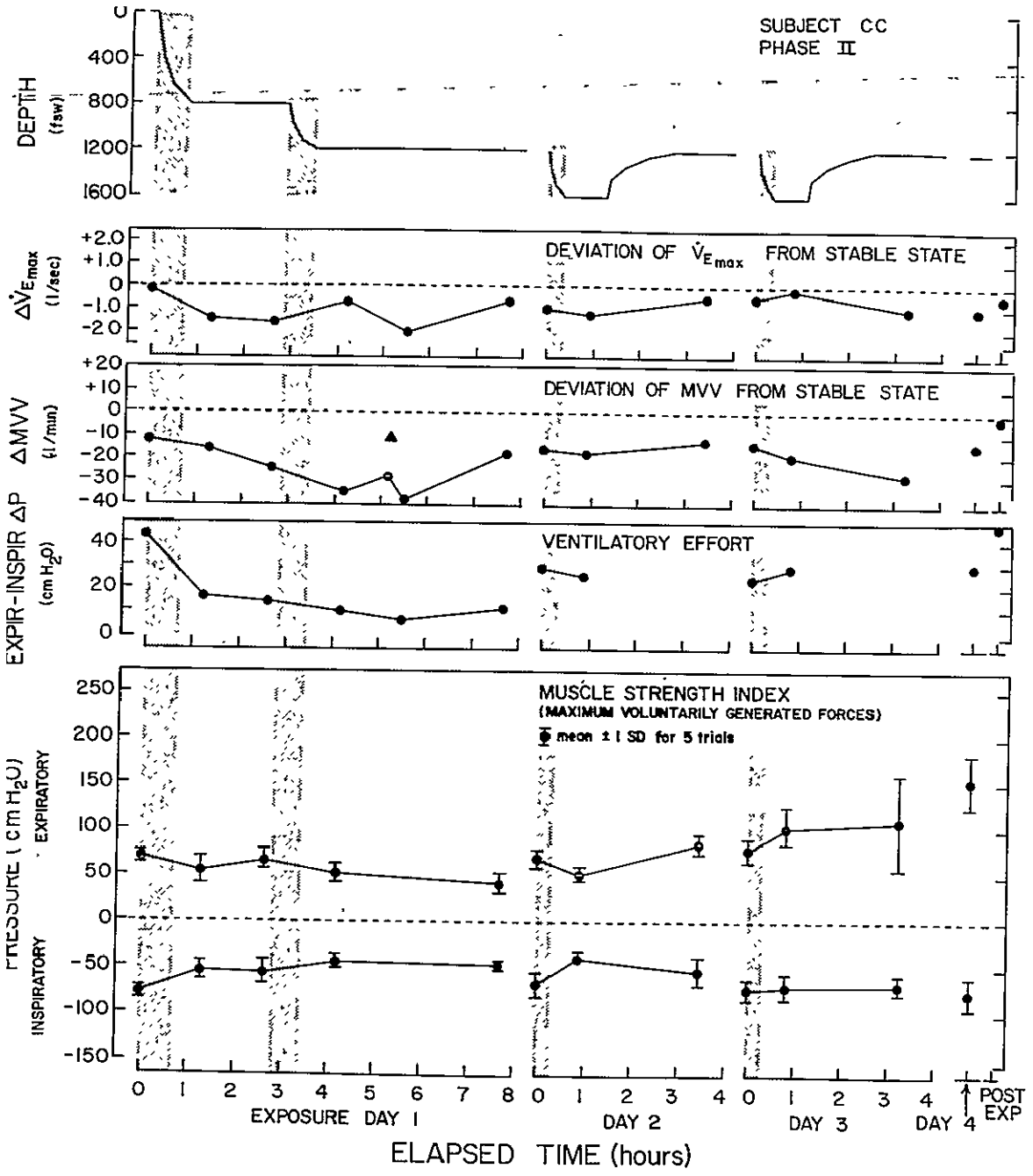

FIG. 5. Maximum breathing capacity, effort index and respiratory muscle strength index measurements during compression-pressure-time exposures. MVV and $\dot{V}_{E_{max}}$ are shown as deviations from their respective performance references. ▲ = extra data point (see text).

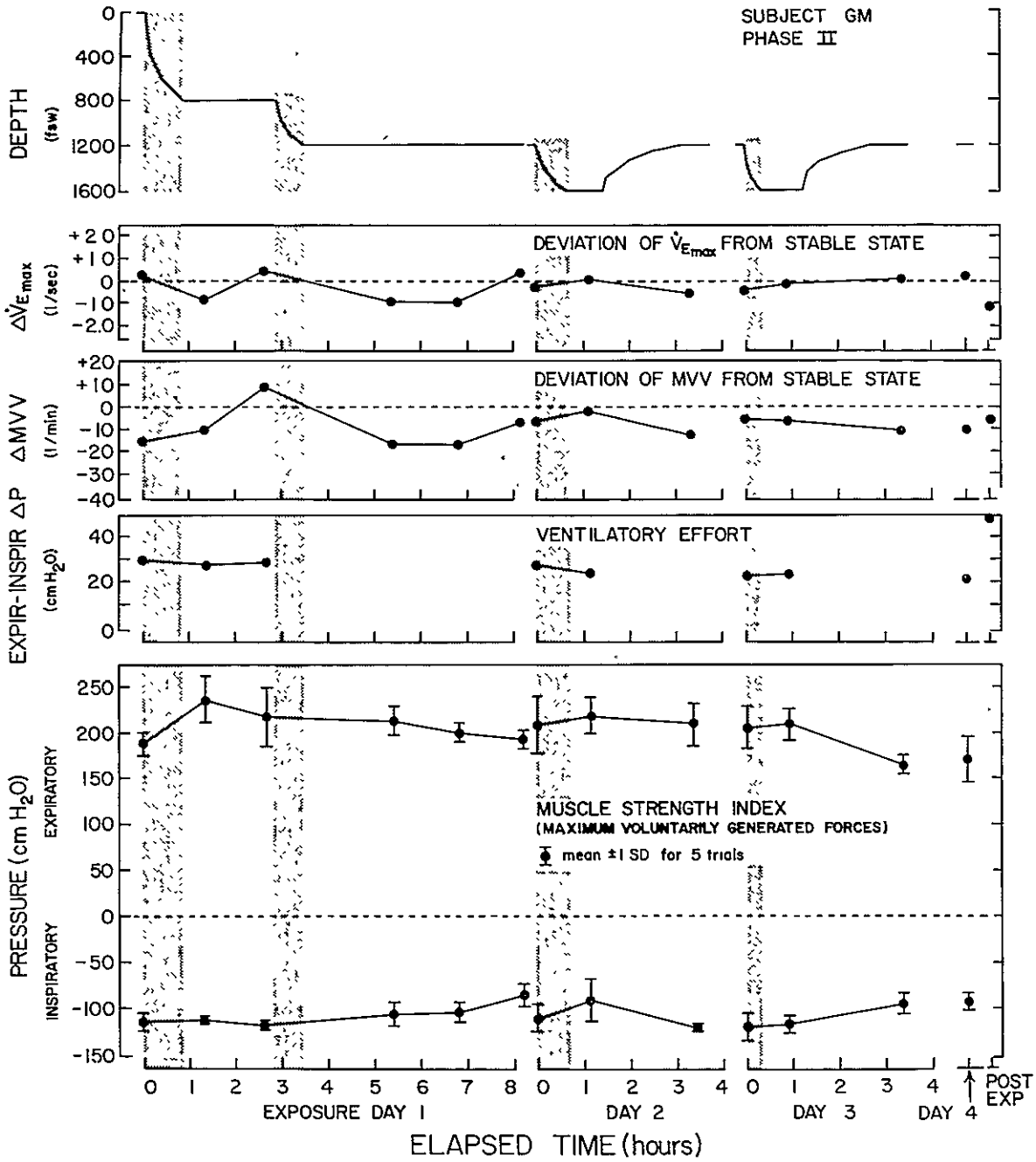

FIG. 6. Maximum breathing capacity, effort index and respiratory muscle strength measurements during compression-pressure-time exposures. MVV and $\dot{V}_{E_{max}}$ are shown as deviations from their respective performance references.

respiratory effort was not markedly affected by the compression and pressure exposures to 800 fsw. Due to nausea and vomiting soon after initial arrival at 1200 fsw, however, the esophageal balloon was removed and further measurements of respiratory effort could not be made on his first exposure day at 1200 fsw.

Muscle strength. Measurements of the respiratory muscle strength index are plotted in Figs. 5 and 6. The values for subject CC (Fig. 5), including his pre-exposure control values obtained just prior to compression, fell below his normal sea level performance range (106.25 ± 5.44 cm H_2O for expiratory effort; 90.30 ± 4.52 cm H_2O for inspiratory effort) but his performance returned to a normal range on day 4. Compared with his pre-exposure control values, CC's performance declined gradually during exposure day 1, was similar at 1200 fsw on days 2 and 3, and decreased slightly on compression to 1600 fsw on day 2. The respiratory muscle strength index improved on day 4, as cited above.

Subject GM had no significant changes in this index with two exceptions (Fig. 6). One was an increased expiratory effort on exposure day 1 with a gradual return to control later that day. The second exception occurred after the excursion-decompression on day 3 when both expiratory and inspiratory efforts were decreased.

Vital capacity. Vital capacity measurements for subjects CC and GM in Phase II show that CC had an approximately 15% increase at 800 fsw over exposure days 1 through 10, while GM had an approximately 13% increase over exposure days 1 through 17 (Tables III and IV). The vital capacity of subject CC at sea level following decompression was comparable to his pre-exposure control, while that of GM was above the pre-exposure level.

DISCUSSION

Particular interests in this study were 1) the relation of any possible subjective sensations of respiratory distress during compression to objective measures of pulmonary function; and 2) comparison of objective changes in pulmonary function on initial compression with effects of prolonged exposure to high helium pressures. Since only three

subjects could practically be investigated in the combined studies of Phase I and Phase II, analysis of findings is descriptive rather than primarily statistical and is aimed at elaborating related features of observations. Factors considered in interpretation of results included: ability to exert force in inspiration and expiration; ability to move gas through the pulmonary airways (maximum expiratory flow, maximum voluntary ventilation); ability to change lung volume (vital capacity); and subjective or objective indices of well-being.

RESPIRATORY MUSCLE STRENGTH INDEX

Measurements of maximum inspiratory and expiratory pressures which could be developed against an occluded airway were used as indices of muscle strength. The maneuvers were performed at close to mid-lung volume (ca. 40% of VC) to avoid a mechanical advantage for either the inspiratory or expiratory muscles due to muscle elongation at high or low lung volumes, respectively (16). Use of an intermediate lung volume also minimized measurement artifacts related to expansion and compression of lung gas during the development of large negative and positive intrapulmonary pressures.

Subject CC generated considerably lower pressures against the closed airway than did subject GM, even at 1.0 ata before compression. His performance actually improved considerably during the exposure to pressure, probably due to his learning ways of more effectively performing the maneuver rather than an actual increase in muscle strength. Both subjects showed a decline in respiratory muscle strength index over the course of exposure day 1. Pre-excursion values at 1200 fsw on exposure days 2 and 3 for both subjects show complete recovery from the decrements at the end of day 1. It is likely that general fatigue and discomforts associated with compression to high pressure (Section E-1) contributed to the gradual reduction in muscle strength index on the first day. The lack of consistent or large changes associated with compression per se indicates that

respiratory muscle competence is largely maintained in the face of increases in hydrostatic pressure.

Prolonged breathing of helium with density approximately seven times that of air at sea level did not condition the respiratory muscles to produce increased force, nor did the chronic exposure to pressure cause failure.

MAXIMUM EXPIRATORY FLOW

During the periods of actual compression, two stresses were generated which could modify maximal expiratory flow. These were a progressively increasing respiratory gas density, and any influence of hydrostatic pressure itself on respiratory function. The intent in this study was to use the former (which is better documented) as the baseline against which to study the latter.

Density Influence

It is known that as breathing gas density increases, the greater pressure losses from increased turbulent flow (10) and convective acceleration (7,21) cause a proportional increment in airway resistance to respiratory gas flow (3,8-10,19). During forced expirations, airway compression may still further limit $\dot{V}_{E_{max}}$ (11,15). Although by that effect it is therefore partially independent of expiratory effort, $\dot{V}_{E_{max}}$ is also limited by physical parameters such as airway resistance upstream of the compressed segment, lung and airway compliance, and gas density itself. With increased ambient pressure, expiratory flow and thus attainable ventilation levels are reduced (21). This was found in the present study as well.

Theoretical considerations of the relationships between density and the factors affecting airway resistance have led some investigators to suggest that $\dot{V}_{E_{max}}$ should decrease with increased gas density according to the equation $\dot{V}_{E_{max}} = A\rho^B$ where ρ is gas density and B (the density exponent) is between -0.500 and -0.428 (21). A number of studies have confirmed this finding (5,9,12,13,17,20-22).

Other studies, however, have shown that $\dot{V}_{E_{max}}$ is reduced less than predicted when measurements are made after an extended stay at depth (2,14) or improved with time when repeated measurements were made after compression (5,17).

Consistent with the latter findings, the stable-state measurements in Phase II (Fig. 3) show that the density exponent for subject CC (-0.351) was closer to zero than -0.428, while the exponent for subject GM (-0.427) was at the the low end of the expected range (density exponents closer to zero indicate less decrement as gas density increases). The lowest values of $\dot{V}_{E_{max}}$ in Phase I were obtained after rapid compression on exposure day 1, with improvement either later that day or on the next day (Figs. 1 and 2). Similarly, in Phase II, most $\dot{V}_{E_{max}}$ values obtained after rapid compression from 1 ata on the first exposure day were below the stable-state reference, but they returned to or near this level on day 2 for GM and day 3 for CC (Figs. 5 and 6). These observations indicate that some factor related to compression itself or to initial exposure to increased density appears to be responsible for transient reduction in ventilatory performance beyond that to be expected from stable exposure to increased density.

MAXIMUM VOLUNTARY VENTILATION

Because MVV is a function of expiratory flow rate, any factors affecting $\dot{V}_{E_{max}}$ affect MVV in the same manner. Both of the stable-state MVV-density equations have exponents substantially less negative than -0.428 (Fig. 4). The excursion measurements of Phase II showed their greatest decrements following the initial compressions to 1200 fsw, and performance improved with increased exposure time (Figs. 5 and 6).

VITAL CAPACITY

Two of the three subjects (CC and GM) in this study had increases in VC during the course of these exposures. Such increases associated with prolonged exposure to elevated pressures and gas densities have been observed in several other studies (4,23) and have always been accompanied by increases in the transpulmonary pressure at total lung capacity. For this reason it has been presumed that the elevation in VC was due to an increase in respiratory muscle strength in response

to increased work of breathing high density gas. However, in these studies the index of muscle strength measured in CC and GM was not appreciably increased (Figs. 5 and 6), despite the fact that the breathing gas density was transiently greater than in the previously cited studies (4,23). Some mechanism other than or in addition to muscular conditioning may be responsible for the changes observed previously.

ACCLIMATIZATION-RELATED PERFORMANCE IMPROVEMENTS

The data for exposure day 1 in Fig. 5 may help to explain the time-related performance improvement found in pulmonary function after acute pressure exposures in this and other studies. The effort index shows that the poorest performances during the MVV maneuvers obtained in the measurement modules (Section D) occurred at times when the peak dynamic expiratory and inspiratory pressures were substantially below the pre-compression level. However, the decrements in CC's respiratory muscle strength measurements were concurrently relatively minor. That dynamic muscle function as well as muscle strength was in fact not seriously affected at these times was demonstrated by the extra data point (▲) shown in Fig. 5, obtained when CC was requested to perform an additional MVV maneuver for photographic purposes. This extra maneuver resulted in an MVV at the pre-compression level. The reduced performance during the modules must therefore have been related to reduced effort.

The bases for a reduced effort were probably complex and not specifically related to neuromuscular function. The poorest performances of pulmonary function were concurrent with symptoms such as nausea, vomiting and general fatigue (Sections E-1 and E-5). It is significant that these feelings of malaise were also associated with decrements in other performance measures which are motivation- and attention-dependent (Sections E-8 and E-10).

SYMPTOMS OF RESPIRATORY DISTRESS AND RELATION TO PULMONARY CAPABILITY

Reports of respiratory distress in other studies at depths between 1300 and 1600 fsw (1,18) were considered significant since they implied that effects not related to gas density might limit helium-oxygen diving to depths much less than limits based on considerations of density alone (6).

Subject CC was the only one who participated in both phases and the only one reporting symptoms of respiratory discomfort (in Phase II). In Phase I he reported no symptoms, and his $\dot{V}_{E_{max}}$ and MVV on day 1 were reduced in about the same proportions as were those of subject WS. Recovery on subsequent days was more complete for CC than for WS (Figs. 1 and 2).

In Phase II, when CC reported symptoms, his acute reductions in $\dot{V}_{E_{max}}$ and MVV were more marked than those of GM, and his ventilatory effort was considerably reduced below the pre-compression level (Figs. 5 and 6). Recovery of $\dot{V}_{E_{max}}$ and MVV on subsequent days was slower for CC than for GM; however, CC's subsequent measurements of $\dot{V}_{E_{max}}$ and MVV were less affected by density (Figs. 3 and 4).

Despite the acute decrements in pulmonary functions which accompanied the symptoms of compression and pressure, CC's ventilatory capability was more than adequate to sustain his ongoing exercise level and there was no evidence of hypercapnia (Section E-14). Furthermore, diaphragmatic electromyograms during rest and exercise showed only normal patterns of electrical activity (Section E-14). Thus, the actual causes for CC's comments relating to respiratory awareness did not originate from derangements in ventilation or gas exchange. The fact that symptoms were also reported on days 2 and 3 of Phase II, after substantial or complete recovery from general malaise and from acute pulmonary function decrements, further suggests a non-ventilatory cause. It is most significant that, in spite of his earlier discomfort, CC's exercise and work capabilities were not compromised in the wet chamber during underwater work at 1600 fsw (Section F).

SUMMARY

Rapid compressions to 800-1200-1600 fsw caused transient decrements in ventilatory performance, in addition to those expected to be related to effects of increased gas density alone. The decrements were most marked when symptoms and manifestations of compression and pressure were prominent, and they receded with time as the subjects adapted to their environment and disturbing symptoms diminished. There was no indication that these transient decrements were due to specific derangements of the respiratory control system. They were in part effort-dependent.

Ventilatory levels in normoxic helium-oxygen were adequate to support the programmed exercise immediately following rapid compressions from 1200 to 1600 fsw.

Pulmonary function measurements after several days at 1200 fsw and during decompression were similar to those measured under equivalent conditions in an earlier study (6).

Subjective sensations of respiratory distress were reported by one subject during exercise at a time when his ventilatory capability was more than adequate to sustain the ongoing exercise level. These sensations did not appear to have a physiological basis related to the mechanical functions of the lung.

Acknowledgments

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E-13. CARDIAC ELECTRICAL AND MECHANICAL FUNCTION

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and C.J. Lambertsen[1]

No clear indication of a "high pressure nervous syndrome" was found during a slow stepwise compression to the pressure equivalent of 1200 fsw (18). However, effects of exposure to rapid compression rates and higher hydrostatic pressures on excitable tissues are apparently responsible for signs such as tremor, muscle fasciculations, electroencephalographic changes and inattentiveness, and symptoms such as nausea and dizziness (4,6,7,9,13,15,16,21,23,34,36) (Section E-1). It was considered that since normal cardiovascular activity depends on the functional integrity of electrically active pacemaker cells and of neuromuscular structures of the myocardium, and on normal arterial vasomotor tone, hydrostatic influences on any of these could alter cardiac function. If cardiovascular function were to be adversely affected by compression-pressure exposures, which is known to occur in animals at extreme pressures (19), the reduced perfusion of other excitable tissues (central or peripheral) might result in localized hypoxia and acidemia. Such influences might exaggerate direct effects of compression and pressure, intensifying the signs and symptoms of such exposures.

Exposures to increased hydrostatic pressure and gas density in helium-oxygen atmospheres have been reported to produce bradycardia (12,14,20,21,24,26,28), even when the effects of hyperoxia and nitrogen narcosis (11) were not factors. This has been reported for conditions of rest (12,14,20,21,26,28) and exercise (12,14,20,26,28). While heart rate has been measured in man during many exposures

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to increased pressure, other indices of cardiac function such as the timing of cardiac events (29), cardiac output (33) and blood pressure (21,33) have only occasionally been monitored.

Measurements of cardiac electrical and mechanical functions were included in Predictive Studies IV as part of the study of possible compression and hydrostatic effects on the peripheral and central nervous systems, on muscle and on autonomic functions. While the importance of measuring blood pressure was recognized, it was not practical to do so without interfering with the numerous other experiments of this integrated study. Electrocardiographic recordings and measurements of cardiac stroke volume were obtained before, during and upon completion of rapid compressions to pressure equivalents of 400-800-1200-1600 fsw. Measurements were taken at rest early in the morning and during actual compressions as part of the experiment modules. They were made before and after a postural change as well as before, during and after recovery from mild exercise.

MATERIALS AND METHODS

APPARATUS

The electrocardiogram (ECG) was recorded using a modified five-electrode configuration, with placement on the upper arms and thighs to avoid disturbing other measurements, to promote mobility and to reduce motion artifacts. Either of two chest leads, placed in the V_1 and V_5 positions, was selected by operation of a toggle switch. Disposable electrodes ("Longlife Electrodes," American Hospital Supply) were employed with a clinical ECG machine (Hewlett Packard, model 1500A). ECG records were examined for pathological effects and for physiological changes including atrial conduction time (PR interval), cardiac ejection time (QT interval) and cardiac rate.

Cardiac stroke volume was obtained from tracings of an electrical impedance cardiograph (Instrumentation for Medicine, Inc., model 304A, Minnesota impedance cardiograph) recorded with an oscillographic recorder with frequency response to 100 Hz (Honeywell, model 906B). ECG electrodes and conductive tape bands for the cardiograph (Instrumentation for Medicine, Inc., cardiograph electrode tape) were

applied by the subjects themselves every morning. Each was trained to position the electrode bands at predetermined locations in an attempt to achieve day-to-day reproducibility of the measurements (10,17). The electrode bands remained in place throughout a given experiment day. Special, custom-made, form-fitting vests, constructed with the cardiograph electrode bands in fixed positions, were used for the early morning measurements.

Cardiac output was recorded while the subjects breathed spontaneously at rest and during exercise on an electrically controlled bicycle ergometer (Collins Pedal-Mode). Measurements could not be taken at maintained end-expiration (10) because of disturbance to concurrent experiments. As a result, not all recordings obtained during exercise could be analyzed.

Electrical connections to the electrodes formed part of the subjects' harnesses (Section D). The same two instruments (electrocardiograph and impedance cardiograph) were used for both the rest and exercise subjects. Encapsulated reed relays, located inside the chamber but under the control of investigators outside the chamber, were used to transfer the electrical connections from the instruments to a particular subject, as required.

EXPERIMENT PROCEDURES

Exercise Tolerance Measurements

Prior to the compression-pressure exposures a single exercise tolerance experiment was performed by each subject to establish his physiological responses to graded levels of exercise (Section E-14). These data provided the reference against which heart rate and cardiac output changes obtained during the compression-pressure exposures could be compared.

Measurements During Compression-Pressure Exposures

Two modules (control, compression) define the experiment procedures for Phase I, while a single module defines procedures for Phase II (Section D). The time-activity schedules of the rest and exercise subjects, specifically as

they relate to the cardiovascular measurements, are shown in Figs. 1A and 1B for Phase I and in Fig. 1C for Phase II. Since the subjects did not need to actively participate in the ECG and impedance cardiograph measurements, they could take part in concurrent experiments.

Two categories of measurements were performed in the rest subjects. The first was done while the subject was seated and engaged in tasks requiring light activity, including audiometry; perceptual, memory, cognitive and psychomotor tests; measurements of arm and hand tremor; ocular fixation and eye tracking; and visual evoked responses (Fig. 1; Sections E-3,4,7,8 and 10). The second involved measurements taken during the first two minutes after rising from the sitting position to stand quietly on the statometer (balance platform) for a quantitative determination of body sway (Section E-6). This postural change provided an opportunity to test the baroreceptor reflex pathway.

Measurements of heart rate and cardiac output before standing were averaged to estimate the overall means in the seated subjects. During the control modules in Phase I (Fig. 1A) these measurements were collected within the 15-minute period which preceded the subject's assumption of the standing position. During the compression modules of Phase I (Fig. 1B) they were spread over 10 minutes. In Phase II (Fig. 1C) the measurements were taken during an eight-minute interval while the subjects were seated.

Three categories of measurement were obtained from the exercise subjects (Fig. 1). The first recordings were made with the subject seated on the bicycle ergometer, before starting exercise, but while engaged in tasks requiring light activity such as measurements of tremor, and perceptual, memory, cognitive and psychomotor functions (Sections E-4 and E-10). The second category of measurements was obtained during exercise, and the third during recovery from exercise (exercise was not performed during or after an excursion-decompression). All pre-exercise measurements were averaged to provide the best available estimate of cardiovascular parameters for these subjects in their rest state. These measurements extended over five minutes in Phase I and 7 minutes in Phase II (Fig. 1). Rest and exercise values of $\dot{V}_{O_2}$ were obtained as an average over the two

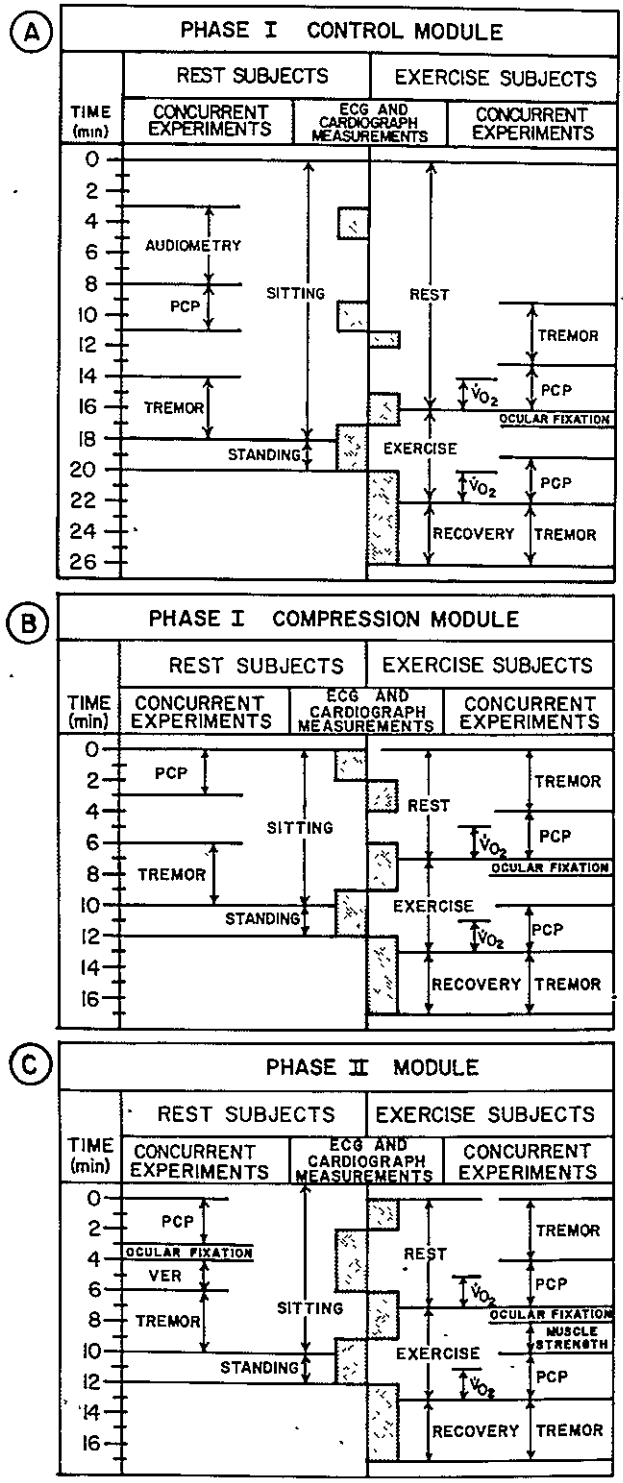


FIG. 1. Timing of cardiac function measurements in the experiment modules. ECG and impedance cardiograph measurement intervals are shown as shaded rectangles in the center of the diagrams. The timing schedule of concurrent experiments is shown at the left for the rest subjects, at the right for the exercise subjects. A, Phase I control module; B, Phase I compression module; C, Phase II module.

minutes immediately preceding exercise, and over the final two minutes of exercise (Fig. 1; Section E-14).

Normoxic levels of inspired (chamber) oxygen (160-175 mm Hg) were maintained during compressions and at stable high pressures. Slightly enriched oxygen levels were used during excursion-decompressions (Section G-1) and during saturation-decompressions (Section G-2).

Early Morning Measurements

Measurements were made directly after the subjects awakened, before breakfast and after only minimal activity, while they were sitting on the bicycle ergometer (Section E-15).

Data Analysis

Heart rates were calculated from the ECG and impedance cardiograph tracings. The ECG tracings also yielded atrial conduction time and cardiac ejection time. The latter data in the exercise subjects were obtainable from tracings recorded only while they were not pedaling, due to baseline shifts during exercise. Stroke volume was extracted from the impedance cardiograph tracings and multiplied by heart rate to calculate cardiac output. Each measure of stroke volume was obtained as the average of all stroke volumes during 3 or 4 respiratory cycles (10 to 25 cardiac cycles).

RESULTS

EXERCISE TOLERANCE MEASUREMENTS

Table I presents pre-exposure values of oxygen consumption, heart rate and cardiac output during progressively increasing exercise levels for five of the six subjects. The exercise series on subject LJ was incomplete and his results are therefore not included. Subject GM had an unusually high heart rate at rest, and therefore the data are plotted both as averages of five subjects (N = 5) and of four subjects (N = 4, GM excluded) in Fig. 2. The

TABLE I. Oxygen Uptake, Heart Rate and Cardiac Output During Progressive Exercise Levels at 1 ata

Subject	Workload (kpm/min)					
	0	300	600	900	1200	1500
Oxygen Uptake (1/min, STPD)						
CC	0.43	1.03	1.44	2.06	3.05	4.15
GM	0.69	1.36	1.93	2.66	3.30	5.00
MP	0.27	1.04	1.58	2.41	3.17	4.13
FS	0.35	0.99	1.65	2.33	2.97	3.81
WS	0.31	0.96	1.43	2.02	2.98	3.85
Mean(N=5)	0.41	1.07	1.61	2.30	3.09	4.19
±SD	0.17	0.16	0.21	0.26	0.14	0.48
Mean(N=4)[a]	0.34	1.01	1.53	2.21	3.04	3.99
±SD	0.07	0.04	0.11	0.19	0.09	0.18
Heart Rate (beats/min)						
CC	86	101	110	151	166	186
GM	136	149	163	180	190	203
MP	82	97	114	148	167	188
FS	81	146	156	181	193	218
WS	74	93	104	151	182	190
Mean(N=5)	92	117	129	162	180	197
±SD	25	28	28	17	13	13
Mean(N=4)[a]	81	109	121	158	177	196
±SD	5	25	24	16	13	15
Cardiac Output (1/min)						
CC	8.0	13.6	15.9	20.4	20.8	24.8
GM	11.0	17.4	24.8	29.5	30.8	28.9
MP	6.1	8.3	10.4	15.7	19.0	21.4
FS	5.5	13.9	12.8	14.8	13.4	17.4
WS	7.3	11.1	13.5	16.7	27.3	-
Mean(N=5)	7.6	12.9	15.5	19.4	22.3	23.1
±SD	2.1	3.4	5.6	6.0	6.9	4.9
Mean(N=4)[a]	6.7	11.7	13.2	16.9	20.1	21.2
±SD	1.1	2.6	2.3	2.5	5.7	3.7

[a]Subject GM excluded.

nearly linear relationships between mean heart rate and oxygen consumption, and between mean cardiac output and oxygen consumption in Fig. 2 are typical (2,3,10).

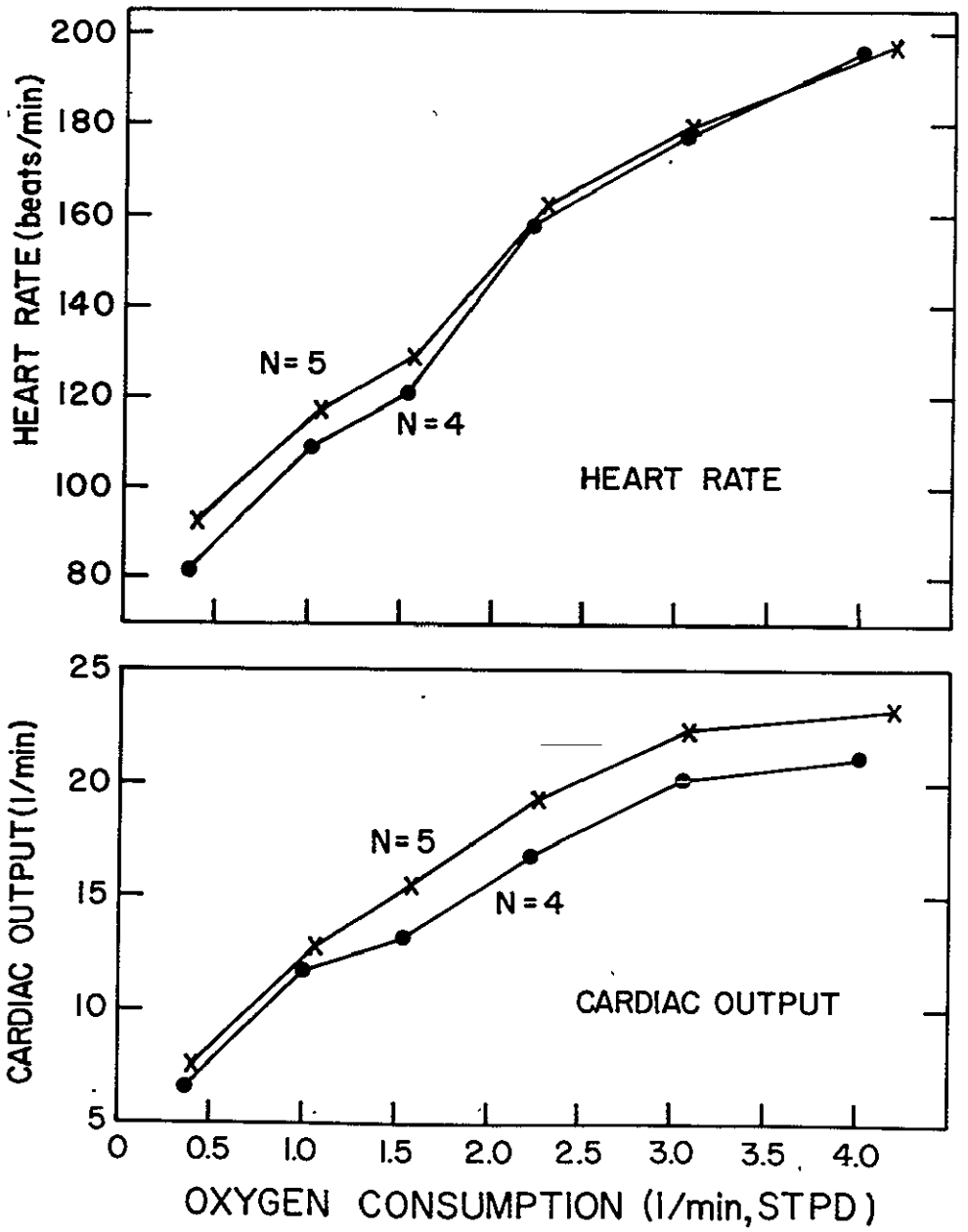


FIG. 2. Pre-exposure values of heart rate and cardiac output as functions of oxygen consumption during progressive levels of exercise. Mean values for five subjects (CC, GM, MP, FS, WS) and for four subjects (GM excluded, see text for explanation).

MEASUREMENTS DURING COMPRESSION AND AT STABLE INCREASED PRESSURE

Electrocardiographic Interpretations

The ECG records of Phase I could not be interpreted due to technical problems (which were resolved prior to Phase II), and consequently were used only for determination of heart rates. All ECG tracings from Phase II were interpreted as being normal and compatible with the clinical cardiac examinations obtained before and after the pressure exposures (Section D). No abnormalities in rhythm or pattern were seen in any subject at rest, during exercise, or during compressions and acute exposures to pressures of 800, 1200 or 1600 fsw, or throughout the full periods of exposure to these pressures.

Measurements While Seated: Exercise and Rest Subjects

Mean values of the parameters describing cardiac function for the four subjects of Phases I and II while seated are tabulated in Tables II and III (heart rate and cardiac output in Phase I [Table II]; timing of cardiac events, heart rate, and cardiac output in Phase II [Table III]). Individual values are given in Appendix Tables 1 and 2 (Phase I) and Appendix Tables 3-6 (Phase II). For the Appendix Tables, data represent the average of all measurements before starting exercise in each module (exercise subjects) and before rising from the seated position to stand on the statometer (rest subjects). Elapsed times in all tables are tabulated and data points in figures are plotted approximately midway in the measurement intervals. When times and depths were different for subject pairs, average values are shown.

Data for Phases I and II are plotted in Figs. 3 and 4, respectively, below compression-pressure-time exposure profiles to allow reference to exposure conditions. Mean early morning heart rates and cardiac outputs for four subjects (Tables XII and XIII) are also shown in Fig. 4. Reference lines are drawn through "normal" heart rates (basal and non-basal [1, p. 337]) and cardiac outputs (basal and while seated at rest [1, p. 327]).

Phase I. Mean heart rates and cardiac outputs are plotted only when N = 4 (Fig. 3, Table II). Other data (Appendix Tables 1 and 2), obtained only for two subjects, are plotted individually.

Cardiac Rate

The pre-compression mean heart rate of 75 beats/minute was slightly above the reference non-basal resting value. It slowed during the compression from 1 ata to the pressure equivalent of 800 fsw to a low of 66 beats/minute. Heart rate increased transiently to a peak of 77 beats/minute during the compression from 800 to 1200 fsw, then declined to about 65 beats/minute during decompression to 800 fsw. The mean heart rate preceding the excursion to 1200 fsw on exposure day 2 was 76 beats/minute, essentially the same as at 1 ata on day 1. During the excursion, the rate fell to approximately 72 beats/minute; it remained at that level throughout the balance of the excursion. Values for CC and LJ on exposure day 5 were in the range of mean values obtained on days 1 and 2. Mean heart rates during the exposure were close to normal, resting, non-basal values. The mean post-exposure value at 1 ata on the day following decompression was elevated when compared with other measurements, possibly because the subjects were not required to stay overnight in the pressure chambers following the late evening completion of decompression.

Cardiac Output

Mean cardiac output on exposure day 1 followed the same pattern of change as did heart rate (Fig. 3). On day 2, cardiac outputs for FS and LJ were relatively constant, while on day 5 cardiac outputs in CC and LJ were elevated before compression began. Mean cardiac output was equal to or greater than values cited for the seated (non-basal) position.

Phase II. All data plotted in Fig. 4 (from Table III) are means for N = 4 except cardiac output on exposure day 1, where N = 3. Other data which were not graphed are included in Appendix Tables 3-6.

Heart Rate

Mean early morning heart rate (Table XIII) on the first exposure day at 1 ata was at the normal basal reference level. The pre-compression 1 ata value was only slightly higher. Unlike Phase I, mean heart rate increased during the compression to 800 fsw. It then decreased to nearly the 1 ata value during the "hold" at 800 fsw, showed a slight, transient increase during compression to 1200 fsw, and subsequently over several hours at 1200 fsw declined to a rate slightly below that for early morning.

The mean early morning heart rate at 1200 fsw on exposure day 2 was the same as at 1 ata on day 1 (Table XIII). The pre-compression rate was close to average non-basal values cited for 1 ata. Compression to 1600 fsw elicited a small, transient increase. As time progressed, the heart rate declined to basal reference values (while at 1600 fsw, during and following decompression to 1200 fsw). The pre-compression heart rate on exposure day 3 was also at the resting, non-basal reference level. Compression to 1600 fsw elicited a larger increase in heart rate than on day 2, followed by a progressive decline as on day 2. Mean early morning heart rate on the eighth exposure day at the stable pressure equivalent of 1200 fsw was close to a basal value, while mean heart rate during performance of the experiment module on that day was slightly higher. Post-exposure mean rate was higher than corresponding values on day 8.

Cardiac Output

The patterns of change in mean cardiac output were similar to those of mean heart rate on all three days on which compressions occurred (Fig. 4). The mean early morning cardiac output on day 1 at 1 ata was between the basal and seated resting reference values, but was elevated on day 2 at 1200 fsw. Most cardiac output values while the subjects were seated during the experiment modules were higher than reference levels cited for seated subjects (1, p. 327).

The mean atrial conduction times were within the normal range (27) throughout the exposure and changes were insignificant. The pattern of change in the mean cardiac

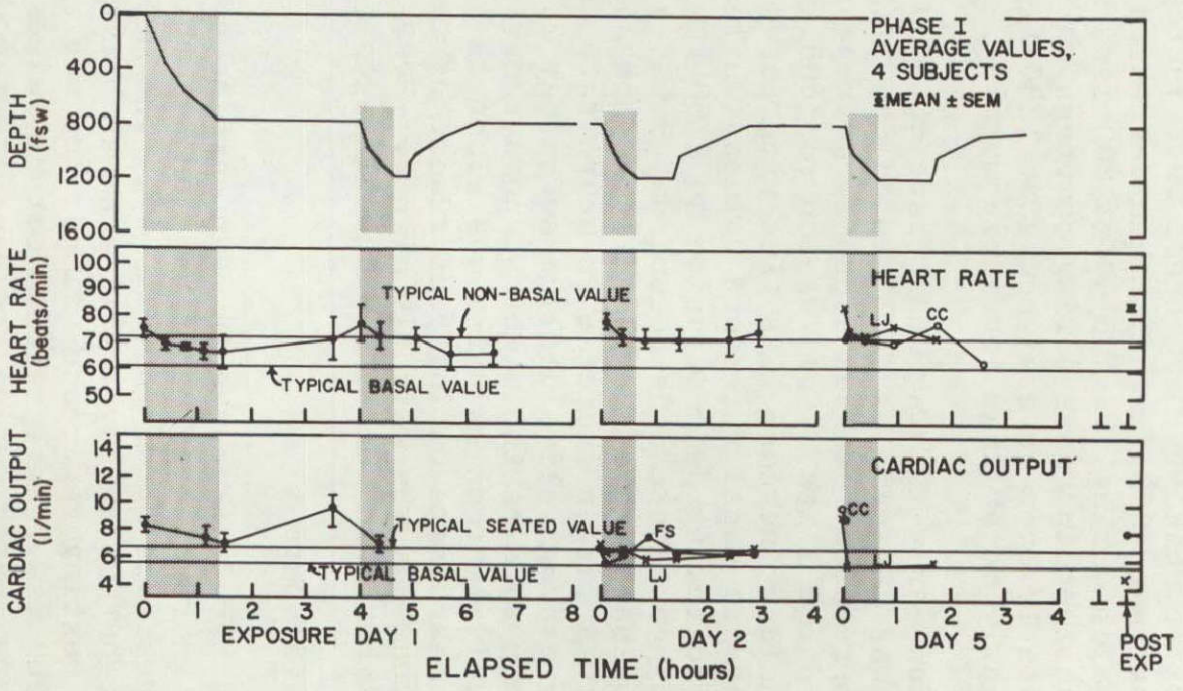


FIG. 3. Heart rate and cardiac output of subjects at rest during compression and exposure to pressures equivalent to 800 and 1200 fsw. The exposure profile is a composite for both subject pairs.

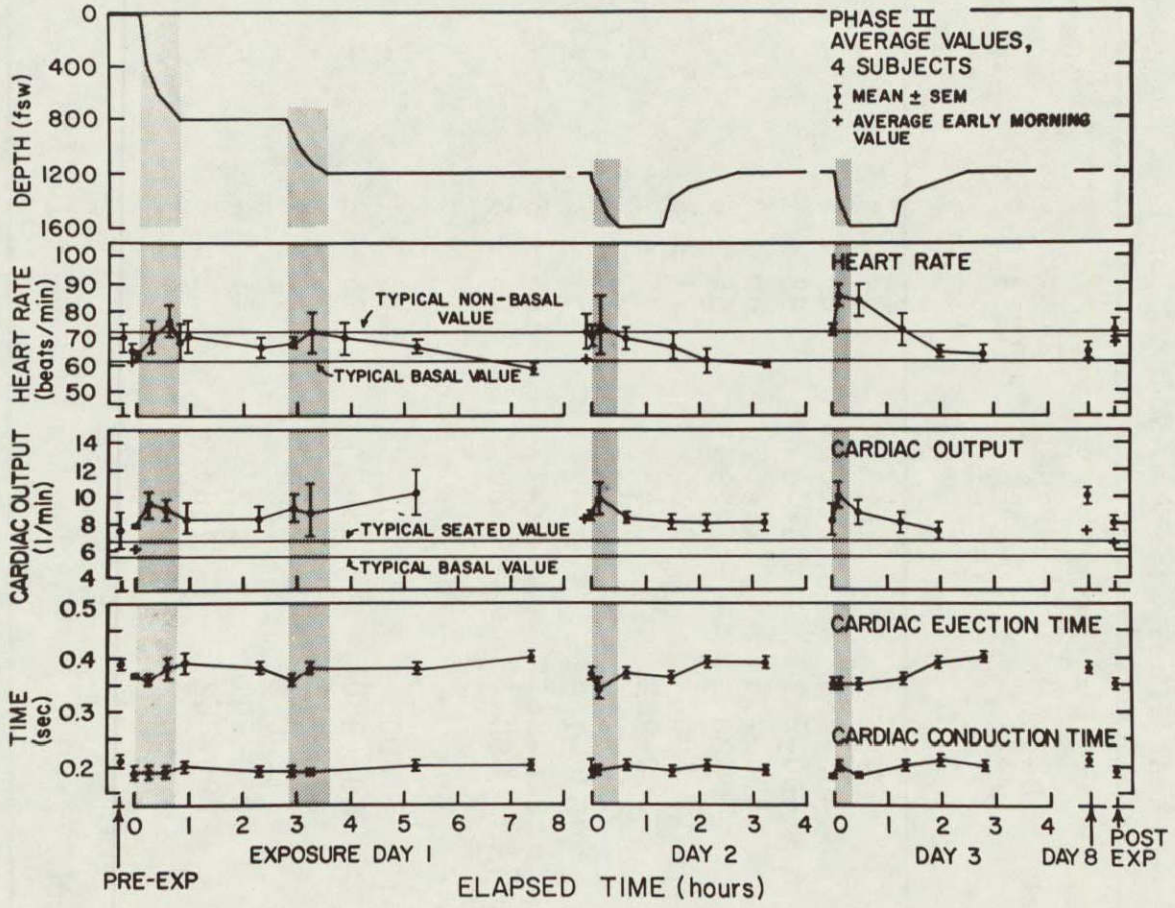


FIG. 4. Heart rate, cardiac output and timing of cardiac events of subjects at rest during compression and exposure to pressures equivalent to 800, 1200 and 1600 fsw. (For cardiac output, N = 3 on day 1.)

TABLE II. Cardiac Function During Compression-Pressure Exposures to 800 and 1200 fsw, Subjects at Rest (Phase I, Mean Values for Four Subjects)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	Heart Rate (beats/min) Mean ± SEM	Cardiac Output (l/min) Mean ± SEM
1	0	0	75 ± 3	8 ± 0.5
	400-488	0:26	69 ± 2	-
	600-652	0:46	68 ± 1	-
	690-738	1:06	66 ± 3	7.2 ± 0.9
	800	1:28	66 ± 6	6.7 ± 0.7
	800	3:34	71 ± 8	9.4 ± 1.2
	800-980	3:58	77 ± 7	-
	1103-1148	4:23	72 ± 5	6.7 ± 0.5
	1050-1005	5:02	71 ± 4	-
	888-865	5:42	65 ± 5	-
	800	6:30	66 ± 5	-
2	800	0	76 ± 1	-
	800-980	0:05	78 ± 3	-
	1090-1157	0:25	72 ± 3	-
	1200	0:49	71 ± 3	-
	1050-1010	1:28	71 ± 4	-
	886-862	2:09	71 ± 6	-
	800	2:52	74 ± 5	-
Post-exp.	0	-	84 ± 1	-

[a]Elapsed time is the average time from the start of compression to the mid-point of the measurement period.

TABLE III. Cardiac Function During Compression-Pressure Exposures to 800, 1200, and 1600 fsw, Subjects at Rest (Phase II, Mean Values for Four Subjects)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	Heart Rate (beats/min)	Cardiac[b] Output (l/min)	Cardiac Conduction Time (sec)	Cardiac Ejection Time (sec)
			Mean ± SEM	Mean ± SEM	Mean ± SEM	Mean ± SEM
Pre-exp.	0	-	70 ± 5	7.5 ± 1.4	-	-
1	0	-	65 ± 3	-	-	-
	0	-	64 ± 1	7.8 ± 0.1	0.21 ± 0.01	0.39 ± 0.01
	0	0	-	-	0.19 ± 0.01	0.37 ± 0
	400-550	0:18	70 ± 6	9.5 ± 1.0	0.19 ± 0.01	0.36 ± 0.01
	654-724	0:37	76 ± 6	9.0 ± 0.8	0.19 ± 0.01	0.38 ± 0.02
	800	0:48	68 ± 7	-	-	-
	800	0:59	70 ± 6	8.3 ± 1.1	0.20 ± 0.01	0.39 ± 0.02
	800	1:10	65 ± 4	-	-	-
	800	2:18	66 ± 4	8.3 ± 0.9	0.19 ± 0.01	0.38 ± 0.01
	860-1020	2:58	68 ± 2	9.1 ± 1.0	0.19 ± 0.01	0.36 ± 0.01
	1110-1155	3:17	72 ± 7	8.9 ± 1.9	0.19 ± 0	0.38 ± 0.01
	1200	3:48	69 ± 6	-	-	-
	1200	5:14	66 ± 3	10.3 ± 1.7	0.20 ± 0.01	0.39 ± 0.01
	1200	7:25	58 ± 2	-	0.20 ± 0.01	0.40 ± 0.01
2	1200	-	71 ± 6	-	-	-
	1200	0	70 ± 4	8.6 ± 0.3	0.19 ± 0.02	0.37 ± 0.01
	1270-1470	0:07	74 ± 11	9.7 ± 1.2	0.19 ± 0.01	0.34 ± 0.02
	1600	0:40	69 ± 4	8.4 ± 0.4	0.20 ± 0.01	0.37 ± 0.01
	1420-1375	1:31	66 ± 5	8.1 ± 0.5	0.19 ± 0.01	0.36 ± 0.01
	1285-1265	2:10	61 ± 4	8.0 ± 0.6	0.20 ± 0.01	0.39 ± 0.01
	1200	3:18	59 ± 1	8.0 ± 0.5	0.19 ± 0.01	0.39 ± 0.01
	1200	3:33	59 ± 5	-	-	-
3	1200	-	82 ± 6	-	-	-
	1200	0	72 ± 2	8.2 ± 1.2	0.18 ± 0	0.35 ± 0.01
	1280-1510	0:07	85 ± 4	10.0 ± 1.0	0.20 ± 0.01	0.35 ± 0.01
	1600	0:30	83 ± 6	8.6 ± 0.9	0.18 ± 0	0.35 ± 0.01
	1600	1:01	73 ± 9	-	-	-
	1467-1395	1:21	72 ± 6	8.0 ± 0.8	0.20 ± 0.01	0.36 ± 0.01
	1285-1263	2:00	64 ± 2	7.4 ± 0.6	0.21 ± 0.01	0.39 ± 0.01
	1200	2:51	63 ± 3	6.9 ± 1.1	0.20 ± 0.01	0.40 ± 0.01
8	1200	-	64 ± 5	-	-	-
	1200	-	64 ± 3	10.1 ± 0.5	0.21 ± 0.01	0.38 ± 0.01
Post-exp.	0	-	80 ± 3	8.0 ± 0.5	0.19 ± 0	0.35 ± 0.01
	0	-	72 ± 4	-	-	-

[a] Elapsed time is the average time from the start of compression to the mid-point of the measurement period.

[b] N=3 for Day 1, no data obtained on subject FS.

ejection times was the inverse of that in the heart rate; these times were also within the normal range (27) throughout the exposure.

Responses to Combined Stresses of Exercise and Compression to High Pressures

Two subjects participated in the exercise experiments in each exposure phase. Results are presented in Tables IV and V and Fig. 5 for Phase I and in Tables VI and VII and Fig. 6 for Phase II. Figures 5 and 6 show heart rate and cardiac output during rest, exercise and recovery (measured four minutes after cessation of exercise). Oxygen consumption (reproduced from Section E-14) was measured only at rest and during exercise. Elapsed times were tabulated and plotted for the mid-point of the entire measurement interval (Fig. 1).

Heart rate, cardiac output and ventilation (Section E-14) in Phase I (Figs. 5A, CC, and 5B, WS) followed similar patterns of change during the compression-pressure exposures. Transient increases in heart rate and cardiac output frequently occurred during compressions. Return to baseline values from these transient changes was usually rapid and complete. Recovery at four minutes after each exercise interval was usually complete or nearly complete for subject CC, but not for WS.

The data of Phase II (Figs. 6A, CC, and 6B, GM) follow patterns generally similar to those of Phase I. Subject CC showed no major differences in response in spite of the faster compression rates and higher maximum pressures in Phase II (Fig. 6A) as compared with Phase I (Fig. 5A).

Interpretation of the findings in exercise as shown in Figs. 5 and 6 is complicated by the changing workload introduced by variations in pedaling rate. Spontaneous changes in pedaling rate caused undesired variations in oxygen consumption during successive intervals of exercise with the same workload setting on the bicycle ergometer (see Section E-14 for explanation). The following analysis of the findings eliminates the workload variation as an extraneous variable.

Individual values for heart rate and cardiac output were plotted against oxygen consumption and compared with the results of the exercise tolerance measurements at 1 ata. Figures 7A and 7D show these comparisons for subject CC (Phases I and II), Fig. 7B for WS (Phase I) and Fig. 7C for GM (Phase II). Heavy lines connect the data from the pre-exposure exercise tolerance experiments at 1 ata, and light lines indicate linear regressions calculated for the relationships on single exposure days.

Figures 7A and 7D show that CC's heart rate during both the Phase I and II pressure exposures scatter along lines parallel to and somewhat below the line defining the relationship at 1 ata. In addition, the cardiac output values in Phase I reflect the technical difficulties due to differences in electrode placements; these daily electrode placements improved in Phase II. Except for day 1 in Phase I, CC's cardiac output responses on any given day of the compression-pressure exposures are essentially parallel to the response at 1 ata.

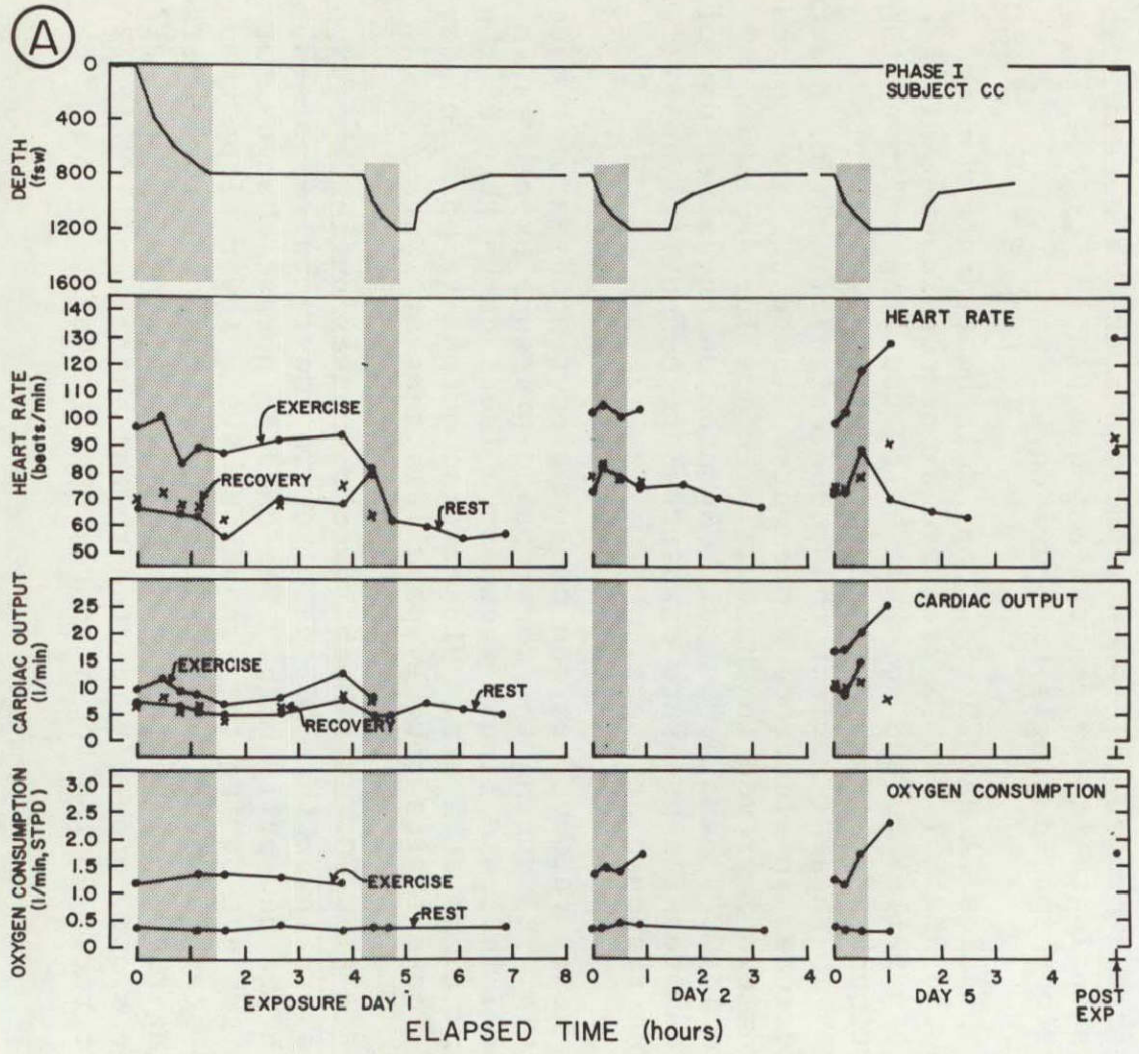
Heart rate vs. oxygen consumption responses of subject WS (Fig. 7B) during the compression-pressure exposures in Phase I follow a linear trend at a level comparable to the observations at 1 ata. His cardiac output response on day 1 of Phase I also roughly parallels that at 1 ata.[2]

In Fig. 7C, GM's heart rate responses during the exposures are lower than but parallel to the 1 ata responses, which are unusually high. The cardiac output changes for this subject during the exposures have a lower slope than at 1 ata; however, his cardiac output response to exercise during the exercise tolerance measurements has an unusually large slope, probably related to the exceptionally high heart rates during the measurements at 1 ata.

Cardiac Response to Postural Change

Heart rate is less reliable than blood pressure as a quantitative measure of the cardiac response to a tilt-

[2] Cardiac output measurements were not obtained on WS's second exposure day.


A
PHASE I
SUBJECT CC
DEPTH (fsw)
0
400
800
1200
1600
HEART RATE
HEART RATE (beats/min)
140
130
120
110
100
90
80
70
60
50
EXERCISE
RECOVERY
REST
CARDIAC OUTPUT
CARDIAC OUTPUT (l/min)
25
20
15
10
5
0
EXERCISE
REST
RECOVERY
OXYGEN CONSUMPTION
OXYGEN CONSUMPTION (l/min, STPD)
3.0
2.5
2.0
1.5
1.0
0.5
0
EXERCISE
REST
0 1 2 3 4 5 6 7 8
EXPOSURE DAY 1
0 1 2 3 4
DAY 2
0 1 2 3 4
DAY 5
POST EXP
ELAPSED TIME (hours)

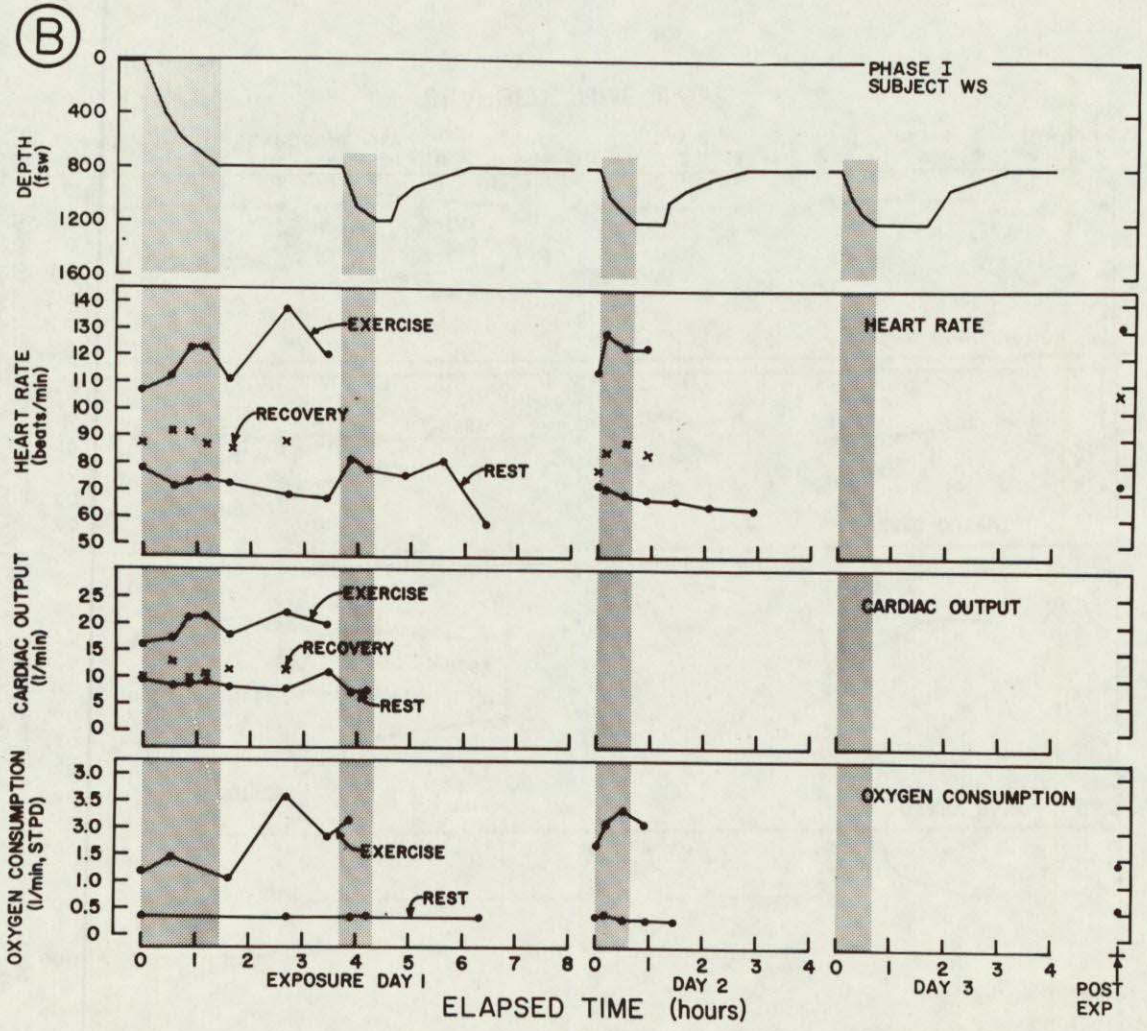
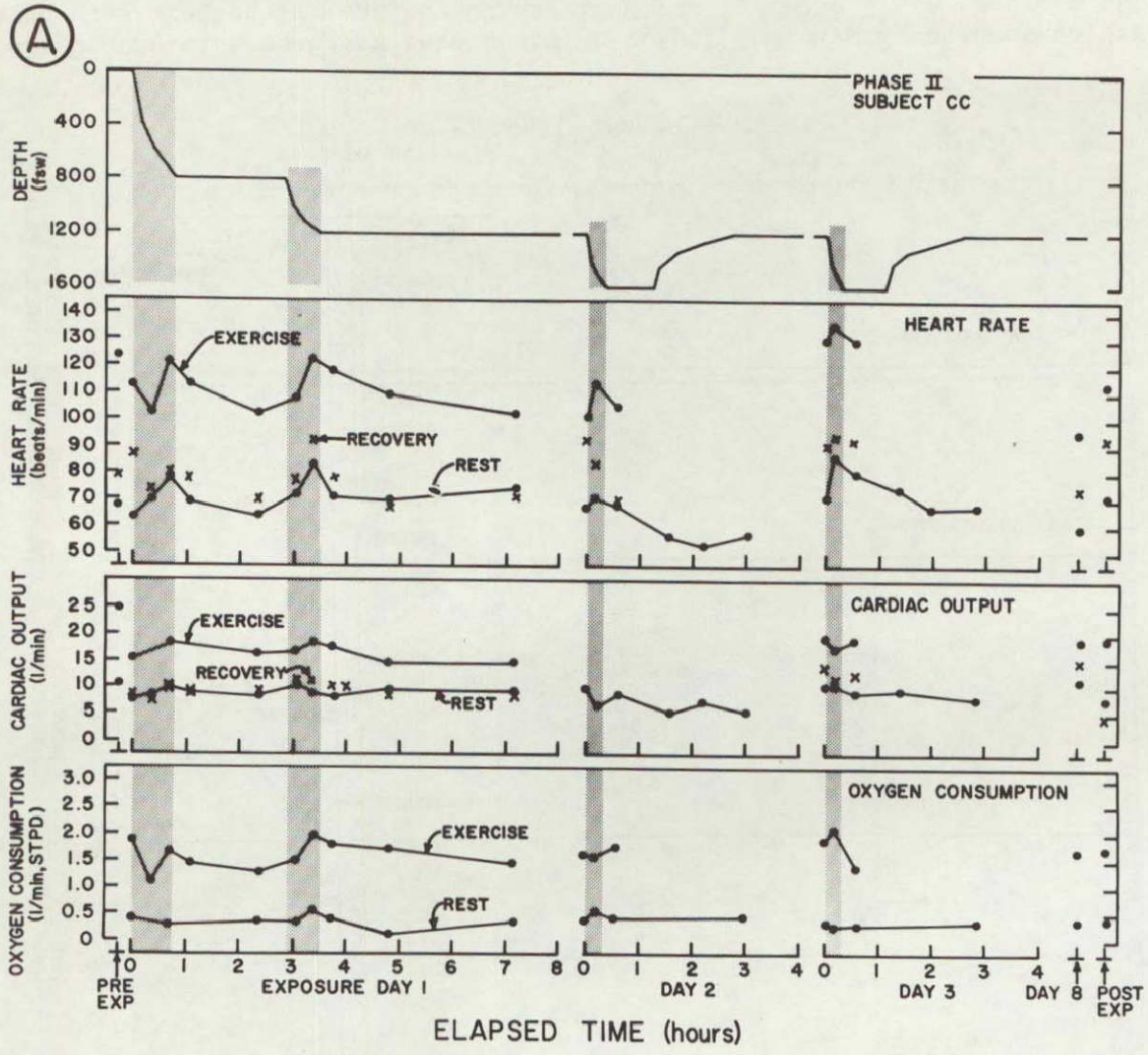


FIG. 5. Effect of exercise on heart rate, cardiac output and oxygen consumption during compression and exposure to pressures equivalent to 800 and 1200 fsw in Phase I. A, Subject CC; B, subject WS.


A
PHASE II
SUBJECT CC
DEPTH (fsw)
0
400
800
1200
1600
HEART RATE
HEART RATE (beats/min)
EXERCISE
RECOVERY
REST
140
130
120
110
100
90
80
70
60
50
CARDIAC OUTPUT
CARDIAC OUTPUT (l/min)
25
20
15
10
5
0
OXYGEN CONSUMPTION
OXYGEN CONSUMPTION (l/min, STPD)
3.0
2.5
2.0
1.5
1.0
0.5
0
0 1 2 3 4 5 6 7 8
0 1 2 3 4
0 1 2 3 4
PRE EXP
EXPOSURE DAY 1
DAY 2
DAY 3
DAY 8
POST EXP
ELAPSED TIME (hours)

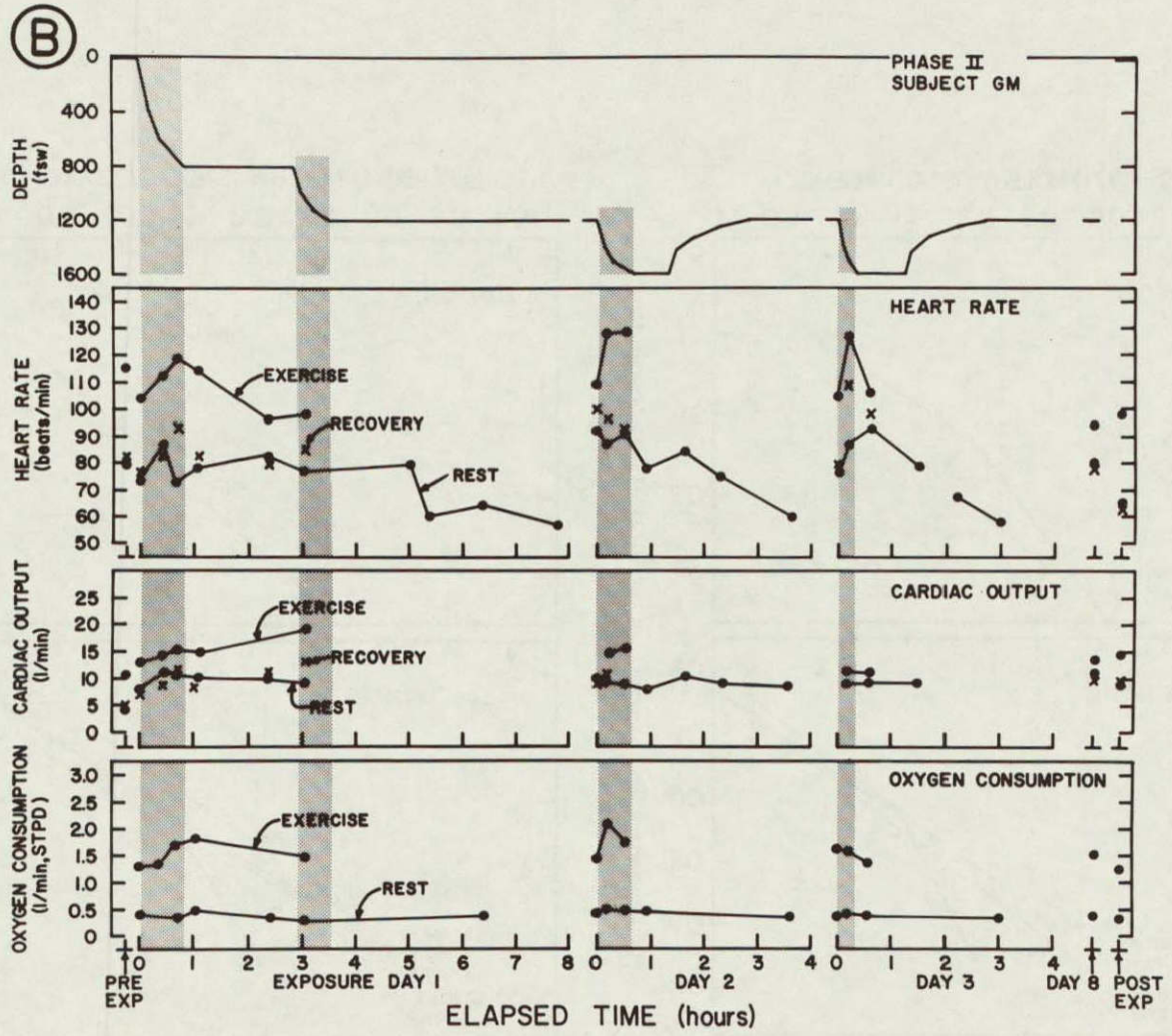


FIG. 6. Effect of exercise on heart rate, cardiac output and oxygen consumption during compression and exposure to pressures equivalent to 800, 1200 and 1600 fsw in Phase II. A, Subject CC; B, subject GM.

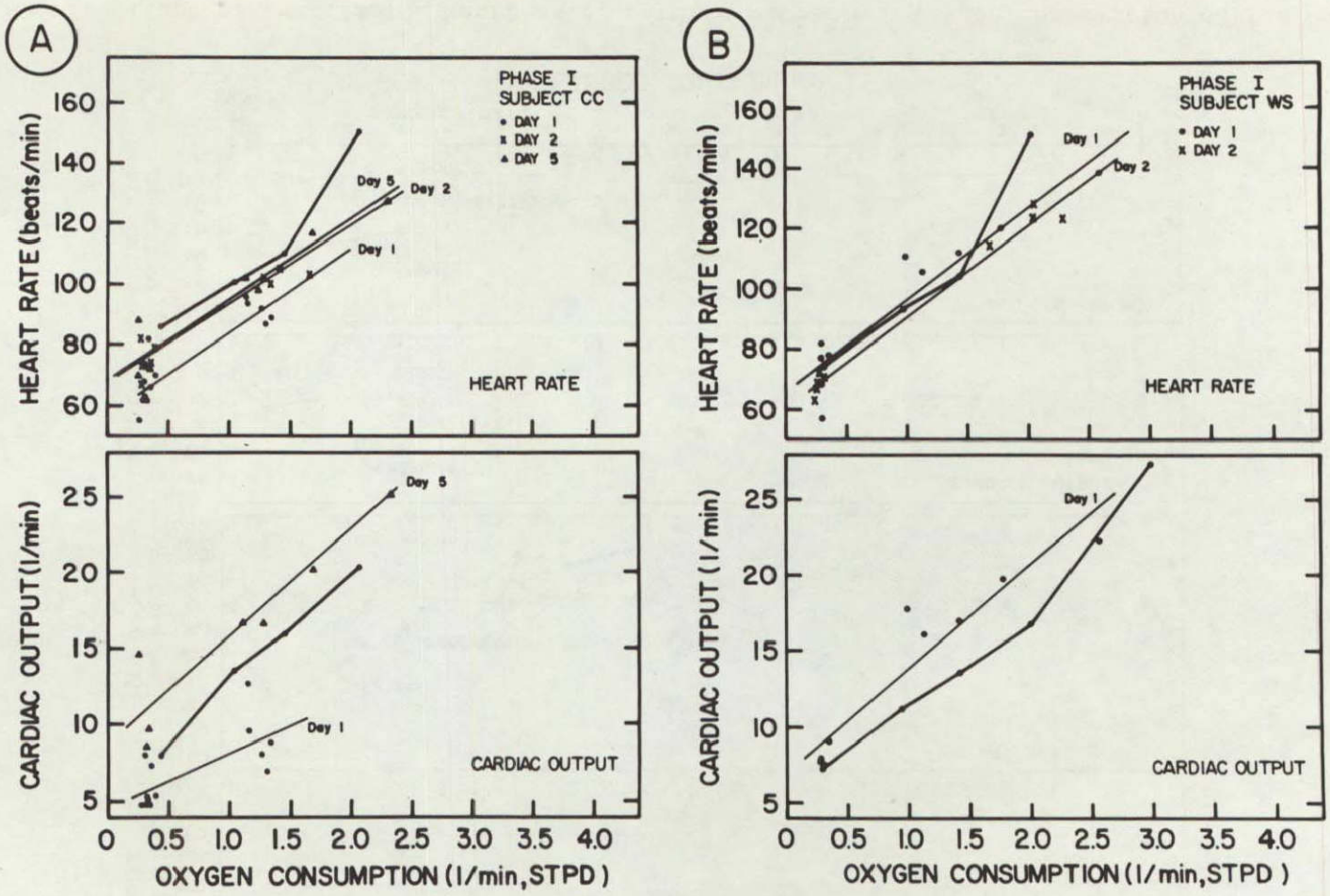

A
PHASE I
SUBJECT CC
• DAY 1
x DAY 2
▲ DAY 5
HEART RATE (beats/min)
160
140
120
100
80
60
Day 5
Day 2
Day 1
HEART RATE
CARDIAC OUTPUT (l/min)
25
20
15
10
5
Day 5
Day 1
CARDIAC OUTPUT
0 0.5 1.0 1.5 2.0 2.5 3.0 3.5 4.0
OXYGEN CONSUMPTION (l/min, STPD)
B
PHASE I
SUBJECT WS
• DAY 1
x DAY 2
HEART RATE (beats/min)
160
140
120
100
80
60
Day 1
Day 2
HEART RATE
CARDIAC OUTPUT (l/min)
25
20
15
10
5
Day 1
CARDIAC OUTPUT
0 0.5 1.0 1.5 2.0 2.5 3.0 3.5 4.0
OXYGEN CONSUMPTION (l/min, STPD)

TABLE IV. Effects of Exercise on Heart Rate, Cardiac Output and Oxygen Consumption During Compression-Pressure Exposures to 800 and 1200 fsw (Phase I, Subject CC)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	REST			EXERCISE			RECOVERY	
			Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)
1	0	0	66	7.3	0.35	96	9.6	1.15	69	6.6
	420-570	0:30	-	-	-	100	11.9	-	72	8.0
	612-701	0:50	64	6.2	-	83	9.3	-	68	5.5
	742-816	1:10	63	5.3	0.32	89	8.8	1.34	67	6.4
	800	1:36	55	4.8	0.28	87	6.9	1.30	62	3.8
	800	2:40	70	5.3	0.39	92	8.1	1.26	67	6.4
	800	3:48	68	8.0	0.30	94	12.7	1.13	75	8.5
	840-1070	4:24	82	4.8	0.34	79	8.2	-	64	7.9
	1110-1185	4:44	62	4.8	0.32	-	-	-	-	-
	1015-940	5:23	60	7.1	-	-	-	-	-	-
	878-840	6:03	55	6.1	-	-	-	-	-	-
	800	6:51	62	5.1	0.32	-	-	-	-	-
2	800	0	73	-	0.28	102	-	1.29	79	-
	840-1070	0:10	82	-	0.29	105	-	1.41	83	-
	1110-1185	0:30	79	-	0.40	100	-	1.34	78	-
	1200	0:53	74	-	0.36	103	-	1.66	77	-
	1015-940	1:40	75	-	-	-	-	-	-	-
	875-838	2:23	70	-	-	-	-	-	-	-
	800	3:10	67	-	0.29	-	-	-	-	-
5	800	0	72	9.7	0.34	98	16.6	1.23	74	10.4
	840-1070	0:10	73	8.5	0.31	102	16.7	1.11	73	10.3
	1110-1185	0:30	88	14.7	0.25	117	20.3	1.69	78	11.2
	1200	1:01	70	-	0.25	128	25.2	2.32	91	7.8
	1015-940	1:50	65	-						
	888-855	2:30	63	-						
Post-exp.	0	-	88	-	-	130	-	1.70	93	-

[a]Elapsed time is from the start of compression to the mid-point of the 15-minute measurement interval.

TABLE V. Effects of Exercise on Heart Rate, Cardiac Output and Oxygen Consumption During Compression-Pressure Exposures to 800 and 1200 fsw (Phase I, Subject WS)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	REST			EXERCISE			RECOVERY	
			Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)
1	0	0	78	9.0	0.35	106	16.1	1.14	88	9.8
	419-563	0:33	71	8.4	-	112	17.0	1.43	91	12.9
	602-676	0:52	73	8.8	-	123	21.0	-	91	9.4
	707-780	1:12	74	9.3	-	123	21.2	-	87	10.5
	800	1:36	72	8.1	-	111	17.8	1.00	85	11.3
	800	2:40	68	7.7	0.28	138	22.3	2.58	88	11.3
	800	3:29	67	11.0	-	120	19.7	1.79	-	-
	840-1070	3:51	82	7.5	0.30	-	-	2.10	-	-
	1110-1185	4:11	77	7.9	0.28	-	-	-	-	-
	1015-940	4:51	75	-	-	-	-	-	-	-
	878-840	5:31	81	-	-	-	-	-	-	-
	800	6:19	57	-	0.29	-	-	-	-	-
2	800	0	72	-	0.27	114	-	1.68	77	-
	840-1070	0:10	71	-	0.32	128	-	2.05	85	-
	1100-1175	0:30	68	-	0.26	123	-	2.28	88	-
	1200	0:55	67	-	0.26	123	-	2.05	83	-
	1015-940	1:25	66	-	-	-	-	-	-	-
	888-850	2:05	64	-	-	-	-	-	-	-
	800	2:53	63	-	0.23	-	-	-	-	-
Post-exp.	0	-	73	-	0.52	131	-	1.30	106	-

[a]Elapsed time is from the start of compression to the mid-point of the 15-minute measurement interval.

TABLE VI. Effects of Exercise on Heart Rate, Cardiac Output and Oxygen Consumption During Compression-Pressure Exposures to 800, 1200, and 1600 fsw (Phase II, Subject CC)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	REST			EXERCISE			RECOVERY	
			Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)
Pre-exp.	0	-	67	10.0	-	123	24.6	-	79	-
1	0	0	63	7.3	0.38	112	15.4	1.88	86	7.9
	400-640	0:22	70	8.4	-	102	-	1.09	73	7.7
	656-792	0:41	78	9.6	0.27	121	18.2	1.62	79	9.6
	800	1:02	69	8.7	-	113	-	1.48	78	9.4
	800	2:23	64	8.2	0.35	102	15.2	1.26	70	8.6
	860-1100	3:02	72	10.3	0.33	108	16.8	1.48	77	10.8
	1110-1195	3:21	83	8.7	0.53	123	18.4	1.97	92	12.4
	1200	3:45	71	8.2	0.41	118	17.3	1.75	78	9.7
	1200	4:46	69	9.4	0.39	109	14.8	1.71	68	8.3
	1200	7:11	74	9.5	0.38	102	14.9	1.45	72	8.8
2	1200	0	67	9.6	0.40	101	-	1.61	92	-
	1280-1580	0:11	71	6.7	0.53	113	-	1.58	83	-
	1600	0:34	68	8.9	0.43	105	-	1.75	69	-
	1415-1330	1:32	56	5.4	-	-	-	-	-	-
	1283-1248	2:10	53	7.4	-	-	-	-	-	-
	1200	3:01	57	5.7	0.46	-	-	-	-	-
3	1200	0	70	10.4	0.34	130	19.5	1.89	90	13.8
	1280-1580	0:11	86	10.4	0.28	136	17.5	2.10	93	11.8
	1600	0:34	80	9.4	0.30	129	19.4	1.57	92	12.5
	1425-1340	1:19	74	9.6	-	-	-	-	-	-
	1285-1250	1:58	67	-	-	-	-	-	-	-
	1200	2:49	67	8.3	0.36	-	-	-	-	-
8	1200	-	59	11.9	0.42	95	19.2	1.69	74	15.3
Post-exp.	0	-	71	8.2	0.42	113	19.2	1.72	92	4.8

[a]Elapsed time is from the start of compression to the mid-point of the 17-minute measurement interval.

TABLE VII. Effects of Exercise on Heart Rate, Cardiac Output and Oxygen Consumption During Compression-Pressure Exposures to 800, 1200, and 1600 fsw (Phase II, Subject GM)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	REST			EXERCISE			RECOVERY	
			Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (l/min STPD)	Heart Rate (beats/min)	Cardiac Output (l/min)
Pre-exp.	0	-	80	4.3	-	115	10.6	-	81	4.8
1	0	0	74	8.1	0.40	104	12.7	1.28	76	7.0
	400-637	0:22	87	11.0	-	112	14.2	1.32	82	8.9
	652-778	0:41	73	10.6	0.35	119	15.0	1.70	93	11.4
	800	1:04	78	10.1	0.46	114	14.8	1.80	82	8.4
	800	2:21	82	10.0	0.35	96	-	-	79	11.0
	860-1100	3:02	77	9.7	0.28	98	18.8	1.47	84	12.9
	1200	5:00	79	-	-	-	-	-	-	-
	1200	5:22	60	-	-	-	-	-	-	-
	1200	6:23	64	-	0.37	-	-	-	-	-
	1200	7:46	57	-	-	-	-	-	-	-
2	1200	0	92	10.2	0.41	109	-	1.42	99	9.2
	1260-1500	0:12	86	9.5	0.46	128	14.9	2.04	96	10.8
	1510-1595	0:31	90	9.3	0.44	128	15.9	1.77	92	-
	1600	0:54	78	8.3	0.43	-	-	-	-	-
	1425-1340	1:37	84	10.1	-	-	-	-	-	-
	1286-1248	2:17	75	9.1	-	-	-	-	-	-
	1200	3:33	60	8.6	0.35	-	-	-	-	-
3	1200	0	76	-	0.37	104	-	1.58	78	-
	1280-1570	0:11	87	9.3	0.39	126	-	1.53	108	11.1
	1600	0:34	93	9.6	0.37	115	-	1.37	97	11.1
	1420-1335	1:30	78	9.3	-	-	-	-	-	-
	1285-1250	2:09	67	-	-	-	-	-	-	-
	1200	3:00	58	-	0.33	-	-	-	-	-
8	1200	-	80	11.4	0.39	94	13.3	1.52	77	9.9
Post-exp.	0	-	65	9.0	0.34	98	14.7	1.26	62	9.2

[a]Elapsed time is from the start of compression to the mid-point of the 17-minute measurement interval.

table test (30). However, both heart rate and cardiac stroke volume may be used as indices of the cardiac response to a postural change (5,30-32,35). The data for heart rate in this study were more complete than for cardiac output, (see Tables II and III); therefore, heart rate was selected as the index for evaluating reflex cardiovascular response to change from the seated to the erect position.

The general pattern of the chronotropic response was an acceleration to a peak in 20 seconds or less. The exact pattern (acceleration, peak value, deceleration) was variable, probably due in part to varying activity levels before standing (Fig. 1), and to variable rates of change from the seated to the standing position. For this reason, the mean heart rate of all measurements before standing was used as the reference value, while the mean heart rate during the first minute after standing was chosen as the heart rate response to postural change. The difference between the two means is defined here as an index of the cardiovascular response to the gravitational effects of changing from the seated to the erect position. This index is tabulated in Tables VIII and IX and is plotted in Fig. 8 for Phase I, and in Tables X and XI and Fig. 9 for Phase II. Elapsed times are tabulated and plotted midway in the measurement intervals.

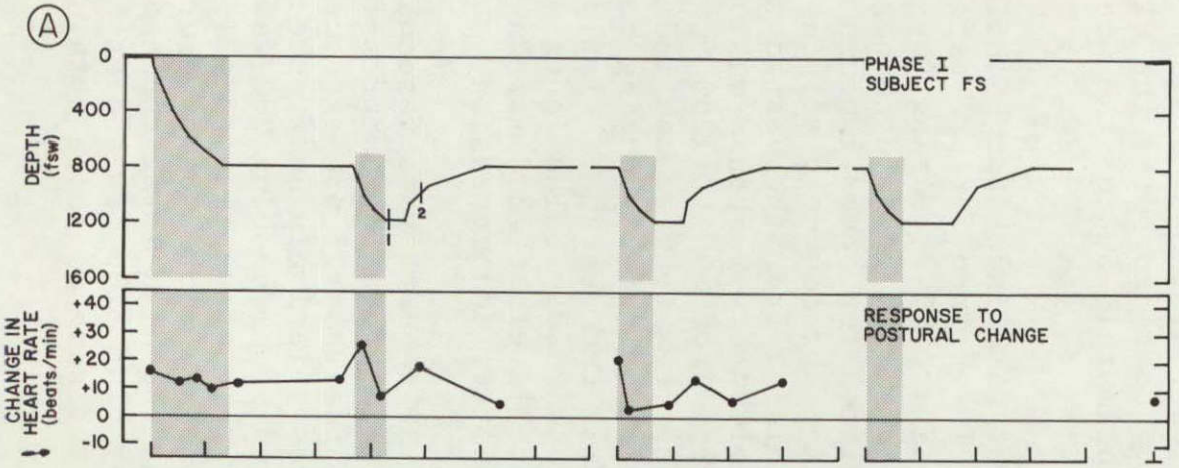
Subject LJ in Phase I (Fig. 8B, Table VIII) showed no change in his response to postural change during the compression from 1 ata to 800 fsw on exposure day 1. The difference between his average heart rates before and after standing (heart rate response index) was between 10 and 14 beats/minute during this compression and for several hours after reaching the stable pressure of 800 fsw. Thereafter, including the excursion to 1200 fsw, his index varied irregularly. On exposure day 2, LJ's index was initially somewhat below that at 1 ata, approximately doubled during compression to 1200 fsw, and subsequently declined to below that at 1 ata. The last two measurements on that day during and shortly after decompression to 800 fsw showed essentially no heart rate response to postural change. On exposure day 5, LJ's heart rate index varied irregularly on standing. Post-exposure at 1 ata, the rise in heart rate on standing was less than at 1 ata on the first exposure day.

The magnitudes and patterns of change in FS's heart rate index on exposure day 1 in Phase I resembled those of LJ (Fig. 8A, Table IX). His index on the second exposure day started at an increase of 21 beats/minute at 800 fsw, was only 3 beats/minute during compression to 1200 fsw and increased during the remainder of the excursion to levels similar to those of day 1. Post-exposure, the rise in heart rate on standing was approximately 50% of the pre-exposure value at 1 ata.

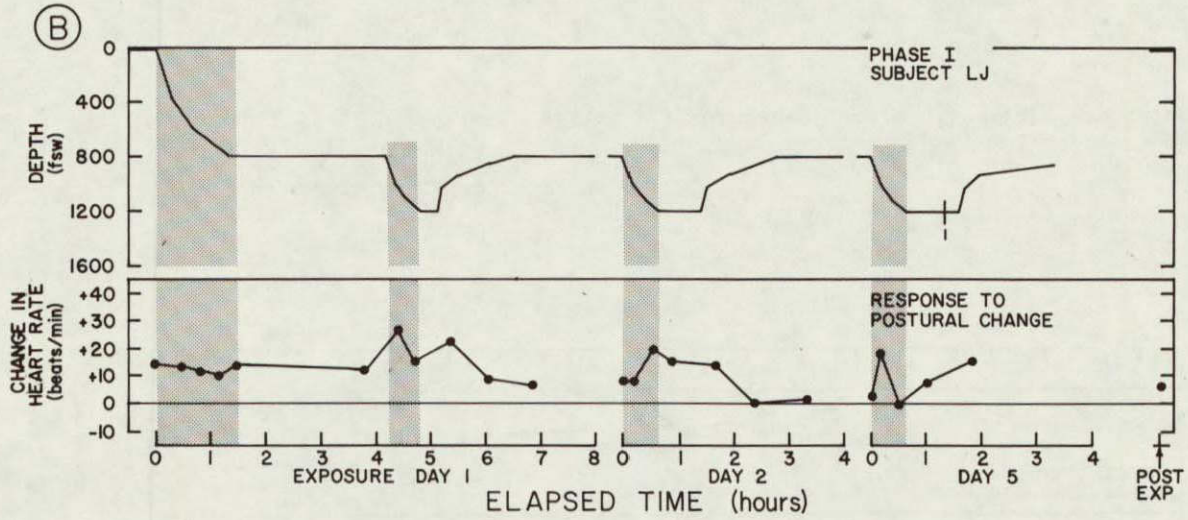
The pre-exposure heart rate index for FS before the beginning of Phase II was lower than for pre-exposure in Phase I (Fig. 9A, Tables IX and XI). During the compression from 1 ata to 800 fsw, there was a major decline in heart rate response, which persisted for several hours at 800 fsw, with essentially no positive chronotropic response to the gravitational stress of arising from the seated to the erect position. The cardio-acceleratory response was observed again during the compression from 800 to 1200 fsw. On exposure day 2 FS showed a heart rate response index near the pre-exposure level; it declined briefly during compression to 1600 fsw and subsequently recovered. A similar pattern was seen on FS's third exposure day. The index was somewhat above the pre-exposure level at 1200 fsw on the eighth exposure day and at 1 ata after decompression.

In Phase II, subject MP's heart rate response index (Fig. 9B, Table X) at 1 ata, before the compression-pressure exposures, was similar to the Phase I pre-exposure values of subjects LJ and FS. It was relatively unaffected by compression, more than doubled upon reaching 800 fsw, and remained elevated for the rest of exposure day 1. No remarkable changes in the index occurred on exposure days 2 and 3, when it remained at the high level reached on day 1 at 800 fsw. Values on day 8 at 1200 fsw and at 1 ata after decompression were the same as those obtained on days 2 and 3.

Dizziness and lightheadedness were among the symptoms reported by the subjects during the compression-pressure exposures (Sections E-1 and E-6). The times and depths of occurrence of these symptoms, as well as condensed versions of the subjects' comments, are included on Figs. 8 and 9 for correlation with the postural change data. Subject FS reported "lightheadedness" shortly after reaching 800 fsw on

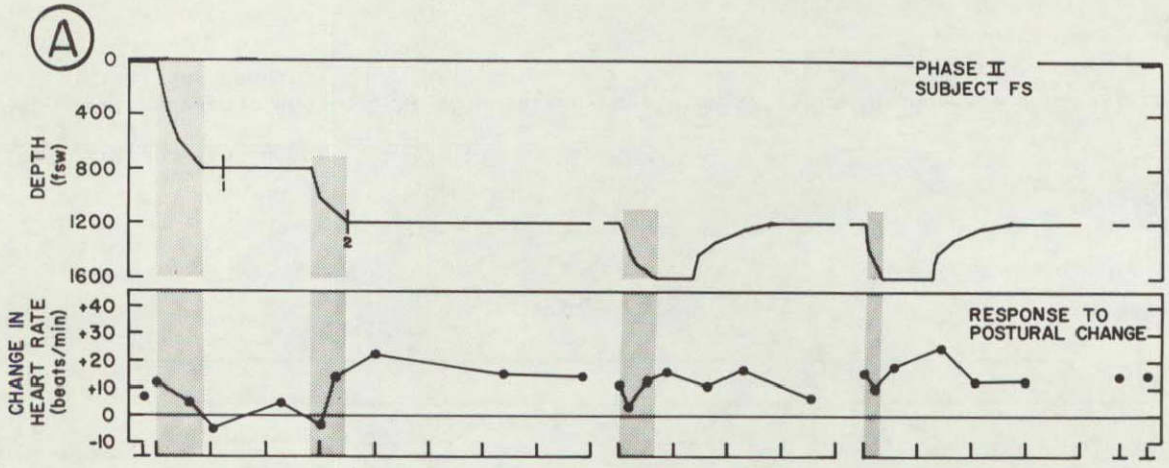


Comment Number	Elapsed Time	Symptoms and Signs Subject FS
1	Day 1 4:21	"I have a sort of dizziness when I get up and down, but I think that's just part of the headache. I pretty much feel okay." Dizzy feeling on standing started soon after leaving 800 fsw.
2	Day 1 4:54	Still had headache but no dizziness.

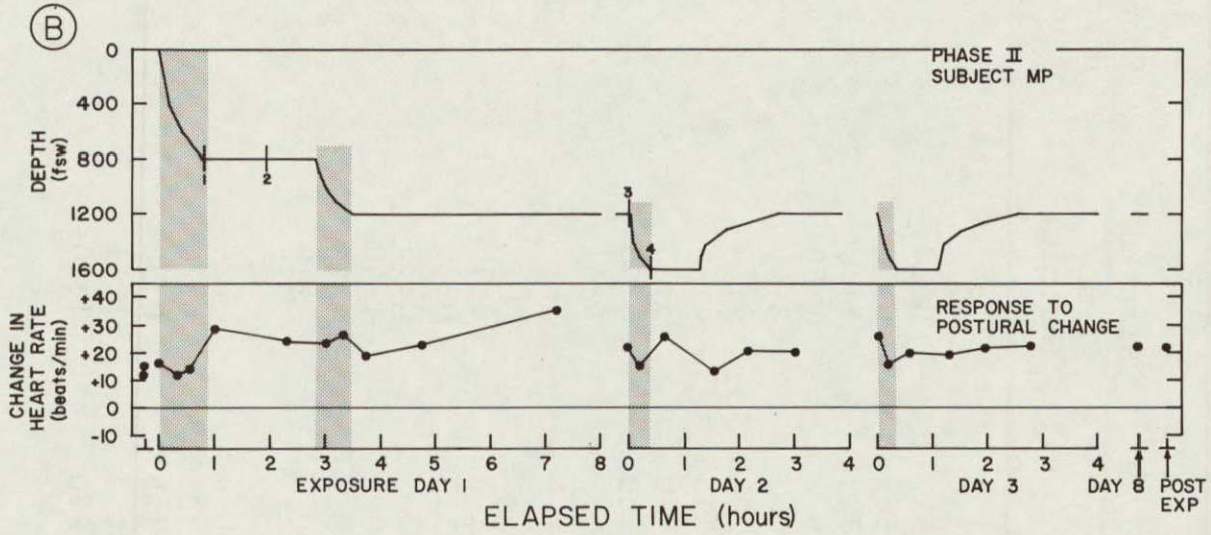


Comment Number	Elapsed Time	Symptoms and Signs Subject LJ
1	Day 5 1:19	Felt nauseous and thought the vestibular electrical stimulation gave unusually strong responses; dizzy on standing.

FIG. 8. Heart rate responses to postural change during compression and exposure to pressures equivalent to 800 and 1200 fsw. A, Subject FS; B, subject LJ.



Comment Number	Elapsed Time	Symptoms and Signs Subject FS
1	Day 1 1:12	"Little bit" lightheaded.
2	Day 1 3:30	On arrival at 1200 fsw felt "slightly lightheaded"; only discomfort was "slight headache."



Comment Number	Elapsed Time		Symptoms and Signs Subject MP
1	Day 1	0:50	Felt "a little dizzy."
2	Day 1	1:56	Some "dizziness on standing," no vertigo, no nausea.
3	Day 2	0:0	"A little bit slow and a little bit tired"; a little dizzy "if I move really fast."
4	Day 2	0:20	"Not really dizzy," "wouldn't want to try standing up without holding onto something," "spinning a little bit," only with eyes closed on balance board.

FIG. 9. Heart rate responses to postural change during compression and exposure to pressures equivalent to 800, 1200 and 1600 fsw. A, Subject FS; B, subject MP.

TABLE VIII. Heart Rate Response to Postural Change During Compression-Pressure Exposures to 800 and 1200 fsw (Phase I, Subject LJ)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	Change in Heart Rate (beats/min)
1	0	0	+14
	490-510	0:30	+13
	653-665	0:50	+11
	771-783	1:10	+10
	800	1:30	+14
	800	3:48	+12
	980-1010	4:24	+27
	1145-1155	4:44	+16
	980-970	5:23	+23
	865-860	6:03	+ 9
	800	6:51	+ 7
2	800	0	+ 9
	980-1010	0:10	+ 9
	1145-1155	0:30	+20
	1200	0:53	+16
	980-970	1:40	+14
	858-853	2:23	0
	800	3:18	+ 2
5	800	0	+ 3
	980-1010	0:10	+19
	1145-1155	0:30	0
	1200	1:01	+ 8
	980-970	1:50	+16
Post-exp.	0	-	+ 6

[a]Elapsed time is from the start of compression to the center of the two-minute measurement interval.

TABLE X. Heart Rate Response to Postural Change During Compression-Pressure Exposures to 800, 1200, and 1600 fsw (Phase I. Subject MP)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	Change in Heart Rate (beats/min)
Pre-exp.	0	-	+11
	0	-	+15
1	0	0	+16
	533-566	0:22	+12
	721-737	0:41	+13
	800	1:02	+28
	800	2:23	+24
	1010-1030	3:02	+23
	1150-1160	3:21	+26
	1200	3:45	+19
	1200	4:46	+22
	1200	7:11	+35
2	1200	0	+21
	1515-1535	0:11	+14
	1600	0:34	+25
	1375-1365	1:32	+13
	1270-1265	2:10	+20
	1200	3:01	+20
3	1200	0	+26
	1500-1520	0:11	+15
	1600	0:34	+19
	1385-1375	1:19	+19
	1270-1265	1:58	+21
	1200	2:49	+22
8	1200	-	+22
Post-exp.	0	-	+21

[a]Elapsed time is from the start of compression to the center of the two-minute measurement interval.

TABLE XI. Heart Rate Response to Postural Change During Compression-Pressure Exposures to 800, 1200, and 1600 fsw (Phase II, Subject FS)

Exposure Day	Depth (fsw)	Elapsed Time[a] (hr:min)	Change in Heart Rate (beats/min)
Pre-exp.	0	-	+ 7
1	0	0	+12
	711-726	0:41	+ 5
	800	1:04	- 5
	800	2:21	+ 4
	1010-1030	3:02	- 4
	1150-1160	3:21	+14
	1200	3:59	+22
	1200	6:23	+15
	1200	7:48	+14
2	1200	0	+11
	1400-1420	0:12	+ 3
	1545-1555	0:31	+13
	1600	0:54	+16
	1390-1380	1:37	+11
	1270-1265	2:17	+16
	1200	3:33	+ 6
3	1200	0	+15
	1500-1520	0:11	+ 9
	1600	0:34	+18
	1420-1410	1:30	+25
	1265-1260	2:09	+12
	1200	3:00	+12
8	1200	-	+14
Post-exp.	0	-	+14

[a]Elapsed time is from the start of compression to the center of the two-minute measurement interval.

exposure day 1 in Phase II at the time his heart rate response to standing was minimal (Fig. 9A). However, there were no other such comments by any subject which correlated with declines in the cardiac rate response index.

EARLY MORNING MEASUREMENTS

Measurements of heart rate (Phases I and II) and cardiac output (Phase II only) in the early morning are listed in Table XII for Phase I and in Table XIII for Phase II. They are shown in Fig. 10 as a function of simulated depth

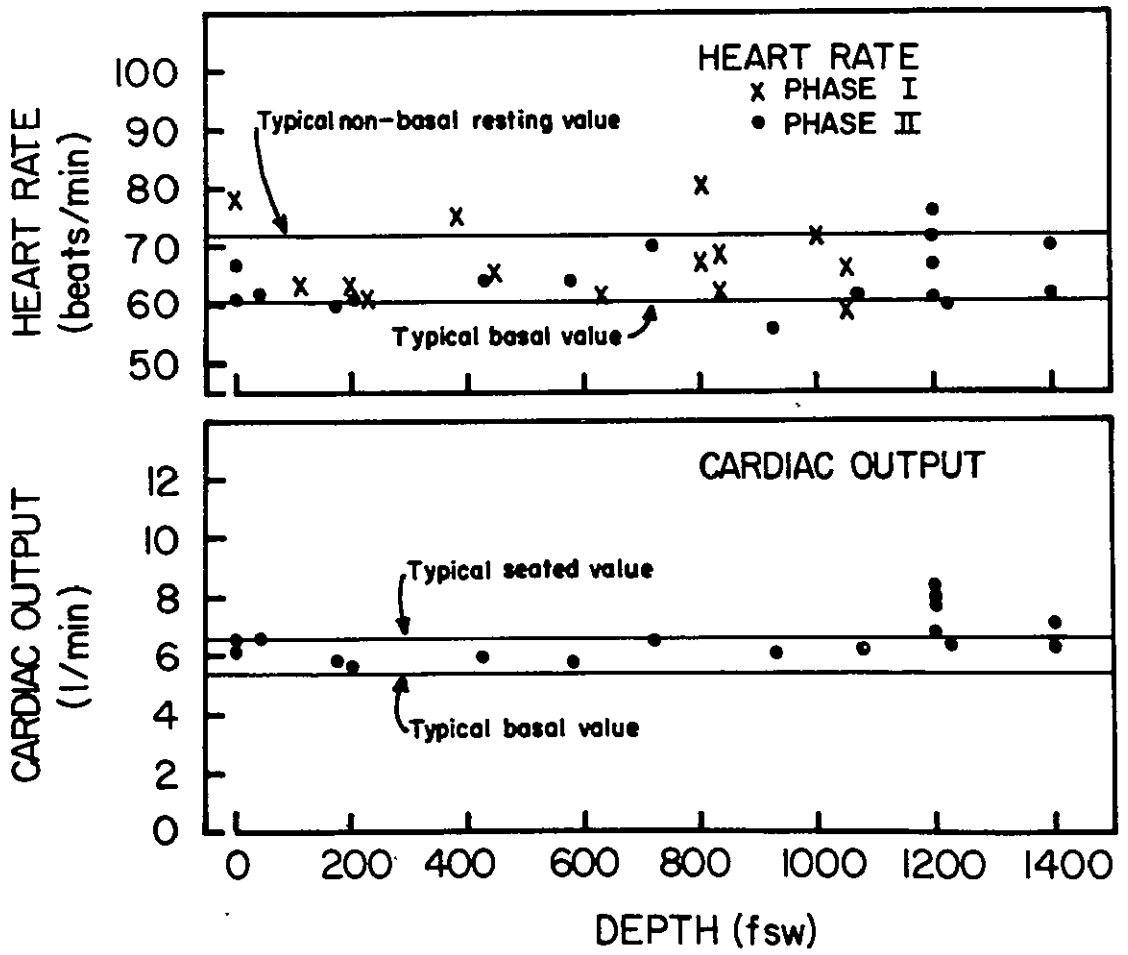


FIG. 10. Early morning values of heart rate and cardiac output at stable high pressures and during saturation-decompressions. Mean values for four subjects.

TABLE XII. Cardiac Function at Stable High Pressures and During Saturation Decompressions (Phase I, Measurements Made in the Early Morning with Subjects at Rest)

Exposure Day[a]	2;3	3;4	4;5	5;6	6;7	7;8	8;9	9;10	10;11	11;12	12;13	13;14	14;15	Post-exp.
Depth (fsw)	800	1000	833	800	1050	1050	836	632	447	383	229	200	111	0
Heart Rate (beats/min)														
Subject														
CC	64	68	58	66	64	58	56	58	56	70	62	54	56	64
LJ	67	89	77	76	-	68	62	66	76	87	54	56	54	74
FS	-	54	64	64	58	50	67	60	54	66	54	64	62	90
WS	70	73	73	112[b]	76	59	-	58	72	78	72	78	80	84
Mean	-	71	68	-	-	59	-	61	65	75	61	63	63	78
±SEM		7	4			4		2	6	5	4	5	6	6

[a]Exposure day for the same equivalent depths are different for the two subject pairs. The exposure day listed first is for CC and LJ, the second for FS and WS.

[b]Febrile response to severe dermatologic infection.

TABLE XIII. Cardiac Function at Stable High Pressures and During Saturation-Decompressions (Phase II, Measurements Made in the Early Morning with Subjects at Rest)

Exposure Day[a]	1	2	3	4	5;6	7	8	9	6;10	7;11	8;12	9;13	10;14	11;15	12;16	13;17	15;19	16;20	17;21	Post-exp.
Depth (fsw)	0	1200	1200	1200	1200	1200	1200	1200	1400	1400	1224	1072	924	715	579	425	200	171	40	0
Heart Rate (beats/min)																				
Subject																				
CC	51	54	-	69	64	-	-	-	63	52	53	54	45	60	58	52	50	48	60	63
MP	55	61	69	65	65	-	-	-	69	60	60	58	56	70	61	59	57	57	59	58
GM	66	69	82	81	72	-	67	73	72	61	60	73	67	70	65	70	60	60	66	82
FS	73	60	64	87	67	73	64	69	77	73	67	63	-	80	70	76	78	76	63	65
Mean	61	61	-	76	67	-	-	-	70	62	60	62	-	70	64	64	61	60	62	67
±SEM	5	3	-	5	2	-	-	-	3	4	3	4	-	4	3	5	6	6	2	5
Cardiac Output (l/min)																				
Subject																				
CC	5.7	8.4	-	8.1	6.9	-	-	-	6.1	5.6	6.2	6.8	5.7	5.2	5.6	6.1	5.3	5.2	6.2	7.6
MP	5.9	6.3	6.4	6.1	6.2	-	-	-	6.8	6.6	6.6	5.2	6.1	6.9	5.9	5.4	5.6	6.0	5.9	5.7
GM	7.2	11.5	10.2	9.1	6.9	-	8.5	6.7	7.7	5.8	6.8	6.6	6.7	6.8	6.1	6.5	6.2	6.2	7.9	6.4
FS	5.4	7.0	7.4	8.1	7.0	8.5	8.3	7.6	7.6	7.1	6.1	6.0	-	7.2	6.0	5.8	5.5	6.3	6.4	5.8
Mean	6.1	8.3	-	7.9	6.8	-	-	-	7.1	6.3	6.4	6.2	-	6.5	5.9	6.0	5.7	5.9	6.6	6.4
±SEM	0.4	1.2		0.6	0.2				0.4	0.3	0.2	0.4		0.4	0.2	0.2	0.2	0.2	0.4	0.4

[a]Exposure day for the same equivalent depths are different for the two subject pairs. The exposure day listed first is for CC and MP, the second for GM and FS.

at stable saturation pressures and during the slow decompressions to 1 ata. Data in Fig. 10 are shown as the mean of four subjects (CC, LJ, FS, WS in Phase I; CC, GM, MP, FS in Phase II). Individual values are plotted in Section E-15. Typical reference basal and non-basal values of heart rate and cardiac output are also shown (1). Morning heart rates were usually within a normal range, although there was considerable scatter for data measured at the same pressure on different days. No systematic relationship with pressure is evident. Cardiac output was also within the normal range and showed no systematic pattern of change with pressure over the exposure period. On days 2, 3 and 4 at 1200 fsw cardiac output was elevated relative to the other days (Table XIII, see also Section E-15).

DISCUSSION

It was considered possible in the design of this Predictive Study that pressure-induced interferences with the functions of ventilation, gas transport and perfusion could lead to hypoxia and altered acid-base balance, altering function of cells already affected by hydrostatic pressure.

Ventilation of the lungs was found to be maintained at essentially normal levels during the compression-pressure exposures (see Sections E-14 and E-16). Studies of cardiac function were considered important since either a direct effect of acute compression or sustained pressure on cardiac pacemaker or conduction activity could lead to serious arrhythmia, and reflex effects via the central and autonomic nervous systems could influence the action of the heart as a pump. An acute decrease in peripheral vascular resistance from any cause, resulting in a transient reduction of venous return, might intensify any effects on the heart itself. If circulation to the brain or spinal cord were thereby compromised, central nervous system hypoxia might potentiate direct effects of hydrostatic pressure on neural excitable tissues. While compression of extreme degree induces cardiac arrest in animals (19), little information has been obtained concerning cardiac responses of man exposed to rapid compression and high pressures in various states of rest and exercise.

CLINICAL EVALUATION AND THE TIMING OF CARDIAC EVENTS

Heart rate and cardiac output are modified by numerous physiological influences (25), but the electrocardiogram shows pathological changes only when cardiac tissue itself is damaged or when its electrical properties have been altered. Electrocardiograms taken following exposure to helium-oxygen at 31 ata have been found to be normal compared with pre-exposure tracings (21). The absence of change in the electrocardiograms of the four subjects who participated in acute exposures to rapid compression in helium-oxygen and at pressures of 25, 37 and 49 ata in Phase II indicates that these exposures did not detectably alter the electrical properties of cardiac tissue. Similarly, the normal appearance of the electrocardiogram following saturation at the pressure equivalent to 1200 fsw for eight days in Phase II, and following decompression for all subjects in Phases I and II, indicates that prolonged exposure to high helium and hydrostatic pressures had no direct effect on the heart.

The absence of any abnormal changes in cardiac conduction and ejection times (Fig. 4) is consistent with the observation that the electrocardiograms were normal. The small changes observed in the PR interval were consistent with the minor normal effect of changes in heart rate on conduction time (27). The changes in QT interval were consistent with the normal inverse relationship between ejection time and heart rate (27).

CARDIAC FUNCTION AT REST

Mean heart rates of the subjects while seated before exercise and prior to standing during and after acute exposures to compression from 1 ata to 800 fsw changed in opposite directions in Phases I and II (Figs. 3 and 4). In Phase I, mean heart rate slowed during the compression from 1 ata to 800 fsw while in Phase II heart rate increased during the compression to the same depth. However, the initial mean heart rate at 1 ata was 75 beats/minute in Phase I, and 64 beats/minute in Phase II (Tables II and III). In both phases, heart rates settled to between 66 and 70 beats/minute during the stable "holds" at 800 fsw, falling to this level in Phase I and rising to this level

in Phase II. Compressions from 800 to 1200 fsw and from 1200 to 1600 fsw usually elicited small, transient increases in heart rate. Nearly all mean heart rates during exposure day 1 in both phases were between the reference basal and resting non-basal values. Furthermore, mean early morning heart rates on days 1 and 2 in Phase II were identical at a typical basal level despite a pressure ratio of 37:1 (Fig. 4).

These data of Predictive Studies IV are in agreement with other observations (8,22) which do not show a consistent pressure- or gas density-related bradycardia in man. Furthermore, mean heart rates measured early in the morning during the slow, multiday decompressions from saturation pressures equivalent to 800 and 1400 fsw to 1 ata in Phases I and II failed to show any evidence for a pressure-related bradycardia (Fig. 10).

Observations of cardiac slowing apparently related to compression or increased pressure (12,14,20,21,24,26,28) may instead be related to anticipation and general excitement causing an elevation of heart rate coincidental with the start of compression. As pressure and elapsed time increase, adaptation to the initial anticipation and excitement may occur and heart rate may decrease to normal values consistent with ongoing rest or activity levels. This sequence of events may have occurred in Phase I.

Mean heart rate in Phase II at 1 ata prior to compression was nearly at the early morning level (Fig. 4). This low initial level may reflect the fact that CC and FS had already participated as subjects in Phase I, while MP and GM had participated in Phase I as operations support personnel and thus were thoroughly familiar with the experiences of the other subjects. During compression to 800 fsw in Phase II, heart rates increased to the same levels as in Phase I, probably in response to the mild exertions of experiment-related activities.

Mean heart rates during excursions on days 2 and 5 in Phase I (Fig. 3) were close to the pre-compression level on day 1. Mean heart rates on days 2 and 3 in Phase II (Fig. 4) began at approximately the same level reached during compressions on day 1, accelerated during compressions, and decreased to levels more nearly typical of resting man

during the excursions. This may be partially explained by the fact that, prior to compression from 1 ata, the subjects had the full assistance of investigative personnel in applying electrodes and otherwise preparing for the day's activities. On subsequent excursion days, however, the subjects themselves converted the pressure chamber from sleeping quarters to a complex experiment laboratory, applied electrodes, and prepared and checked out all internal experiment instrumentation.

Patterns of change in cardiac output on any single day are reliable since the electrodes remained in place throughout a day's measurements. In addition, early morning values of cardiac output (using the custom-fitted elastic vests with sewn-in electrode tapes) were probably comparable from day to day; they were between typical basal and seated values (Fig. 4). Values during the experiment modules were usually somewhat higher than early morning values; this was expected since the subjects were actively preparing for and carrying out experiment procedures subsequent to early morning measurements.

Neither heart rate nor cardiac output changes were suggestive of hypotension while at rest during the compression-pressure exposures. This is particularly relevant to periods when signs and symptoms of acute exposure to high pressure were most severe (Section E-1). Alveolar carbon dioxide tensions were in the normocapnic range and alveolar oxygen tensions were in the normoxic range in the two subjects in whom these parameters were measured in Phase II (CC and GM, Sections E-14 and E-16). With unimpaired circulation it can be presumed that the acid-base state and oxygenation of excitable tissue were within the normal range and did not contribute to the signs and symptoms associated with compression and pressure.

Heart rates measured early in the morning varied from day to day, showed no consistent patterns of change, and were usually between the reference basal and resting non-basal values (1) in Phases I and II (Fig. 10). Cardiac output in the early morning (Phase II) was within the range of reference basal and seated values obtained by dye dilution methods (1,10,17). Several values of cardiac output obtained early in the exposure (days 2, 3 and 4, Fig. 10

and Table XIII) are higher than the other measurements. The elevated cardiac outputs on days 3 and 4 were associated with higher heart rates as well (Table XIII; see also Section E-15).

RESPONSES AT REST, DURING EXERCISE, AND FOLLOWING FOUR MINUTES OF RECOVERY

Individual cardiac rate and output responses of subjects at rest prior to exercise followed patterns similar to those of the mean values during the compression-pressure exposures (Figs. 3-6). Cardiac responses during exercise closely followed the patterns of change in oxygen consumption, indicating no major effect of compression and pressure. Cardiac recovery from the influences of exercise was usually complete or nearly so four minutes after cessation of exercise.

The relationships between cardiac function and oxygen consumption can be appreciated best by inspection of Fig. 7. Although the bicycle ergometer workload setting was constant, pedaling rate variations caused workload and, therefore, oxygen consumption to vary over a wide range (Section E-14). Pedaling rate was not synchronized to audible or visual cues because this procedure would have interfered with communications and other aspects of the experiment program (Section E-14).

In comparing heart rate and cardiac output responses during the compression-pressure exposures with measurements obtained during progressively increased exercise levels at 1 ata, the slope of the heart rate responses is more important than absolute levels of heart rate and cardiac output which may vary from day to day (absolute levels of cardiac output measured by the impedance cardiograph method may also change from one day to another due to variations in electrode placement).

All heart rate responses to change in oxygen consumption were parallel or nearly parallel to the pre-exposure responses in Phases I and II. The cardiac output responses of subjects CC and WS also were nearly parallel to the responses obtained at 1 ata. Subject GM's cardiac output responses in Phase II exposures had a lower slope than at 1 ata. However, the heart rates obtained in this subject

at 1 ata were unusually high even at rest, and the slope of his cardiac output response to exercise at 1 ata was atypically large. Actually, GM's cardiac output responses to exercise in Phase II were comparable in slope with those of the other subjects.

The ventilatory responses of these subjects to exercise were also very nearly the same during the pressure exposures as during progressive exercise at 1 ata (Section E-14). End-tidal gas tensions during exercise were within the normal range (Section E-14). Thus, rapid compression in normoxic helium-oxygen to pressure equivalents of 800-1200-1600 fsw did not appear to compromise the functional effectiveness of the cardiopulmonary functions of gas exchange, circulation and tissue perfusion, either at rest or during light exercise.

CARDIAC RESPONSE TO POSTURAL CHANGE

Dizziness and lightheadedness are among the symptoms associated with compression to high pressures. Similar symptoms occur in individuals suffering from "orthostatic hypotension" (25). Such individuals have an inadequate vasomotor responsiveness to the abrupt fall in blood pressure when they stand.

It was considered possible that the relatively sudden rise in ambient and skin temperatures during compressions from 1 ata to 800 and 1200 fsw on the first exposure days (Section E-15) might result in lowered peripheral resistance (Section E-15), at least until adaptation could occur. If hydrostatic pressure effects directly on vascular smooth muscle or indirect effects via the sympathetic nervous system were to reduce vasomotor tone, hypotension might contribute to the symptoms of compression and pressure. Although the active conditions involved in the measurements in this study were different from a precisely controlled tilt-table test, the method provided cardiac response to abrupt change in gravitational stress on standing. It was considered that in the presence of functional hypotension or an inadequate vasomotor response to orthostatic stress in any of the subjects studied (LJ and FS in Phase I, MP and FS in Phase II),

this test should have exaggerated the effects of such conditions and induced dizziness, lightheadedness and possibly even syncope.

However, syncope was not experienced by any subject either spontaneously or as a result of the postural change experiments. While eight incidents of dizziness or "lightheadedness" were reported by the rest subjects in Phases I and II (Figs. 8 and 9), these reports did not indicate faintness and they correlated only once with decrements in the heart rate response to gravitational stress (Fig. 9, subject FS). Furthermore, the heart rate response index did not correlate with quantitative measurements of body sway (Section E-6). Finally, the exercise subjects, who intermittently performed pedaling exercises but did not undergo postural change, reported symptoms of dizziness or lightheadedness as frequently as did the subjects who stood quietly at rest (Section E-1). It is therefore apparent that neither spontaneous nor postural hypotension was a likely factor in producing the symptoms and signs of compression to high pressure in the subjects of Predictive Studies IV.

CONCLUSIONS

Rapid compressions and exposures to pressures of 25, 37 and 49 ata in helium-oxygen did not alter the electrocardiogram or affect cardiac conduction or ejection times. There was no consistent evidence of a pressure-related bradycardia in the subjects of these studies. Heart rates were quantitatively appropriate for activity levels early in the morning shortly after awakening, both while seated and while engaged in light activities, and during light exercise on a bicycle ergometer. Cardiac outputs estimated indirectly were also usually at appropriate levels. The expected heart rate response to postural change was present, with a few exceptions during periods of the more severe exposures. Only a single incident of dizziness and lightheadedness reported by the subjects participating in the postural change experiments was associated with a large decrement in the chronotropic response to standing erect.

It is concluded that neither compression nor hydrostatic effects in combination with high helium concentrations

appreciably altered cardiovascular functions in the subjects of Predictive Studies IV. In the presence of normal ventilatory and cardiac function, compression-pressure effects on excitable tissues were probably not exacerbated by local derangements of oxygenation or acid-base state due to disturbances in perfusion.

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APPENDIX E-13

APPENDIX TABLE 1. Heart Rate During Compression-Pressure Exposures to 800 and 1200 fsw (Phase I, Individual Values for Subjects at Rest)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Subject				Mean
			CC	LJ	FS	WS	
Heart Rate (beats/min)							
1	0	0	67	80	72	79	75
	400-488	0:26	65	73	66	70	69
	600-652	0:46	65	70	66	69	68
	690-738	1:06	65	66	59	74	66
	800	1:28	57	77	56	74	66
	800	3:34	64	91	55	72	71
	800-980	3:58	79	91	59	79	77
	1103-1148	4:23	65	82	62	77	72
	1050-1005	5:02	67	80	61	77	71
	888-865	5:42	60	73	54	73	65
	800	6:30	60	81	61	61	66
2	800	0	73	75	79	75	76
	800-980	0:05	80	84	73	73	78
	1090-1157	0:25	78	73	64	71	72
	1200	0:49	79	71	68	67	71
	1050-1010	1:28	74	80	65	66	71
	886-862	2:09	69	87	68	60	71
	800	2:52	67	87	74	66	74
5	800	0	72	83	-	-	-
	800-980	0:05	74	73	-	-	-
	1100-1145	0:25	71	72	-	-	-
	1200	0:56	70	76	-	-	-
	1050-1010	1:45	77	72	-	-	-
	892-865	2:25	63	-	-	-	-
Post-exp.	0	-	83	85	84	85	84

APPENDIX TABLE 2. Cardiac Output During Compression-Pressure Exposures to 800 and 1200 fsw (Phase I, Individual Values for Subjects at Rest)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Subject				Mean
			CC	LJ	FS	WS	
Cardiac Output (l/min)							
1	0	0	6.9	7.6	8.5	9.0	8.0
	400-488	0:26	7.6	-	7.4	8.2	-
	600-652	0:46	6.2	-	6.1	7.8	-
	690-738	1:06	6.1	7.8	5.5	9.3	7.2
	800	1:28	5.2	8.0	5.7	7.8	6.7
	800	3:34	8.0	10.9	6.8	11.9	9.4
	800-980	3:58	7.7	-	7.0	7.5	-
	1103-1148	4:23	5.4	6.8	6.8	7.9	6.7
	1050-1005	5:02	7.1	9.6	13.1	-	-
	888-865	5:42	6.8	6.6	-	-	-
	800	6:30	5.1	9.2	-	-	-
2	800	0	-	6.9	6.7	-	-
	800-980	0:05	-	6.3	5.7	-	-
	1090-1157	0:25	-	6.6	6.2	-	-
	1200	0:49	-	5.8	7.5	-	-
	1050-1010	1:28	-	6.0	6.5	-	-
	886-862	2:09	-	6.2	6.2	-	-
	800	2:52	-	6.7	6.4	-	-
5	800	0	9.7	9.1	-	-	-
	800-980	0:05	8.9	5.4	-	-	-
	1050-1010	1:45	-	5.7	-	-	-
Post-exp.	0	-	-	4.7	8.0	-	-

APPENDIX TABLE 3. Heart Rate During Compression-Pressure Exposures to 800, 1200 and 1600 fsw (Phase II, Individual Values for Subjects at Rest)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Subject				Mean
			CC	MP	GM	FS	
Heart Rate (beats/min)							
Pre-exp.	0	-	77	60	78	64	70
1	0	-	68	67	57	67	65
	0	-	65	64	65	62	64
	0	0	64	63	-	-	-
	400-550	0:18	66	65	88	62	70
	654-724	0:37	72	62	86	84	76
	800	0:48	58	58	89	66	68
	800	0:59	70	56	84	68	70
	800	1:10	-	60	72	62	65
	800	2:18	65	60	77	62	66
	860-1020	2:58	72	63	71	66	68
	1110-1155	3:17	73	61	92	63	72
	1200	3:37	80	71	-	-	-
	1200	3:48	88	63	66	59	69
	1200	3:59	82	58	-	-	-
	1200	4:47	-	-	79	68	-
	1200	5:14	75	64	66	60	66
	1200	5:25	-	-	60	54	-
	1200	6:10	-	-	58	53	-
	1200	7:16	75	62	-	-	-
	1200	7:25	61	53	54	62	58
	1200	7:43	67	50	-	-	-
	1200	8:02	-	-	67	73	-
2	1200	-	74	60	85	64	71
	1200	0	66	64	82	67	70
	1270-1470	0:07	72	66	90	68	74
	1510-1555	0:27	-	-	90	61	-
	1600	0:40	68	63	80	65	69
	1420-1375	1:31	61	59	81	65	66
	1285-1265	2:10	52	60	71	60	61
	1200	3:18	61	58	56	60	59
	1200	3:33	54	69	-	55	59
3	1200	-	94	80	-	73	82
	1200	0	70	76	73	69	72
	1280-1510	0:07	82	77	95	87	85
	1600	0:30	85	69	99	80	83
	1600	1:01	89	70	-	60	73
	1467-1395	1:21	73	64	88	63	72
	1285-1263	2:00	62	61	70	64	64
	1200	2:51	69	61	65	57	63
	1200	3:25	67	-	-	59	-
8	1200	-	70	54	75	56	64
	1200	-	60	58	72	65	64
	1200	-	56	65	-	-	-
Post-exp.	0	-	84	82	-	73	80
	0	-	71	79	61	75	72

APPENDIX TABLE 4. Cardiac Output During Compression-Pressure Exposures to 800, 1200 and 1600 fsw (Phase II, Individual Values for Subjects at Rest)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Subject				Mean
			CC	MP	GM	FS	
Cardiac Output (l/min)							
Pre-exp.	0	-	10.0	7.3	5.2	6.9	7.5
1	0	-	8.0	-	8.0	9.6	-
	0	-	7.9	7.5	7.9	9.6	7.8
	0	0	7.0	7.2	-	-	-
	400-550	0:18	8.2	8.7	11.6	-	9.5
	654-724	0:37	8.4	7.9	10.6	-	9.0
	800	0:59	8.8	6.2	10.0	-	8.3
	800	2:18	8.2	6.7	9.9	-	8.3
	860-1020	2:58	10.4	7.2	9.7	-	9.1
	1110-1155	3:17	8.3	5.9	12.5	-	8.9
	1200	3:48	10.9	7.0	-	-	-
	1200	5:14	9.8	7.5	13.5	-	10.3
	1200	7:25	9.6	7.7	-	-	-
2	1200	-	-	-	8.3	7.8	-
	1200	0	9.1	7.6	8.9	8.9	8.6
	1270-1470	0:07	7.7	8.3	9.8	13.0	9.7
	1510-1555	0:27	-	-	10.3	8.3	-
	1600	0:40	8.9	7.1	8.5	9.0	8.4
	1420-1375	1:31	7.1	7.6	9.2	8.3	8.1
	1285-1265	2:10	6.9	7.1	8.7	9.3	8.0
	1200	3:18	8.0	6.7	7.8	9.3	8.0
3	1200	0	10.4	6.4	-	7.8	8.2
	1280-1510	0:07	11.8	7.0	10.5	10.8	10.0
	1600	0:30	10.4	6.4	9.6	7.8	8.6
	1467-1395	1:21	8.8	6.0	9.4	7.6	8.0
	1285-1263	2:00	7.9	5.6	8.2	7.8	7.4
	1200	2:51	8.8	5.1	-	6.9	6.9
8	1200	-	-	-	8.0	8.1	-
	1200	-	11.1	9.2	10.9	9.2	10.1
	1200	-	12.5	8.6	-	-	-
Post-exp.	0	-	8.3	7.3	9.3	7.2	8.0

APPENDIX TABLE 5. Cardiac Conduction Time During Compression-Pressure Exposures to 800, 1200 and 1600 fsw (Phase II, Individual Values for Subjects at Rest)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Subject				Mean
			CC	MP	GM	FS	
PR Interval (sec)							
1	0	-	0.22	0.21	0.20	0.19	0.21
	0	0	0.20	0.19	0.20	0.17	0.19
	400-550	0:18	0.22	0.19	0.19	0.17	0.19
	654-724	0:37	0.21	0.17	0.21	0.17	0.19
	800	0:59	0.21	0.19	0.18	0.20	0.20
	800	2:18	0.21	0.19	0.20	0.17	0.19
	860-1020	2:58	0.22	0.18	0.19	0.18	0.19
	1110-1155	3:17	0.20	0.19	0.19	0.18	0.19
	1200	5:14	0.20	0.20	0.22	0.17	0.20
	1200	7:25	0.21	0.19	0.20	0.18	0.20
2	1200	0	0.23	0.18	0.17	0.16	0.19
	1270-1470	0:07	0.19	0.18	0.21	0.16	0.19
	1600	0:40	0.22	0.20	0.20	0.18	0.20
	1420-1375	1:31	0.21	0.17	0.19	0.19	0.19
	1285-1265	2:10	0.23	0.20	0.21	0.17	0.20
	1200	3:18	0.22	0.19	0.18	0.17	0.19
3	1200	0	0.19	0.18	0.19	0.17	0.18
	1280-1510	0:07	0.21	0.23	0.18	0.17	0.20
	1600	0:30	0.19	0.18	0.18	0.17	0.18
	1467-1395	1:21	0.21	0.19	0.19	0.19	0.20
	1285-1263	2:00	0.24	0.20	0.20	0.18	0.21
	1200	2:51	0.23	0.20	0.20	0.17	0.20
8	1200	-	0.24	0.20	0.20	0.18	0.21
Post-exp.	0	-	0.19	0.18	0.19	0.19	0.19

APPENDIX TABLE 6. Cardiac Ejection Time During Compression-Pressure Exposures to 800, 1200 and 1600 fsw (Phase II, Individual Values for Subjects at Rest)

Exposure Day	Depth (fsw)	Elapsed Time (hr:min)	Subject				Mean
			CC	MP	GM	FS	
QT Interval (sec)							
1	0	-	0.35	0.42	0.38	0.39	0.39
	0	0	0.36	0.37	0.37	0.36	0.37
	400-550	0:18	0.37	0.38	0.33	0.36	0.36
	654-724	0:37	0.35	0.42	0.35	0.39	0.38
	800	0:59	0.36	0.43	0.35	0.40	0.39
	800	2:18	0.37	0.41	0.35	0.40	0.38
	860-1020	2:58	0.33	0.37	0.37	0.35	0.36
	1110-1155	3:17	0.36	0.40	0.35	0.40	0.38
	1200	5:14	0.35	0.40	0.40	0.40	0.39
	1200	7:25	0.38	0.42	0.41	0.40	0.40
2	1200	0	0.35	0.39	0.34	0.39	0.37
	1270-1470	0:07	0.34	0.37	0.29	0.36	0.34
	1600	0:40	0.37	0.37	0.36	0.39	0.37
	1420-1375	1:31	0.35	0.40	0.34	0.36	0.36
	1285-1265	2:10	0.40	0.39	0.35	0.40	0.39
	1200	3:18	0.39	0.41	0.38	0.39	0.39
3	1200	0	0.35	0.33	0.36	0.37	0.35
	1280-1510	0:07	0.32	0.37	0.34	0.37	0.35
	1600	0:30	0.35	0.37	0.33	0.36	0.35
	1467-1395	1:21	0.36	0.37	0.35	0.35	0.36
	1285-1263	2:00	0.39	0.41	0.37	0.39	0.39
	1200	2:51	0.38	0.39	0.40	0.41	0.40
8	1200	-	0.37	0.40	0.37	0.38	0.38
Post-exp.	0	-	0.35	0.35	0.37	0.34	0.35

E-14. VENTILATORY AND METABOLIC RESPONSES TO EXERCISE DURING RAPID COMPRESSION TO EXTREME PRESSURES

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Previous studies have shown that man's capacity for physical work is not seriously impaired by exposure to dense inspired gases and high ambient pressures (2,9,12). During a saturation exposure in a normoxic helium-oxygen atmosphere at an ambient pressure equivalent to 1200 fsw, two well-conditioned subjects successfully completed 24 continuous minutes of exercise at sequential workloads of 300,600,900 and 1200 kilopond-meters/minute (kpm/min) (9). The same subjects also completed all but the highest workload at the same depth while breathing a neon-helium-oxygen mixture with a density equivalent to that of helium-oxygen at 5000 fsw (ca. 25 g/l). These efforts were possible despite the expected reduction in ventilation and elevation of alveolar P_{CO_2} which accompanied the increased respiratory gas density. Similar responses to a lower range of workloads (275-735 kpm/min) were found in three subjects during a saturation exposure to helium-oxygen at a pressure equivalent to 1000 fsw (12). Relatively slow compressions with multiple stages over several days were used for both saturation exposures.

While the high work capacities cited have been demonstrated by some subjects at density equivalents of great pressures (9), in others even light work appeared to induce symptoms of fatigue and dyspnea with a sensation of impending loss of consciousness (1,14). In pressure exposures at two

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different laboratories these symptoms occurred at approximately 1300-1600 fsw following compressions at rates greater than those used for the previously described saturation exposures (9,12). The symptoms were not explained by respiratory insufficiency; measurements showed that ventilatory responses to light exercise were similar to those found at sea level (10), and that arterial P_{CO_2} was not elevated (14). Even at rest the same subjects experienced the diverse neurological, psychomotor and subjective effects which have been associated with rapid compression and exposure to extreme hydrostatic pressures (8). It is conceivable that such pressure effects may induce symptoms by altering normal cardiopulmonary responses to exercise. Moreover, the increased neuromuscular activity of exercise may possibly exacerbate concurrent hydrostatic effects.

To identify possible interactions between compression and pressure on the one hand, and exercise responses on the other, respiratory function at rest and during exercise was evaluated by taking measurements of ventilation, gas exchange, end-tidal respiratory gases and diaphragmatic electrical activity before, during, and after compression from 0 to 400-800-1200-1600 fsw.

METHODS

The overall study was conducted in two phases with two exercise subjects in each phase. Phase I consisted of rapid compression from sea level to a pressure equivalent to 800 fsw with subsequent saturation at that depth and repetitive excursions to 1200 fsw. Although the primary objective of this phase was to evaluate decompression procedures, most of the scheduled respiratory function measurements were also obtained. Phase II consisted of rapid compression to a saturation depth of 1200 fsw with repetitive excursions to 1600 fsw. All scheduled respiratory function measurements were performed in this phase except on day 1 when difficulties precluded complete data acquisition in one subject (GM) at the peak of compression effect.

Measurements of respiratory function were made repetitively at sea level, during compression and at stable elevated pressures as part of a series of 17-minute modules which included many nonrespiratory measurements (Section D).

For the exercise subjects each module was partitioned into seven minutes of rest, six minutes of exercise and four minutes of recovery. Gas exchange was measured during the last two minutes of the rest and exercise periods; other parameters were measured in all three periods. Throughout each module, the subject sat on a bicycle ergometer (Collins Pedal-Mode) with the workload set at 450 kpm/min (75 watts).

Ventilatory measurements were not made during decompression while the subjects received a hyperoxic breathing gas by face mask. Exercise was not performed either during decompression or immediately after its completion, since this might have induced decompression sickness.

SUBJECTS

All subjects were professional divers with underwater oil-field experience. They were physically fit and were encouraged to exercise regularly prior to the study. Vital data are given in Section D.

Maximum work capacity of each subject was determined prior to Phase I by a procedure which involved the performance of consecutive three-minute workloads on the ergometer with progressive increments of 300 kpm/min to the point of exhaustion. Ventilatory and metabolic responses to exercise were measured in subjects CC and WS in Phase I and in subjects CC and GM in Phase II of the compression-pressure exposures.

BREATHING GAS ADMINISTRATION AND COLLECTION

A low resistance, low dead-space (60 cc), non-rebreathing valve assembly constructed in this laboratory consisted of two Collins "V" valves mounted in a nylon "Y" hose fitting (3.2 cm i.d.). The inspiratory side of the valve was open to the chamber atmosphere, while the expiratory side was connected to a 20-liter mixing chamber by a 1.4-meter length of flexible tubing (5 cm i.d.). A 0.6-meter length of the same tubing connected the outflow from the mixing chamber to a pneumotachograph assembly designed for use with high flow rates and high density gases (6).

Total pressure drops across both inspiratory and expiratory sides of the breathing gas system were measured at constant flow rates ranging to more than 360 1/min (Fig. 1). Data were obtained for air at sea level and for He at 800, 1200 and 1600 fsw. The resulting curves may be used to estimate maximum resistance to breathing for a known peak flow at any of these ambient pressures.

Mean expired gas samples were withdrawn from the mixing chamber outflow. Inspired gas was sampled from the chamber atmosphere near the inspiratory side of the non-rebreathing valve assembly, and end-tidal gas samples were drawn from the Y fitting between the inspiratory and expiratory valves.

VENTILATION MEASUREMENTS

Expired volume was obtained by integration of the flow measured by the pneumotachograph system. Flow was obtained as differential pressure measured with transducers (Validyne, models DP45 and DP103). Integrator output on a strip chart recorder was analyzed for expired minute volume ($\dot{V}_E$) and respiratory frequency (f). Tidal volume (V_T) was calculated as $\dot{V}_E/f$ and all volumes were corrected to BTPS conditions. The pneumotachograph assembly, pressure transducer and integrator were calibrated before and after each module by repeated injections of chamber gas from a one-liter syringe.

RESPIRATORY GAS MEASUREMENTS

Inspired, end-tidal and mean expired gas samples were analyzed for P_{O_2} and P_{CO_2} at sea level with rapid response gas analyzers (Applied Electrochemistry, model S-3A oxygen analyzer; Beckman LB-2 carbon dioxide analyzer). For analysis of gas samples collected at high ambient pressures the basic sensitivity of each analyzer was increased by use of an external amplifier, which also provided zero suppression. Nylon tubing, which was found to be satisfactorily impermeable to gases, was used to conduct gas samples from points of collection inside the chamber to the chamber hull, and from the hull to analyzers outside the chamber. Both analyzers were calibrated before and after each module.

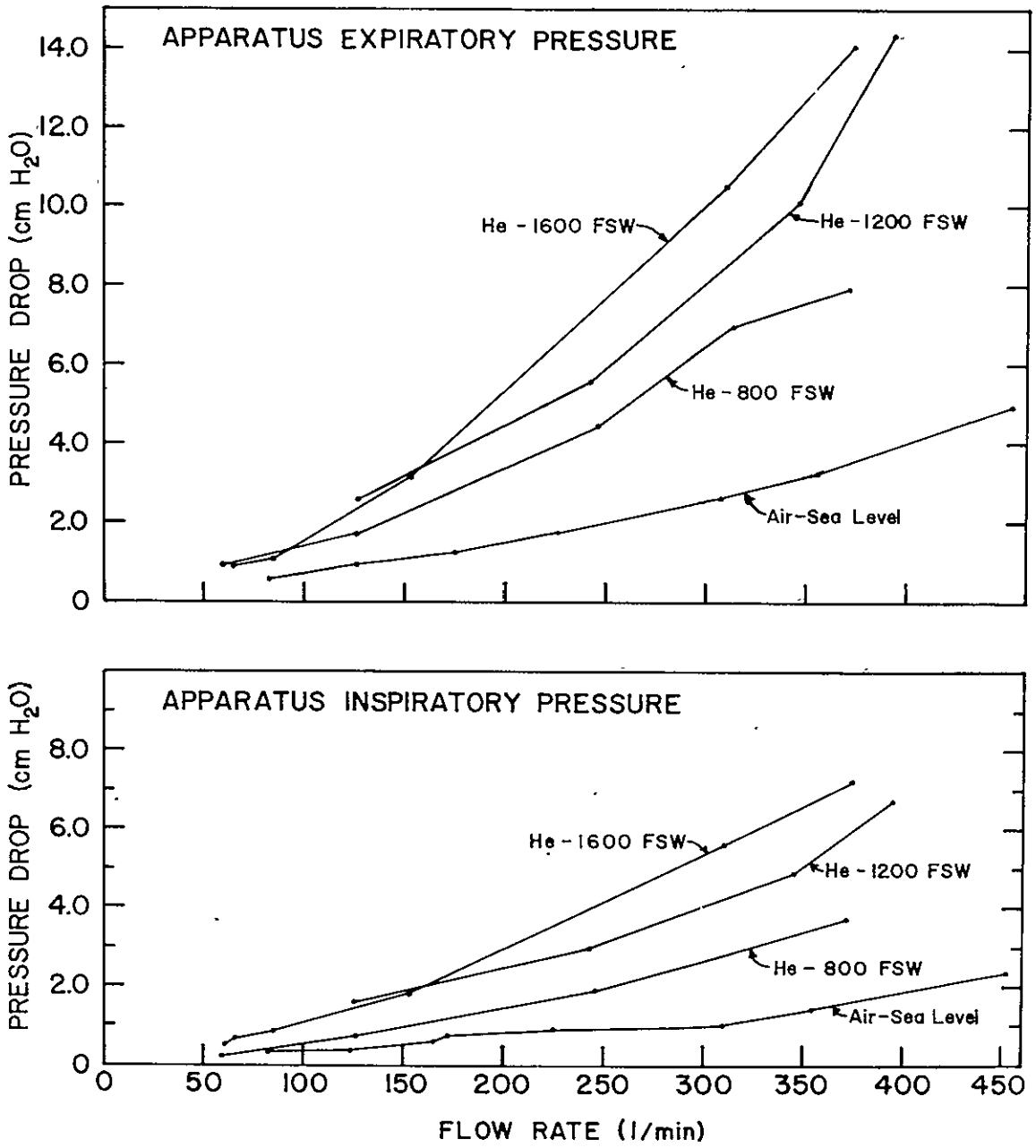

FIG. 1. Effect of flow rate and ambient pressure (gas density) on total pressure drop across inspiratory and expiratory sides of the breathing gas administration system. Pressure differentials were measured with a water manometer at constant flow rates ranging to more than 360 l/min. Flow rate refers to actual flow through the pneumotachograph inside the chamber, corrected to 37°C.

Calibration gases were appropriately premixed for each operational pressure range, and their compositions were carefully analyzed by gas chromatography with gravimetrically prepared gas mixtures used as primary standards. The maximum calibration error at the highest pressure was ±1 mm Hg. Oxygen and carbon dioxide gas exchange volumes were corrected to STPD conditions. End-tidal P_{O_2} and P_{CO_2} were obtained for each breath as the peaks of the end-expiratory plateaus and are reported as estimated alveolar values ($P_{A_{O_2}}$, $P_{A_{CO_2}}$). Methods for end-tidal measurements are elaborated in Section E-16.

DIAPHRAGMATIC ELECTROMYOGRAM (EMG)

Electrical activity of the diaphragm (EMG) was monitored by three 5 mm electrode coils wound onto one end of a 76 cm vinyl catheter (2.5 mm o.d.; 1.5 mm i.d.) which contained a thermistor for measurement of the esophageal temperature (Section E-15). The catheter was introduced through the nose into the esophagus to the level of the diaphragm as determined by maximum EMG signal level. Electrical signals were recorded differentially from two wire coils spaced 8 mm on either side of a third, reference coil. The signal, amplified by a battery-powered low noise preamplifier (MED Associates, model 110) followed by a power line isolation amplifier (Analog Devices, model 273K), was recorded on magnetic tape. The taped signal was subsequently transferred onto a second magnetic tape recorder at 60 inches/second and then played back at 15/16 inches/second onto an oscillographic strip chart recorder for analysis.

RESULTS

PRELIMINARY MEASUREMENTS OF EXERCISE CAPACITY

Before the two exercise subjects for Phase I were selected, maximum work capacities of all six subjects were determined on the bicycle ergometer (Table I). Maximum $\dot{V}_{O_2}$ levels for subjects GM, FS and WS were approximately halfway between values found in untrained college students

and endurance-trained athletes (11). Although the maximum $\dot{V}_{O_2}$ of subject CC was relatively low when compared with the other diver-subjects, it still indicated a higher than average level of physical fitness.

TABLE I. Individual Values at Maximum Work Capacity

Subject	Workload (kpm/min)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{O_2}$ (ml/kg/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)
CC	1500	4.15	50.9	4.62	1.11	182.1	3.31	55
LJ	1500	3.70	51.0	3.86	1.04	160.5	2.51	64
GM	1800	5.01	61.0	4.37	0.87	157.8	3.16	50
MP	1500	4.13	55.4	4.52	1.11	170.3	3.70	46
FS	1800	4.35	61.8	4.24	0.98	155.4	2.68	58
WS	1800	5.33	63.4	4.95	0.93	207.7	3.06	68
Mean		4.45	57.2	4.43	1.01	172.3	3.07	56
±SD		0.61	5.6	0.37	0.10	20.0	0.43	8
±SEM		0.25	2.3	0.15	0.04	8.2	0.18	3

MEASUREMENTS DURING STUDY OF COMPRESSION EFFECTS

Data obtained under conditions of rest, exercise and recovery as part of the repetitive modules used to track compression effects are summarized in Tables II and III for Phase I (subjects CC and WS) and in Tables IV and V for Phase II (subjects CC and GM). Elapsed times at the middle of the intervals for gas exchange measurements at rest are given in these tables. Times for exercise and recovery are 6 and 10 minutes later, respectively. Pressures shown correspond to these times.

TABLE II. Ventilation and Gas Exchange Responses to Exercise During Compression and

Exposure Day	Elapsed Time[a] (hr:min)	REST Depth (fsw)	REST $P_{I_{O_2}}$ (mm Hg)	REST $\dot{V}_E$ (l/min BTPS)	REST V_T (l BTPS)	REST f (br/min)	REST $\dot{V}_{O_2}$ (l/min STPD)	REST $\dot{V}_{CO_2}$ (l/min STPD)	REST R
1	0	0	-	13.19	0.69	19	0.34	0.33	0.94
	0:26	460	-	7.32	0.43	17	-	-	-
	0:46	630	-	11.27	0.66	17	-	-	-
	1:06	730	181	11.04	0.69	16	0.32	0.30	0.94
	1:26	800	188	9.44	0.56	17	0.28	0.24	0.87
	2:00	800	181	12.72	0.75	17	0.38	0.32	0.82
	3:44	800	166	11.74	0.69	17	0.30	0.32	1.06
	4:20	940	162	13.50	0.79	17	0.34	0.38	1.13
	4:40	1135	166	11.46	0.72	16	0.32	0.29	0.90
	6:47	800	156	10.13	0.56	18	0.32	0.26	0.81
	6:53	800	154	11.12	0.62	18	0.32	0.27	0.85
	6:57	800	-	9.58	0.60	16	-	-	-
2	0	800	166	12.18	0.72	17	0.28	0.30	1.08
	0:06	920	173	13.44	0.75	18	0.29	0.35	1.20
	0:26	1130	163	13.73	0.81	17	0.40	0.33	0.82
	0:49	1200	171	12.96	0.68	19	0.36	0.31	0.87
	3:04	800	160	10.61	0.56	19	0.29	0.24	0.83
	3:10	800	160	11.11	0.62	18	0.30	0.25	0.82
	3:14	800	-	11.13	0.59	19	-	-	-
5	0	800	166	14.61	0.77	19	0.34	0.37	1.11
	0:06	920	170	14.12	0.74	19	0.31	0.38	1.22
	0:26	1130	158	12.16	0.76	16	0.25	0.31	1.22
	0:49	1200	157	12.62	0.74	17	0.25	0.31	1.22
Post-exp.	-	0	-	19.99	0.95	21	-	-	-

[a]Elapsed time is the middle of the interval for gas exchange measurements at rest; mid-point of the exercise measurements is 6 minutes later; recovery is 10 minutes later.

Exposure to Pressures Equivalent to 800 and 1200 fsw (Subject CC, Phase I)

EXERCISE								RECOVERY			
Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	Depth (fsw)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)
0	-	34.57	1.44	24	1.15	1.15	1.00	0	13.83	0.73	19
520	-	32.36	1.54	21	-	-	-	560	12.82	0.64	20
660	-	32.44	1.35	24	-	-	-	680	11.87	0.70	17
760	179	34.99	1.52	23	1.34	1.21	0.91	780	12.04	0.75	16
800	168	33.37	1.45	23	1.30	1.12	0.86	800	12.73	0.71	18
800	181	33.85	1.47	23	1.26	1.10	0.88	800	13.69	0.76	18
800	164	34.78	1.51	23	1.13	1.20	1.07	800	10.67	0.67	16
-	-	-	-	-	-	-	-	1070	10.42	0.61	17
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
800	166	42.16	2.11	20	1.29	1.43	1.11	800	14.30	0.80	18
1020	164	38.28	1.66	23	1.41	1.28	0.91	1060	13.96	0.78	18
1160	158	38.84	1.62	24	1.34	1.27	0.95	1180	13.63	0.76	18
1200	171	48.12	1.85	26	1.66	1.58	0.95	1200	16.36	0.82	20
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
800	166	39.68	1.72	23	1.23	1.30	1.05	800	13.77	0.76	18
1020	165	31.48	1.75	18	1.11	1.07	0.96	1060	14.55	0.81	18
1160	156	56.09	2.16	26	1.69	2.01	1.19	1180	17.58	0.92	19
1200	166	77.46	2.58	30	2.32	2.59	1.12	1200	19.71	0.99	20
0	-	62.20	2.14	29	1.74	1.77	1.02	0	18.48	0.92	20

TABLE III. Ventilation and Gas Exchange Responses to Exercise During Compression and

Exposure Day	Elapsed Time[a] (hr:min)	REST							
		Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R
1	0	0	-	11.80	0.66	18	0.35	0.33	0.95
	0:28	460	-	9.24	0.62	15	-	-	-
	0:48	630	-	11.91	0.79	15	-	-	-
	1:08	730	-	10.79	0.77	14	-	-	-
	1:32	800	-	11.42	0.82	14	-	-	-
	2:06	800	186	8.68	0.58	15	0.28	0.23	0.81
	3:25	800	-	14.65	0.86	17	-	-	-
	3:47	920	177	13.79	0.81	17	0.30	0.36	1.20
	4:09	1130	176	10.74	0.77	14	0.28	0.30	1.05
	6:15	800	171	10.96	0.61	18	0.35	0.28	0.81
	6:21	800	171	9.28	0.52	18	0.28	0.23	0.81
	6:25	800	-	10.13	0.68	15	-	-	-
2	0	800	170	10.52	0.62	17	0.27	0.26	0.96
	0:06	920	166	11.28	0.63	18	0.32	0.33	1.03
	0:26	1130	161	9.45	0.59	16	0.26	0.24	0.93
	0:51	1200	163	8.64	0.58	15	0.26	0.22	0.84
	2:43	800	161	7.51	0.54	14	0.22	0.18	0.82
	2:49	800	161	10.67	0.67	16	0.32	0.24	0.77
	2:53	800	-	9.87	0.62	16	-	-	-
Post-exp.		0	-	14.97	1.00	15	0.52	0.41	0.78

[a]Elapsed time is the middle of the interval for gas exchange measurements at rest; mid-point of the exercise measurements is 6 minutes later; recovery is 10 minutes later.

Exposure to Pressures Equivalent to 800 and 1200 fsw (Subject WS, Phase I)

EXERCISE								RECOVERY			
Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	Depth (fsw)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)
0	-	34.10	1.42	24	1.14	1.10	0.96	0	11.63	0.65	18
520	192	44.20	2.01	22	1.43	1.58	1.10	560	14.09	0.88	16
660	-	50.81	2.31	22	-	-	-	680	14.27	0.84	17
760	-	55.19	2.30	24	-	-	-	780	13.12	0.88	15
800	188	45.35	2.06	22	1.00	1.57	1.58	800	10.42	0.65	16
800	186	69.81	2.68	26	2.58	2.33	0.90	800	16.18	0.90	18
800	182	46.48	2.21	21	1.79	1.52	0.85	800	16.06	0.89	18
1020	181	49.74	2.49	20	2.07	1.65	0.80	1060	14.85	0.87	17
1160	176	50.38	2.40	21	1.82	1.70	0.94	1180	18.30	1.02	18
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
800	172	42.87	2.14	20	1.68	1.50	0.89	800	14.73	0.82	18
1020	164	51.36	2.34	22	2.05	1.81	0.88	1060	16.25	0.96	17
1160	162	55.60	2.32	24	2.28	1.94	0.85	1180	15.75	0.93	17
1200	163	50.26	2.39	21	2.04	1.72	0.84	1200	12.75	0.75	17
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-
0	-	47.16	2.05	23	1.30	1.48	1.14	0	14.01	0.88	16

TABLE IV Ventilation and Gas Exchange Responses to Exercise During Compression and Exposure to Pressures Equivalent to 800, 1200

		REST										EXERCISE		
Exposure Day	Elapsed Time[a] (hr·min)	Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	$P_{A_{O_2}}$ (mm Hg)	$P_{A_{CO_2}}$ (mm Hg)	Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)
1	0	0	-	12.71	0.67	19	0.38	0.33	0.87	107	37.8	0	-	54.56
	0:19	504	172	14.50	0.76	19	-	-	-	151	-	600	169	39.24
	0:38	704	168	12.33	0 65	19	0.27	0.31	1 15	140	40.2	752	168	58.97
	0:59	800	-	10 82	0.64	17	-	-	-	-	-	800	176	56 40
	2·20	800	166	13.60	0.80	17	0.35	0.34	0.96	128	-	800	167	40.14
	2:59	980	167	15.52	0.82	19	0.33	0.40	1 23	134	-	1050	167	48.48
	3:18	1140	171	18 09	1.00	18	0.52	0.50	0.94	133	44.4	1170	174	65.52
	3:42	1200	164	14.60	0.77	19	0.41	0.33	0 80	123	41.3	1200	163	60.14
	4:43	1200	162	12.68	0.70	18	0.39	0.30	0.76	120	39.3	1200	162	53.99
	7:08	1200	164	12.03	0.71	17	0 38	0.28	0.75	122	39.8	1200	164	41.76
2	0	1200	178	13.11	0.82	16	0.40	0.34	0.84	139	38.0	1200	173	47.09
	0:08	1460	189	14.52	0.81	18	0.53	0.37	0.70	160	41.5	1540	195	56.29
	0:31	1600	194	14.23	0.84	17	0.43	0.34	0.80	152	37.0	1600	194	49.63
	2:52	1200	207	13 09	0 69	19	0.46	0.27	0.59	182	30.3	"	"	"
	2:58	1200	205	13.04	0.72	18	0.41	0.30	0.74	163	35.9	-	-	-
	3:02	1200	-	11.30	0 63	18	-	-	-	159	33.8	-	-	-
3	0	1200	155	14.56	0.81	18	0.34	0.34	0.98	121	35.0	1200	155	64 96
	0:08	1460	156	14.95	0.79	19	0.28	0.38	1.36	125	40.4	1540	154	73.01
	0:31	1600	149	13.92	0.73	19	0.30	0.32	1.06	110	39.4	1600	149	64 06
	2:40	1200	174	10.96	0.64	17	0.36	0.24	0.67	128	37.4	-	-	-
	2:46	1200	173	9.95	0.55	18	0.31	0.22	0.71	126	37.3	-	-	-
	2:50	1200	-	10.00	0.56	18	-	-	-	125	37.6	-	-	-
8	-	1200	384	17.67	0.93	19	0.42	0.37	0.88	361	30.3	1200	391	50.30
Post-exp.	-	0	-	14.02	0.70	20	0.42	0.36	0.86	111	37.5	0	-	59.51

[a]Elapsed time is the middle of the interval for gas exchange measurements at rest; mid-point of the exercise measurements is 6 minutes later; recovery is 10 minutes later.

and 1600 fsw (Subject CC, Phase II)

EXERCISE									RECOVERY					
V_T (l BTPS)	f (br/min)	Effort Index (cm H_2O)	Pedaling Rate (rpm)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	$P_{A_{O_2}}$ (mm Hg)	$P_{A_{CO_2}}$ (mm Hg)	Depth (fsw)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$P_{A_{O_2}}$ (mm Hg)	$P_{A_{CO_2}}$ (mm Hg)
1.82	30	14.5	92	1.88	1.88	1.00	110	42 2	0	16 15	0.73	22	113	35.5
1.64	24	-	71	1.09	1.34	1.24	146	-	632	12.93	0.65	20	137	39.4
2.11	28	-	94	1.62	1.90	1.17	138	44.8	784	16.89	0.84	20	136	38.3
2.17	26	29 1	88	1.48	1.71	1.15	-	-	800	17.46	0.87	20	144	38.5
1.74	23	17.3	61	1.26	1.28	1.01	125	-	800	15.33	0.85	18	130	-
1.94	25	-	69	1.48	1.50	1.01	129	-	1090	15.96	0.84	19	128	40.1
2.18	30	-	84	1.97	2.02	1.03	129	43.7	1190	21.28	1.01	21	138	39.8
2.23	27	26.2	83	1.75	1.73	0.99	124	41 3	1200	20.04	0.94	21	130	40 7
1.93	28	-	85	1 71	1.55	0.91	116	42.7	1200	16.49	0.84	20	126	38.9
1.90	22	9.6	64	1.45	1.28	0.88	115	43.9	1200	15.61	0.87	18	127	39.7
1.96	24	27.0	69	1.61	1.54	0.95	132	41.9	1200	16.75	0.84	20	137	36.3
2.16	26	-	66	1.58	1.57	1.00	148	42.9	1580	18.58	0.98	19	162	36.5
2.07	24	26.5	72	1.75	1.52	0.87	150	39.3	1600	17.80	0.99	18	156	37.2
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2.60	25	36.5	87	1.89	2.05	1.08	118	43.4	1200	18.99	0.95	20	126	34.8
2.70	27	-	94	2.08	2.17	1.04	118	42.8	1580	17.01	0.85	20	125	36.6
2.46	26	35.9	86	1.57	1.85	1.18	110	40.3	1600	18.53	0.93	20	119	36.3
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2.29	22	13.4	72	1 69	1.42	0.84	351	40.7	1200	20.30	1 07	19	365	29.6
2.12	28	19.1	92	1.72	1.84	1.06	115	37.3	0	14.86	0.74	20	113	35 7

TABLE V. Ventilation and Gas Exchange Responses to Exercise During Compression and Exposure to Pressures Equivalent to 800, 1200 and

		REST												
Exposure Day	Elapsed Time[a] (hr:min)	Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	$P_{A_{O_2}}$ (mm Hg)	$P_{A_{CO_2}}$ (mm Hg)	Depth (fsw)	$P_{I_{O_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)
1	0	0	-	12.38	0.65	19	0.40	0.31	0.78	-	-	0	-	31.37
	0:19	504	-	12.39	0.62	20	-	-	-	-	-	600	199	29.43
	0:38	704	193	12.78	0.71	18	0 35	0.34	0.96	-	-	752	191	44.26
	1:01	800	191	13.64	0.72	19	0.46	0 31	0.68	-	-	800	188	40.82
	2:18	800	175	11.89	0.66	18	0.34	0.27	0.77	-	-	800	171	36.90
	2:59	980	182	12.05	0.71	17	0.28	0.30	1.07	-	-	1050	178	35.94
	3:18	1140	175	20.1	1.06	19	0 50	0.60	1.19	-	-	-	-	-
	6:20	1200	175	13 80	0.81	17	0.37	0.30	0.83	-	-	1200	175	36.79
	7:43	1200	170	12.79	0.67	19	0 32	0 28	0.87	-	-	1200	170	33.30
2	0	1200	164	14.86	0.82	18	0.41	0.34	0.84	122	37.1	1200	164	37.45
	0:09	1380	179	13.10	0.66	20	0.46	0 32	0.71	134	40.7	1450	172	44.56
	0:28	1540	168	13.32	0.70	19	0.44	0.30	0.67	129	33.1	1570	167	41 41
	0:51	1600	160	14.74	0.78	19	0.43	0 31	0.73	119	35.1	1600	159	35.66
	2:59	1200	177	12.42	0 65	19	0.35	0.27	0.77	136	34.6	-	-	-
	3:05	1200	177	12.96	0.68	19	0.41	0.28	0.68	132	36.4	-	-	-
	3:09	1200	-	10.96	0.64	17	-	-	-	129	35 6	-	-	-
3	0	1200	154	14.33	0.80	18	0.37	0.33	0.90	-	-	1200	153	41.92
	0:08	1460	167	15.74	0.87	18	0.38	0 37	0.96	136	38.6	1540	164	39.47
	0:31	1600	166	13.72	0.76	18	0.37	0 30	0.79	124	36.3	1600	164	36.54
	2:57	1200	173	10.78	0 60	18	0.33	0.25	0.76	130	35.6	-	-	-
	3:03	1200	173	11.79	0.66	18	0.37	0.26	0.69	132	34.2	-	-	-
	3:07	1200	-	10.38	0.58	18	-	-	-	127	35.1	-	-	-
8		1200	168	15.77	0.93	17	0 38	0.34	0.89	137	36.2	1200.	171	39.83
Post-exp.		0	-	11.45	0.72	16	0 34	0.20	0.86	108	35.4	0	-	36.26

[a]Elapsed time is the middle of the interval for gas exchange measurements at rest; mid-point of the exercise measurements is 6 minutes later; recovery is 10 minutes later

1600 fsw (Subject GM, Phase II)

EXERCISE									RECOVERY					
V_T (l BTPS)	f (br/min)	Effort Index (cm H_2O)	Pedaling Rate (rpm)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	R	$P_{A_{O_2}}$ (mm Hg)	$P_{A_{CO_2}}$ (mm Hg)	Depth (fsw)	$\dot{V}_E$ (l/min BTPS)	V_T (l BTPS)	f (br/min)	$P_{A_{O_2}}$ (mm Hg)	$P_{A_{CO_2}}$ (mm Hg)
1.26	25	10.7	60	1.28	1.06	0.83	-	-	0	11.55	0.64	18	-	-
1.23	24	20.5	70	1.32	1.12	0.85	-	-	632	13.83	0.77	18	-	-
1.70	26	21.8	78	1.71	1.47	0.86	-	-	784	15.00	0.75	20	-	-
1.63	25	21.4	74	1.80	1.36	0.76	-	-	800	11.50	0.72	16	-	-
1.60	23	18.7	78	-	-	-	-	-	800	13.21	0.70	19	-	-
1.44	25	19.9	67	1.47	1.18	0.80	-	-	1090	21.67	1.20	18	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
1.67	22	-	62	1.54	1.22	0.79	-	-	1200	13.92	0.77	18	-	-
1.59	21	-	56	1.33	1.04	0.78	-	-	1200	14.75	0.82	18	-	-
1.56	24	21.7	68	1.42	1.23	0.86	111	41.5	1200	16.08	0.85	19	126	38.1
1.94	23	28.0	72	2.04	1.56	0.76	119	44.4	1490	19.64	0.94	21	134	35.0
1.72	24	26.4	70	1.77	1.33	0.75	113	44.9	1590	16.96	0.89	19	129	35.5
1.78	20	23.5	59	1.47	1.14	0.78	107	39.8	1600	15.39	0.86	18	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
1.91	22	24.2	69	1.58	1.41	0.89	104	41.9	1200	16.71	0.81	19	118	36.0
1.79	22	26.0	60	1.53	1.34	0.87	110	39.8	1580	17.36	0.87	20	134	35.0
1.74	21	21.9	57	1.37	1.21	0.88	109	41.8	1600	14.50	0.76	19	128	36.1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
1.81	22	-	66	1.52	1.19	0.79	129	41.1	1200	17.43	0.97	18	140	33.7
1.45	25	-	66	1.26	1.20	0.96	106	38.9	0	12.10	0.71	17	111	34.5

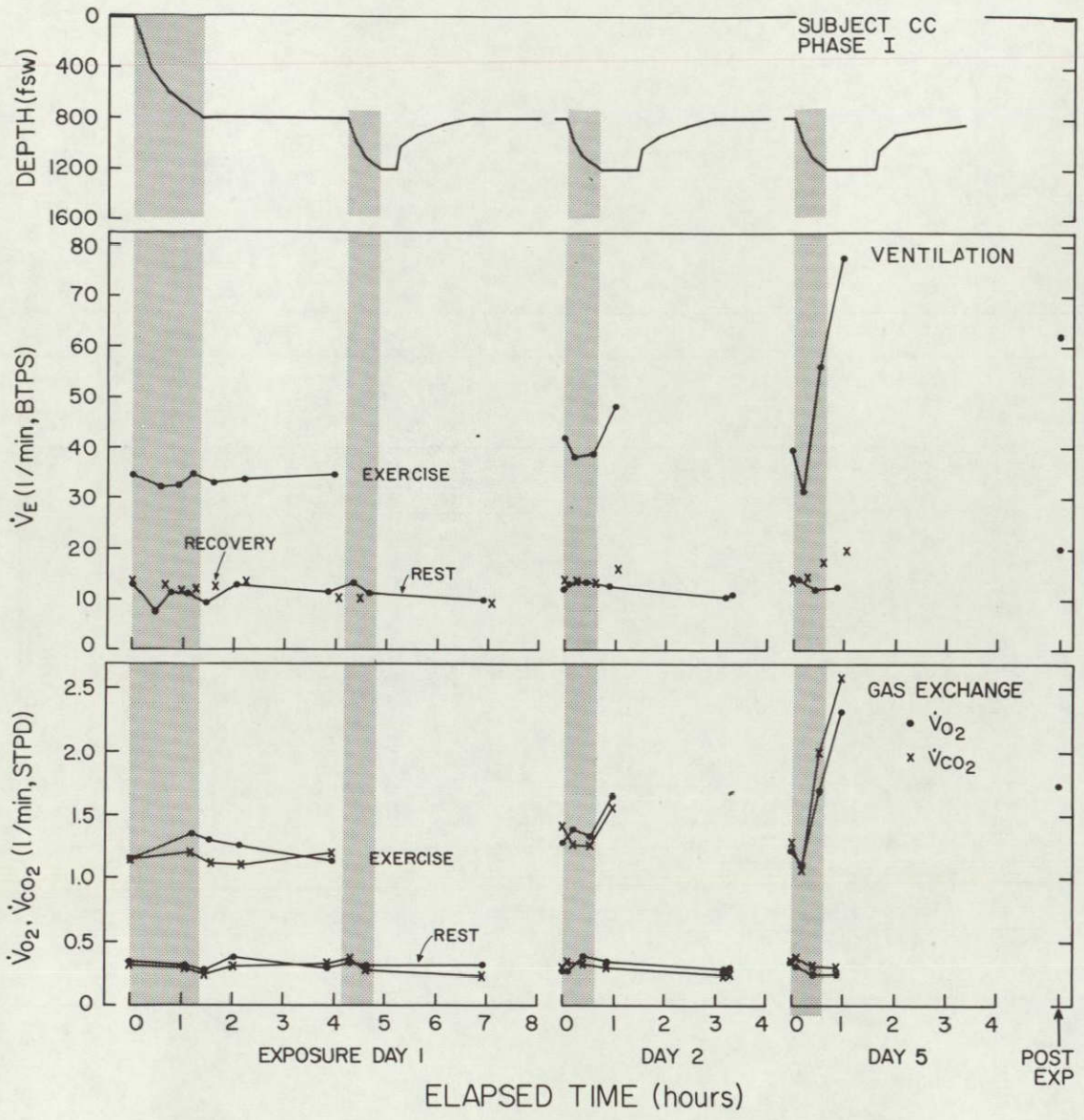

FIG. 2. Ventilation and gas exchange during compression and exposure to pressures equivalent to 800 and 1200 fsw. The simulated dive profile and measurements obtained during rest, exercise and (for ventilation) recovery are shown. Changes during exercise are caused by workload variations. (See text for explanation.)

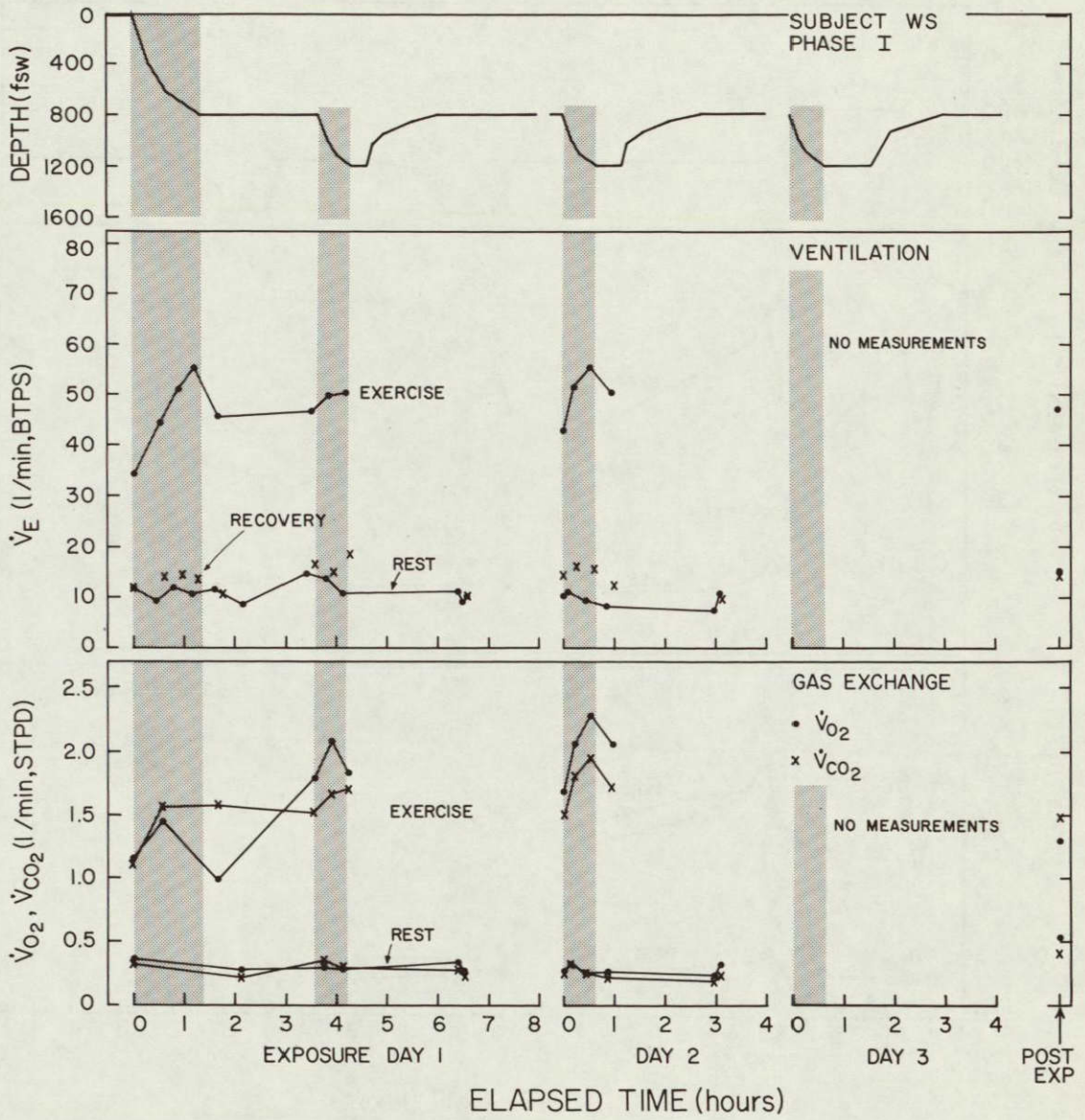

FIG. 3. Ventilation and gas exchange during compression and exposure to pressures equivalent to 800 and 1200 fsw. (See legend, Fig. 2 for additional details.)

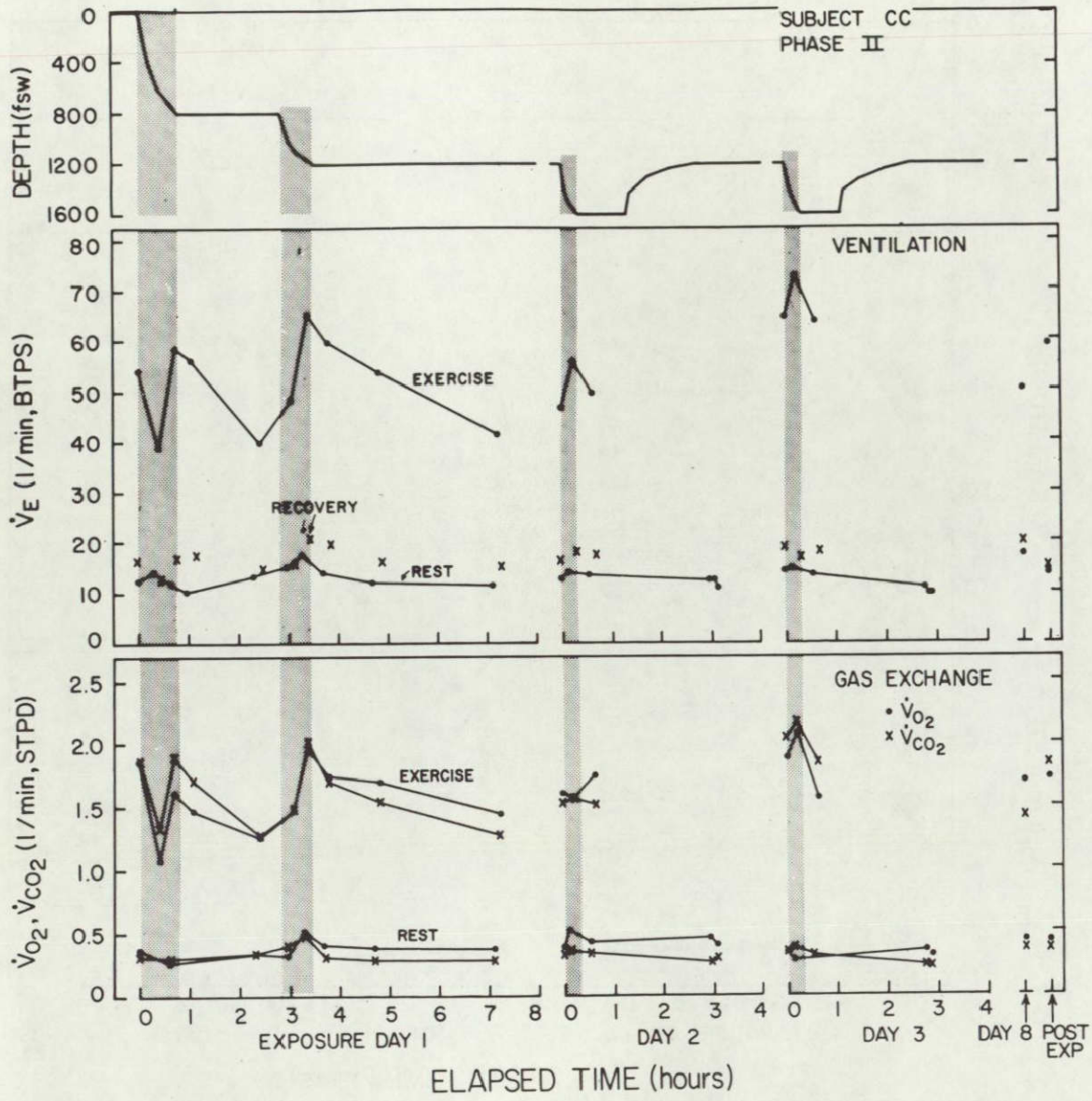

FIG. 4. Ventilation and gas exchange during compression and exposure to pressures equivalent to 800, 1200 and 1600 fsw. (See legend, Fig. 2 for additional details.)

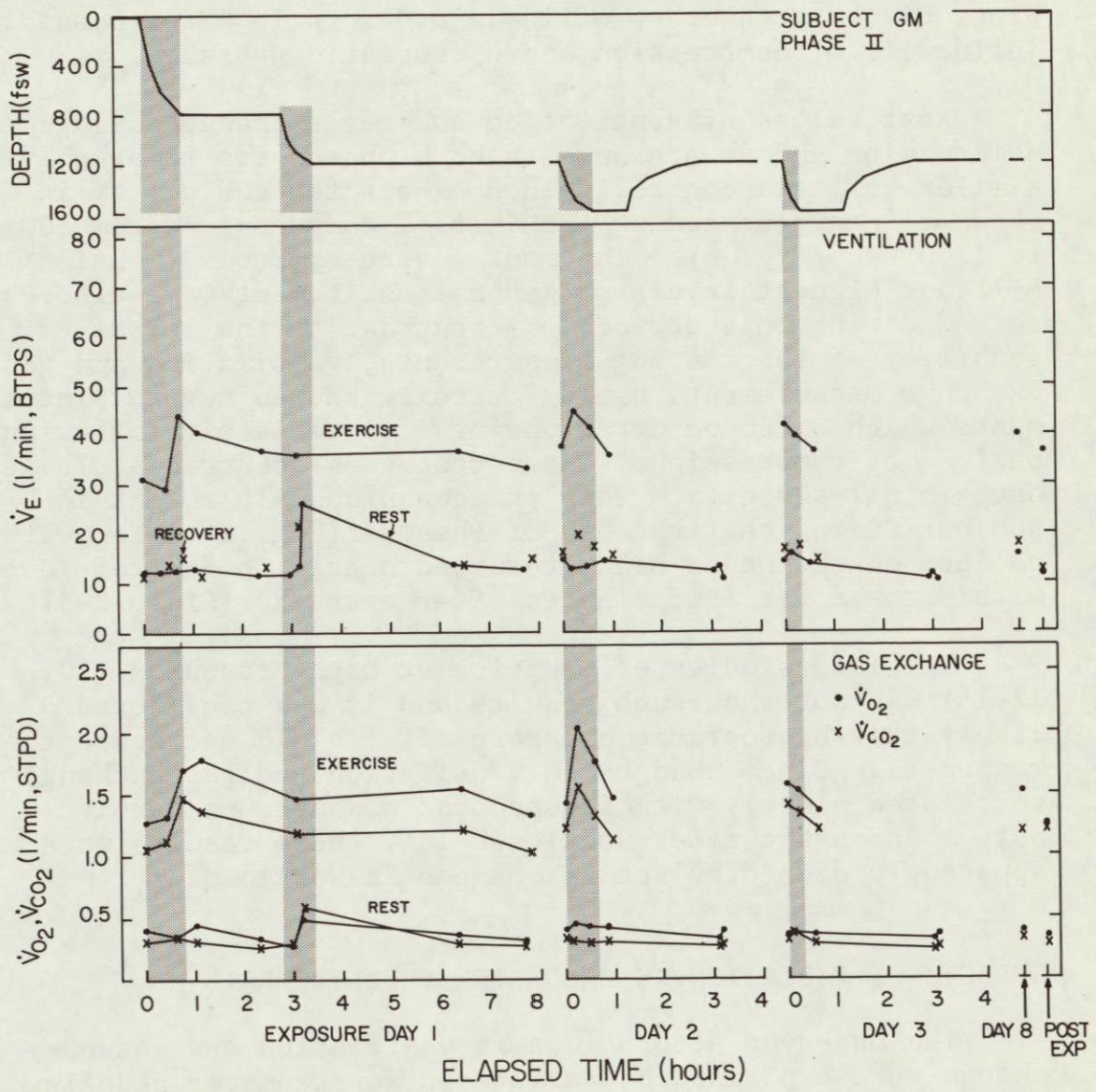

FIG. 5. Ventilation and gas exchange during compression and exposure to pressures equivalent to 800, 1200 and 1600 fsw. (See legend, Fig. 2 for additional details.)

In Figs. 2-5 individual measurements of $\dot{V}_E$, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ are plotted against elapsed time and compared with plots of the pressure profile in order to look for possible influences of compression and hydrostatic pressure.

Rest values of ventilation and gas exchange during compression and at pressure in both phases are remarkably similar to 1 ata control values except for subject GM in Phase II near the end of the initial compression from 800 to 1200 fsw (Fig. 5). The most severe symptoms (Section E-1) and highest levels of general excitement were also observed during this period. In contrast to the relative stability of the resting measurements, ventilation and gas exchange measurements during exercise showed several increments which often occurred during or immediately following periods of compression. These responses occurred at least once in all subjects. They are conspicuously absent in subject CC on the first day of Phase I (Fig. 2). Recovery values, on the other hand, returned nearly to control levels within the first four minutes after exercise (Figs. 2-5).

Previous studies of exercise at high pressures (10, 12-14) did not show such results and it was considered likely that hydrostatic pressure effects did not cause these responses. Since changes in ventilation and gas exchange correlated closely with independent measures of cardiac output and heart rate (Section E-13), these results were apparently caused by actual changes in workload.

EFFECTS OF VARIATION IN ERGOMETER PEDALING RATE

The observed alterations in ventilation and gas exchange were explained by variations in ergometer pedaling rate in the different modules. Videotape recordings taken during the compression-pressure exposures confirmed that pedaling rate varied (Tables IV and V), and further analysis showed that $\dot{V}_{O_2}$ correlated with pedaling rate. Use of either an audible or a visual cue to keep pedaling rate constant was impractical in these experiments because the subjects had to participate actively in procedures such as eye tracking and perceptual-cognitive-psychomotor testing while pedaling. A variable pedaling rate was originally not considered to pose a major problem since the ergometer used is electronically compensated to maintain a nearly constant workload over a wide range of pedaling rates (4).

measurements to a pedaling rate of 120 rpm showed that the actual workload would then increase by approximately 34%. These variations are consistent with data provided by the manufacturer at the same workload setting (4)[2]. The measurements indicate that over the pedaling range of 40 to 120 rpm (Fig. 6) about 0.36 1/min of the total observed 1.73 1/min increment in $\dot{V}_{O_2}$ may be attributed to an increase in ergometer workload. The remainder of the $\dot{V}_{O_2}$ increment (1.37 1/min) represents the metabolic cost of energy lost as heat while more rapidly moving a fixed mass of legs, shoes and clothing.

Since the $\dot{V}_{O_2}$-pedaling rate data for the exercise subjects of Phase II are similar to those for the group of six subjects (Fig. 6), the variations in $\dot{V}_E$, $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ found during exercise at high pressures (Figs. 2-5) are attributed to changes in actual workload. Therefore the levels of exercise performed in the different modules of both Phases I and II were determined from the measured $\dot{V}_{O_2}$, rather than from the ergometer setting. Influences of variable workloads must also be considered in the interpretation of other data obtained from the subjects during exercise.

ELECTRICAL ACTIVITY OF THE DIAPHRAGM

Diaphragmatic EMG measurements of sufficiently high quality for analysis were obtained from subjects CC in Phase I and CC and GM in Phase II, both at rest and during exercise. Strip chart tracings of the EMG signals taken when the subjects reported their most severe symptoms were compared with tracings at sea level, at low pressures, at stable high pressures and during excursions. None of the tracings showed impaired synchronization of normal

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[2] Actual workload of the electronically controlled bicycle ergometer is independent of pedaling rate within ±2% over a relatively wide range of load settings and pedaling rates. With load settings below 100 watts, however, workload becomes proportional to pedaling rate and this influence becomes progressively stronger as the load setting is reduced.

inspiratory bursts of activity or any spurious electrical activity which might have been caused by pressure effects on nervous tissue. This indicates that neither the neural output of the respiratory centers nor the transmission of impulses to the diaphragmatic muscles was altered by either rapid compression or by pressure itself.

DISCUSSION

The planned direct comparison of physiological responses to exercise during compression and at pressure against sea level controls required maintenance of a constant workload and oxygen consumption in all modules (1 ata controls, during compression, at high pressure). Since pedaling rate and $\dot{V}_{O_2}$ varied more than anticipated while the subjects were preoccupied with mental performance testing, Figs. 2-5 show a combined effect of pressure and of variable exercise levels. However, responses to these spontaneously variable workloads during the two pressure exposures can still be compared with responses to equivalent workloads at sea level.

COMPARISON OF EXERCISE RESPONSES AT SEA LEVEL AND AT INCREASED AMBIENT PRESSURES

Ventilatory responses are altered smoothly and progressively by increasing exercise (5). Therefore, data describing the relationships of $\dot{V}_E$, V_T and f to workload obtained during the maximum exercise testing at one ata can be used as controls for comparison with ventilatory responses to work during the compression and pressure exposures. Such comparisons of ventilatory data obtained at different ambient pressures for each of the three exercise subjects are shown in Figs. 7A, 8A and 9A which use $\dot{V}_{O_2}$ as an index of work intensity.

In general the volume rate ($\dot{V}_E$) and pattern (V_T, f) of ventilation for light-to-moderate exercise during compression to simulated depths of 800, 1200 and 1600 fsw are not grossly different from responses at sea level. When $\dot{V}_{CO_2}$ is used as the work index (Figs. 7B, 8B and 9B), there appears to be better agreement with less scatter of ventilatory responses at

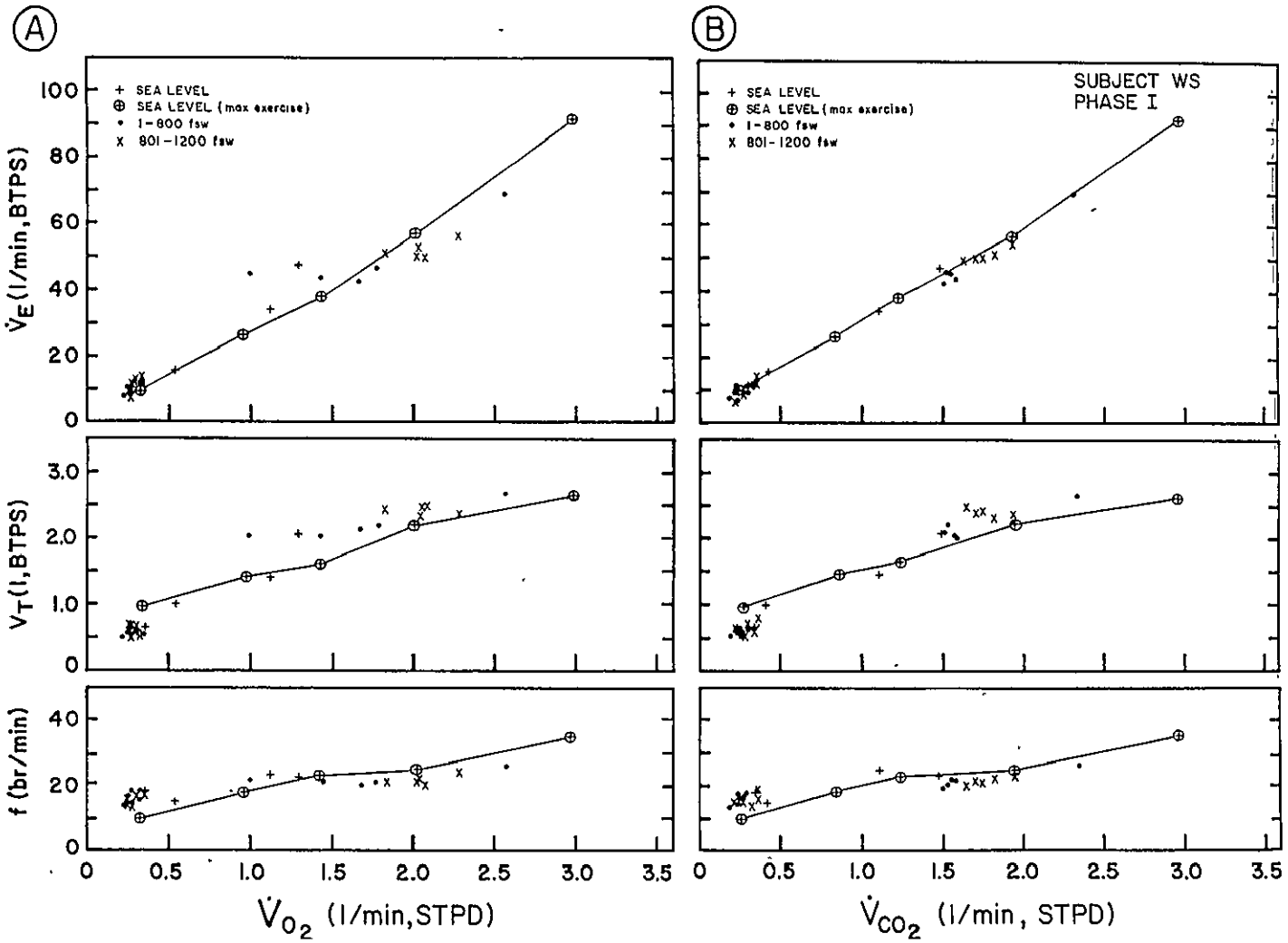

FIG. 7. Relationships of ventilation, tidal volume and respiratory frequency to A, oxygen uptake, and B, carbon dioxide output at 1 ata and pressures up to 1200 fsw. Measurements taken with subject at rest and during exercise.

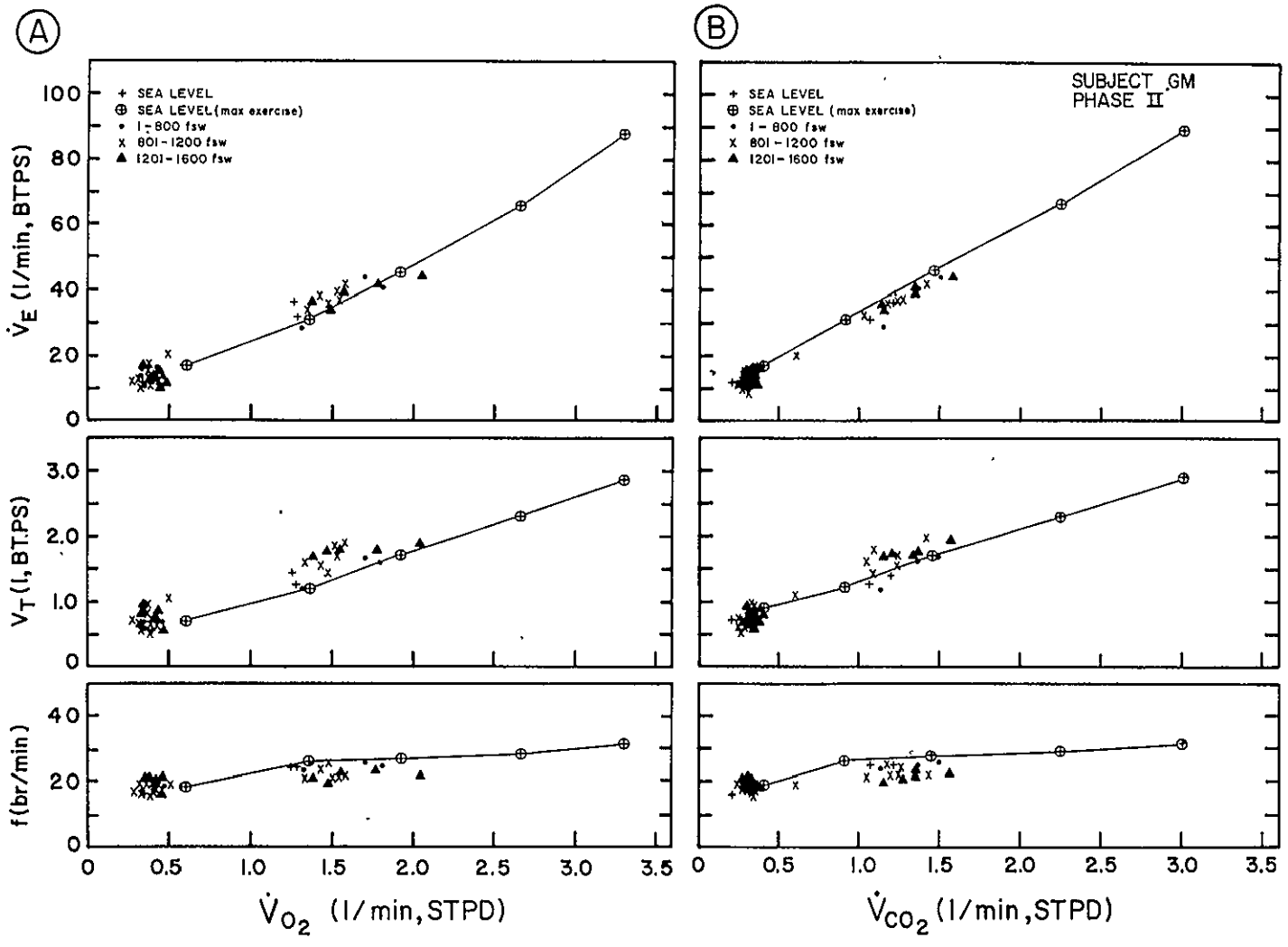

FIG. 8. Relationships of ventilation, tidal volume and respiratory frequency to A, oxygen uptake, and B, carbon dioxide output at 1 ata and pressures up to 1600 fsw. Measurements taken with subject at rest and during exercise.

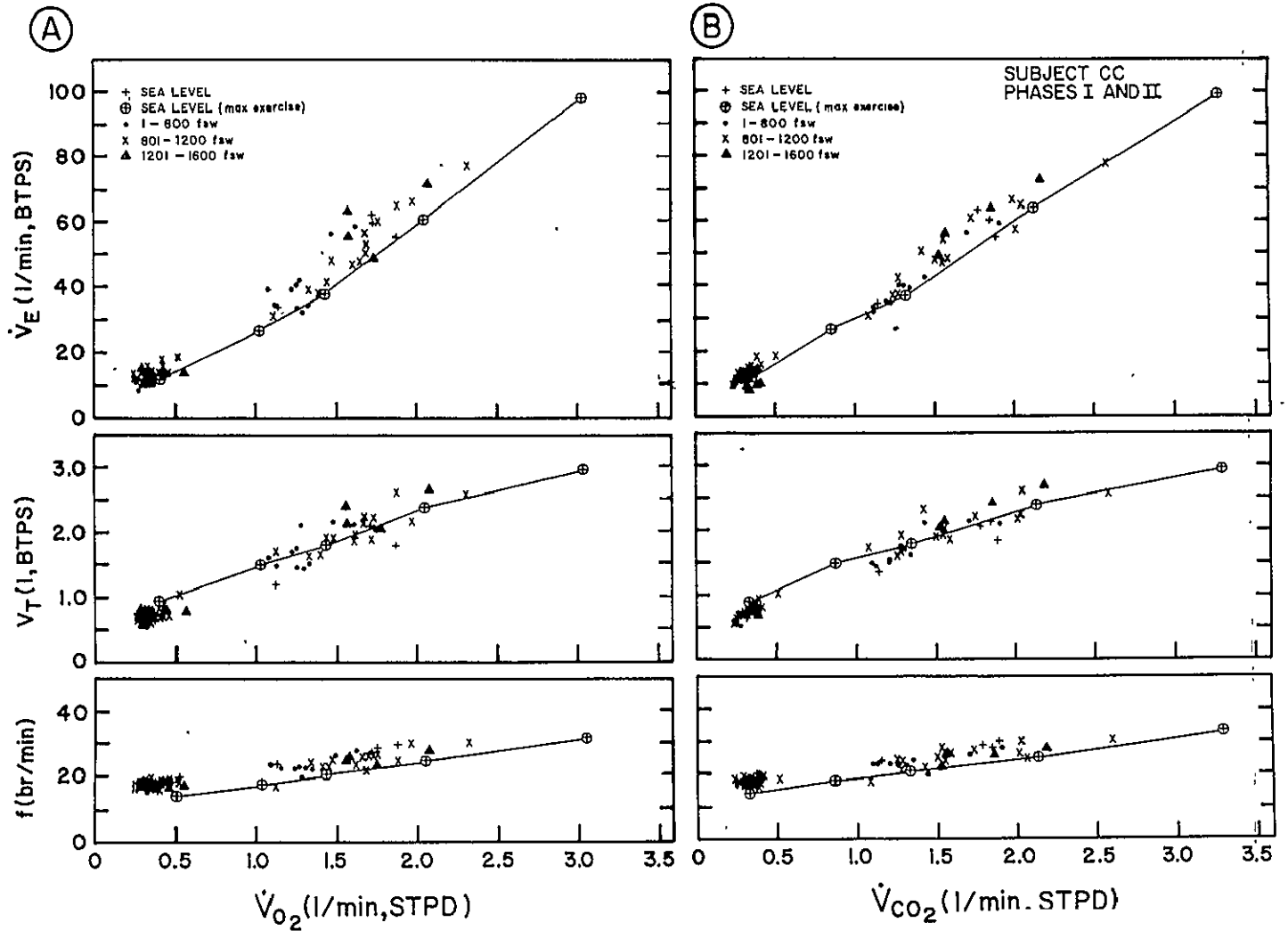

FIG. 9. Relationships of ventilation, tidal volume and respiratory frequency to A, oxygen uptake, and B, carbon dioxide output at 1 ata and pressures up to 1600 fsw. Measurements taken with subject at rest and during exercise.

increased pressures. This suggests that while $\dot{V}_{O_2}$ expresses work level, $\dot{V}_{CO_2}$ rather than $\dot{V}_{O_2}$ is the dominant link between ventilation and metabolic activity.

The data summarized in Figs. 7-9 provide no evidence of significant ventilatory impairment during light-to-moderate exercise either during compression or at increased ambient pressures. The adequate ventilatory capability of the subjects at the selected work level is further confirmed by end-tidal P_{CO_2} measurements which show no CO_2 retention during exercise (Tables IV and V).

COMPARISON WITH OTHER STUDIES

Although some subjects are able to maintain sea level values of ventilation at pressures equivalent to 600 fsw (2), most previous studies have shown a reduced ventilatory response to exercise when the breathing gas density is increased, with its concurrent increment in work of breathing (9,12,13). Typically the requirement for a greater effort to perform a given level of $\dot{V}_E$ is associated with a reduction in f and an increase of V_T. When $\dot{V}_{CO_2}$ was used as the workload index, a similar pattern of slight reductions in $\dot{V}_E$ and f and an increase in V_T was seen in subjects WS and GM during exercise at increased ambient pressures (Figs. 7B and 8B).

Subject CC was the only one who reported breathing difficulties during exercise at depth, which he variously described as: "overbreathing the system," "not getting enough oxygen," "breathing too fast" and "increased resistance" (Section E-1). On the initial compressions of day 1 in Phase II, while pedaling at rates greater than planned, he reported that: "I was going too fast for my lungs to take care of me. I was going so fast that I wasn't getting enough oxygen." On day 2 he indicated that his breathing

did not bother him while "just doing what exercise I could handle." In conjunction with the wide variation in ergometer pedaling rate and the parallel changes in ventilation (Table IV, Fig. 6), these comments indicate that the periods of excessive breathing resistance perceived by CC were associated with his self-generated increments in workload. This possibility is supported by the fact that CC did not report increased resistance during the initial compression to 800 and 1200 fsw in Phase I when his ventilatory and gas exchange responses to exercise were relatively constant at the intended levels, about half the maximum levels observed on the first day of Phase II (Figs. 2 and 4). Figure 1 shows that increased ventilatory rates would be accompanied by increments in external breathing resistance. Because distortion of the respiratory system, chest wall and diaphragm may occur in response to such increases in external work of breathing, the resulting change in total work of breathing may be three to eight times the change in external work (7).

Measurements of esophageal pressure obtained during exercise in Phase II may be directly related to the symptoms reported by subject CC. The total esophageal pressure swing (average total difference between maximum expiratory and maximum inspiratory pressures over several breaths during exercise) is designated as the "effort index" in Tables IV and V for CC and GM, respectively. CC's effort index varied widely and approximately in parallel with his ventilatory fluctuations (Table IV) while the values for effort index and ventilation in GM, who denied sensations of increased breathing resistance, were much more uniform (Table V).

Plots of $\dot{V}_E$ against the effort index (esophageal pressure swing) also indicate that these relationships differ in the two subjects (Fig. 10). The esophageal pressure swings for a given level of $\dot{V}_E$ in subject GM (Fig. 10B) were consistently increased at higher ambient pressures. This shift can be attributed to both the increased inspired gas density at higher pressures and the concurrent change in breathing pattern with augmentation of $\dot{V}_E$ and reduction in f (Fig. 8B). When $\dot{V}_E$ is plotted against effort index for subject CC (Fig. 10A) there is a wider scatter of data and no consistent shift to the right at higher pressures. Throughout the range of ambient pressures to which both subjects were

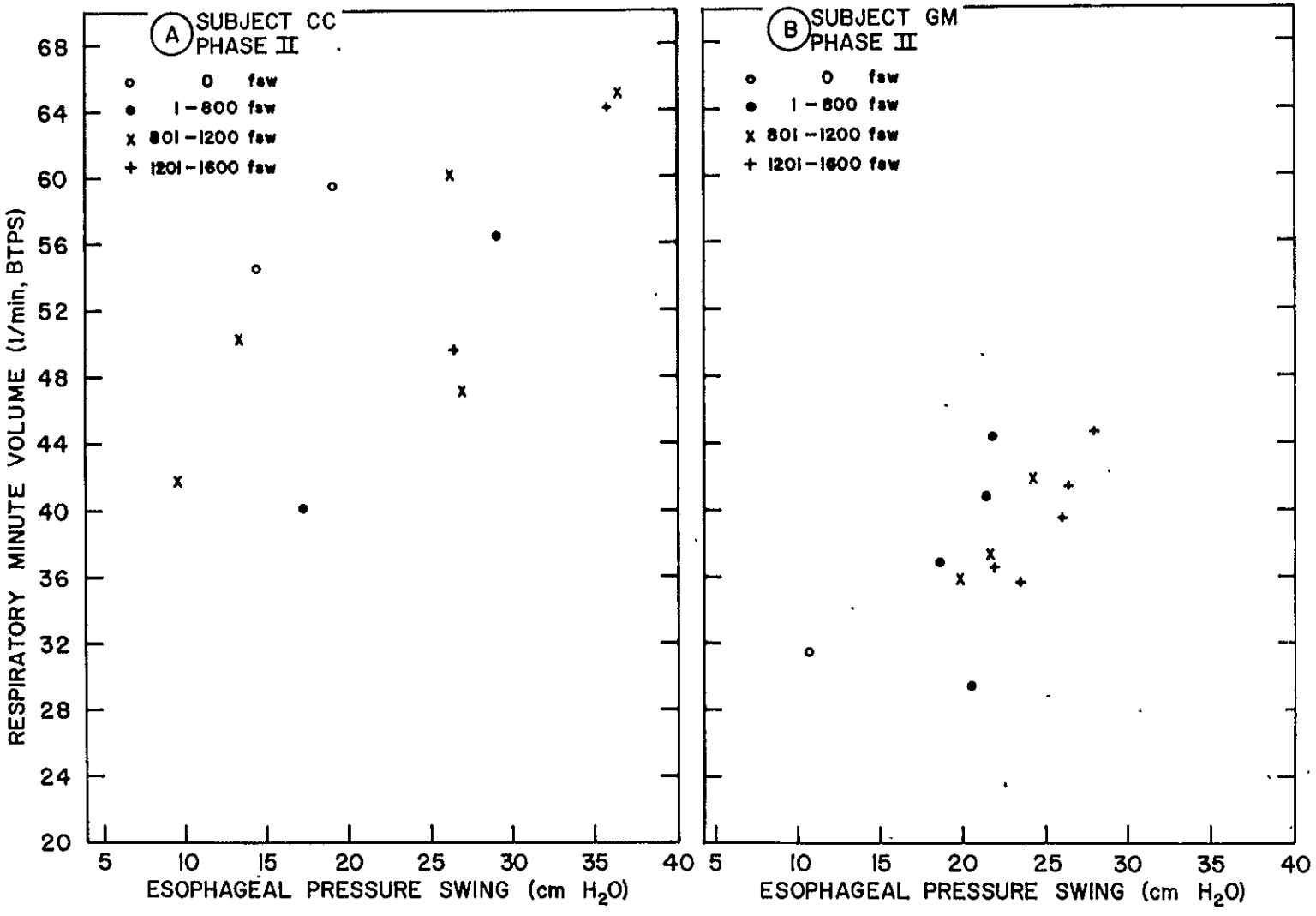

FIG. 10. Relationship of ventilation to esophageal pressure swing ("effort index") during exercise. (Phase II, subjects GM and CC. See text for additional details.)

exposed, the esophageal pressure swings and V_E levels in subject CC were higher than those measured in subject GM. These measurements are consistent with greater work of breathing in subject CC and may explain why he experienced breathing difficulties while subject GM apparently did not.

POSSIBLE INFLUENCES OF HYDROSTATIC PRESSURE ON PHYSIOLOGICAL RESPONSES TO EXERCISE

Studies in animals have shown that extreme hydrostatic pressures can cause hyperactivity of some brain areas at the same time that cortical activity is depressed (8). These studies are consistent with the possibility of increased central respiratory reactivity at increased ambient pressures.

However, the data summarized in Tables II-V and Figs. 7-9 demonstrate no significant hydrostatic effects on either ventilatory or metabolic responses to exercise. The subjects' ventilatory responses are generally appropriate to the corresponding oxygen consumption and workloads, and recovery from light-to-moderate exercise does not appear to be prolonged. Slight decrements in ventilation and alterations in the breathing pattern in subjects WS and GM during exercise at depth (Figs. 7B and 8B) are attributed to the concurrent increases in respired gas density, airway resistance and work of breathing (9,13).

HYDROSTATIC PRESSURE EFFECTS AT REST AND DURING EXERCISE

Exercise was incorporated into the design of these studies in order to evaluate its possible influences on hydrostatic pressure effects. A relevant observation is that the symptoms and signs produced by initial compressions to 800 and 1200 fsw were generally more severe in the exercise than in the rest subject (see Section E-1), particularly for subjects CC in Phase I and GM in Phase II.

Both subjects experienced severe symptoms which appeared to be intensified by slight elevations in ambient temperature (Section E-15). It is also significant that the chamber area occupied by the exercise subject was usually several degrees C warmer than that of his resting companion, and that the intermittent periods of exercise caused transient elevations of several tenths of a degree C in rectal temperature (Section E-15). Relative severity of symptoms may also be a function of individual differences in susceptibility; however in this study there was no opportunity to reverse identities of the exercise and rest subjects.

Comparisons of hydrostatic effects at rest with those during exercise are more valid when made in the same subject. Such comparisons were possible for both the electroencephalographic measurements of brain electrical activity (Section E-2) and the evaluation of perceptual and psychomotor performance (Section E-10).

Movement artifacts hindered monitoring of brain wave activity during many exercise periods. Those records suitable for analysis, however, showed no significant differences between the active and resting states.

Satisfactory perceptual and psychomotor performance measurements were obtained during most exercise periods. Within each module, performance was tested over a three-minute interval initially at rest and six minutes later during light exercise. Results of the two test periods were similar except for one generally consistent difference, seen most frequently in subject CC upon resumption of testing after a break of several hours. When performance during the first interval at rest was below the established control level, the scores for the tests given during exercise often returned to or exceeded control values. Whether this "alerting" effect was actually related to the exercise could not be determined. A similar effect of exercise has been reported in studies on interactions of nitrous oxide narcosis, exercise and psychomotor performance (3).

SUMMARY AND CONCLUSIONS

Ventilatory and metabolic responses to light exercise during rapid compression and exposure to hydrostatic pressures equivalent to depths of 400,800,1200 and 1600 fsw

resembled responses to comparable workloads at normal ambient pressure.

Two of the three exercise subjects had slight reductions in $\dot{V}_E$ and f with an increase in V_T compared with sea level responses for the same level of exercise. These findings are in agreement with previously observed effects of increased inspired gas density at high ambient pressures. The same two subjects had no apparent pulmonary symptoms during exercise at increased pressures. The third subject had repeated sensations of dyspnea and increased resistance during exercise at depth. Since the data show no significant hydrostatic effects on either ventilation or gas exchange during exercise at increased pressures, these symptoms may have been caused by intermittent, abrupt increments in work of breathing and ventilation, associated with self-generated increases in workload.

Even though the exercise subjects had more severe general symptoms than the rest subjects during and after the initial rapid compressions to 800 and 1200 fsw, no significant differences in the severity of hydrostatic effects at rest and during exercise in the same subject were found. In particular, consistent rest-exercise differences were not detected in brain electrical activity or in perceptual and psychomotor performance.

Acknowledgments

The technical assistance of Mr. W. Herr in instrumentation setup, measurement during the studies, computer programming and data analysis was outstanding. Mr. D. Fisher fabricated the esophageal balloon and electrode catheters .

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E-15. THERMAL AND METABOLIC HOMEOSTASIS

C.D. Puglia[1], W. Herr[1], J.M. Clark[1] and R. Gelfand[1]

Thermal homeostasis in man is severely challenged by exposure to high density helium-oxygen environments. Heat loss from both the body surface (convective) and the lungs (respiratory) is increased by the high thermal conductivity of dense helium at elevated pressures (6,7,9,20,22). Raising ambient temperature reduces this heat loss and provides thermal comfort; however, the thermal comfort temperature range is both elevated and narrowed with increasing depth (10,12,14,16,19). Elevation of ambient temperature results in an increased skin temperature, reducing the temperature gradient between the body surface and the chamber gas (14, 16,18). This tightening of the thermal coupling between man and the gaseous environment causes thermal homeostasis to be stressed by even small deviations of ambient temperature from the narrowed comfort zone. It has been shown that deep body temperatures under these conditions of high helium pressures have increased (14), decreased (16), or remained essentially unchanged (15,18). The inconsistencies can be partially explained by diurnal variations and different levels of activity.

Investigation of ambient temperature extremes was not included in this study. The purpose here was to minimize thermal stresses in order to isolate and investigate the direct effects of rapid compression and extreme hydrostatic pressures on physiological responses and performance. Since it has been shown that ambient temperature changes can modify hydrostatic effects (1-3), it was considered vital to closely control and monitor relevant environmental parameters even during compression, and to define the thermal-metabolic state of each subject accurately throughout the saturation-excursion exposures.

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6-7

6-C

MATERIALS AND METHODS

SUBJECTS

Physical characteristics of all subjects, summarized in Table I, were obtained 21 days prior to the beginning of Phase I. All subjects were professional divers; three (LJ, FS, WS) had resided for at least one year in the warm climate of the Gulf states, two (CC, MP) had resided in Scotland, and one (GM) in New York prior to this study. All subjects came to Philadelphia for the four months of continuous preparation which preceded the pressure exposures of Predictive Studies IV.

TABLE I. Subject Characteristics

Subject	Age (yrs)	Height (cm)	Weight[a] (kg)	Surface Area[b] (m^2)	Total Skinfold Thickness (mm)		Fat[e] (% of Body Wt.)
					5 sites[c]	3 sites[d]	
CC	25	180	82.6	1.964	41.9	22.9	14.8
LJ	29	178	72.4	1.844	31.8	18.2	11.8
GM	23	183	81.0	2.036	30.9	19.3	12.6
MP	29	173	75.8	1.964	32.4	16.6	10.6
FS	25	169.5	72.5	1.816	31.6	10.0	4.2
WS	24	191	85.0	2.066	19.9	11.1	5.5

[a] Median of ten-day period ending three days before start of Phase I.

[b] Calculated using method of DuBois and DuBois (4).

[c] Biceps, triceps, subscapular, paraumbilical, neck.

[d] Biceps, triceps, subscapular.

[e] Calculated from sum of skinfold thickness at three sites: biceps, triceps and subscapular using a regression formula for density and Siri's equation for fat as % of body weight (5).

APPARATUS

Temperature, Humidity, Gas Velocity and Body Weight Sensors

Temperatures were measured with Yellow Springs Instruments (YSI) thermistors and recorded by a 25-channel data acquisition system onto a paper tape printer and a digital magnetic tape recorder for off-line analysis on a digital computer. YSI type 401 thermistors were used for rectal and wet-dry bulb, type 409 for chamber wall, types 408 and 409 for skin, type 405 for ambient gas, type 520 for expired air, and type 511 for esophageal temperature. Thermistors were calibrated in a water bath against a thermometer calibrated by National Bureau of Standards. The maximum deviation of any of the thermistors from the reference was 0.25°C.

Other Sensors

Chamber ambient temperature was also monitored with copper-constantan thermocouples of the chamber system. Relative humidity of chamber gas was obtained by means of a Honeywell model Q457A gold grid, temperature-compensated sensor. These measurements were recorded at 30-minute intervals throughout the entire day. The horizontal component of gas velocity was sensed with a Scientific Associates no. 442-2 cup anemometer (minimum detectable velocity 0.1 m/sec). Body weight was determined on a Fairbanks platform balance (model no. 41-1000-CM) accurate to ± 100 g. A Lange caliper (Cambridge Scientific Industries, Cambridge, Md.) was used to measure skinfold thickness.

METHODS

Thermistor Assemblies

Two interchangeable assemblies of thermistors were monitored by the 25-channel digital magnetic tape system. On experiment days "subject assemblies" were connected and temperature was recorded every minute, both during and

between modules. At all other times an "environmental assembly" was connected and ambient temperatures were recorded every 10 minutes.

The subject assemblies, incorporated into harnesses (Section D), consisted of skin temperature thermistors for 10 sites on each subject (forehead, chest, stomach, back, upper arm, forearm, hand, outer thigh, calf and foot), rectal and esophageal thermistors, and one thermistor for environmental temperature. Skin temperature thermistors were secured to shaved skin with 3M Transpore tape. The rectal thermistor was inserted 10 cm into the rectum and its cable taped to the buttock. The esophageal thermistor was placed in a vinyl catheter (0.12 inch external diameter) which also contained electrodes for measurement of the diaphragmatic electromyogram (Section E-14). Positioning of this catheter in the lower third of the esophagus was determined by proper placement of the diaphragmatic electromyographic electrodes.

The environmental assembly consisted of an array of six ambient temperature sensors in chamber 2 and five in chamber 3, one chamber wall temperature sensor each in chambers 2 and 3, a wet-dry bulb assembly in chamber 2, two rectal thermistors, and one (hand-held) skin temperature sensor for early morning measurements in chamber 2.

Mean Skin Temperature

Mean skin temperature ($\bar{T}_{sk}$) was calculated by one of three methods as shown below(11):

Method 1 (Hardy/Dubois)

$$\bar{T}_{sk} = (0.07\ T_{forehead} + 0.14\ T_{forearm} + 0.05\ T_{hand} + 0.07\ T_{foot} + 0.13\ T_{calf} + 0.19\ T_{thigh} + 0.35\ T_{stomach})$$

Method 2 (Modified Teichner)

$$\bar{T}_{sk} = (0.149\ T_{forehead} + 0.372\ T_{thigh} + 0.186\ T_{back} + 0.186\ T_{chest} + 0.107\ T_{upper\ arm})$$

Method 3

$$\bar{T}_{sk} = (0.50\ T_{chest} + 0.36\ T_{calf} + 0.14\ T_{forearm})$$

Either method 1 or 2 was used for calculation of $\bar{T}_{sk}$ during the experiment modules. Method 1 was the preferred method, used when all the required temperature probes remained in place. When any of those probes was displaced, method 2 was used. Method 3 was used to estimate $\bar{T}_{sk}$ for all the early morning measurements.

Caloric Intake

This was calculated daily throughout the pressure exposure by weighing the food consumed (amount served minus amount returned) and using caloric values from standard tables.

Early Morning Measurements

Gas exchange measurements were made directly after awakening with the subjects fasting and sitting quietly for 15 minutes on the bicycle ergometer. The same apparatus used for gas exchange determinations in the experiment modules (Section E-14) was used to measure oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide elimination ($\dot{V}_{CO_2}$) and respiratory exchange ratio (R) for the final two minutes of this period. Heart rate, rectal temperature and skin temperatures (type 408 probe, moved from point to point) were measured at the same time, as was cardiac output in Phase II only (Section E-13). Body weight was also measured during this period--after voiding urine but before defecation.

RESULTS

Environmental data presented here are from chamber 2 in which the physiological measurements were made. Ambient temperature was obtained from the thermistor probe located near the exercise subject. Tables II and III correlate chamber measurements of depth, temperature, and relative

TABLE II. Chamber Temperature and Humidity During Experiment Modules in Phase II (Subjects CC and MP)

Exposure Day	Depth (fsw)	Clock Time	Compression Rate (ft/min)	Chamber Temperature (°C)			Relative Humidity (%)
				Start	End	Range	
1	0-400	1204-1217	32	23.4	25.8	23.4-32.9	57
	400-600	1217-1229	16	25.8	31.0	25.5-31.0	-
	600-800	1229-1254	8	31.0	31.2	31.0-31.7	70
	800	1254-1454		31.2	31.5	30.5-32.1	70
	800-1000	1454-1504	20	31.5	30.2	30.2-31.9	-
	1000-1100	1504-1514	10	30.2	31.0	30.2-31.2	74
	1100-1200	1514-1534	5	31.0	31.0	29.3-31.1	75
	1200	1534-1945		31.0	31.9	30.8-33.0	74
2	1200-1400	1332-1337	40	30.7	28.8	28.8-31.0	75
	1400-1500	1337-1342	20	28.8	30.5	28.0-30.5	-
	1500-1600	1342-1352	10	30.5	30.6	30.5-30.6	-
	1600	1352-1447		30.6	30.9	30.4-32.4	76
	1600-1425	1447-1453		30.9	34.2	30.9-34.4	-
	1425-1335	1453-1511		34.2	31.4	31.1-34.2	83
	1335-1255	1511-1543		31.4	31.8	31.4-31.9	76
	1255-1200	1543-1616		31.8	31.8	31.6-32.0	73
	1200	1616-1659		31.8	31.1	31.0-32.0	71
3	1200-1400	1246-1251	40	31.7	38.1	31.7-38.1	-
	1400-1500	1251-1256	20	38.1	34.1	34.1-38.1	-
	1500-1600	1256-1306	10	34.1	30.7	29.3-34.1	63
	1600	1306-1350		30.7	32.0	30.4-32.1	70
	1600-1425	1350-1356		32.0	34.8	32.0-34.8	-
	1425-1335	1356-1414		34.8	31.1	29.7-34.8	82
	1335-1255	1414-1446		31.1	31.3	31.0-31.3	72
	1255-1200	1446-1519		31.3	31.3	31.0-31.4	69
	1200	1519-1600		31.3	31.1	30.9-31.6	67

TABLE III. Chamber Temperature and Humidity During Experiment Modules in Phase II (Subjects FS and GM)

Exposure Day	Depth (fsw)	Clock Time	Compression Rate (ft/min)	Chamber Temperature (°C)			Relative Humidity (%)
				Start	End	Range	
1	0-400	1258-1311	32	22.4	25.1	22.4-27.3	69
	400-600	1311-1323	16	25.1	25.9	25.1-35.3	69
	600-800	1323-1350	8	25.9	31.0	24.3-31.7	79
	800	1350-1548		31.0	29.6	26.8-31.9	80
	800-1000	1548-1558	20	29.6	29.6	29.4-30.2	77
	1000-1100	1558-1608	10	29.6	29.7	29.2-29.7	72
	1100-1200	1608-1628	5	29.7	31.8	29.3-32.3	-
	1200	1628-2107		31.8	31.0	29.7-35.2	81
2	1200-1400	1433-1443	20	31.2	30.7	30.6-32.9	70
	1400-1500	1443-1453	10	30.7	31.6	30.7-31.6	-
	1500-1600	1453-1513	5	31.6	31.5	31.3-31.8	71
	1600	1513-1555		31.5	36.1	30.8-36.7	72
	1600-1425	1555-1601		36.1	30.3	30.3-36.1	-
	1425-1335	1601-1619		30.3	30.9	30.1-38.1	78
	1335-1255	1619-1651		30.9	31.2	30.9-31.6	72
	1255-1200	1651-1724		31.2	31.4	31.0-35.8	70
	1200	1724-1803		31.4	31.4	30.6-31.9	71
3	1200-1400	1358-1403	40	31.0	28.9	28.9-33.3	71
	1400-1500	1403-1408	20	28.9	34.1	27.3-34.2	71
	1500-1600	1408-1418	10	34.1	32.5	31.1-34.3	-
	1600	1418-1513		32.5	30.2	28.8-34.7	80
	1600-1425	1513-1519		30.2	36.8	30.0-36.8	-
	1425-1335	1519-1537		36.8	31.9	29.8-36.8	76
	1335-1255	1537-1609		31.9	30.6	29.8-32.3	73
	1255-1200	1609-1642		30.6	30.6	27.9-37.1	71
	1200	1642-1723		30.6	30.9	30.1-31.3	71

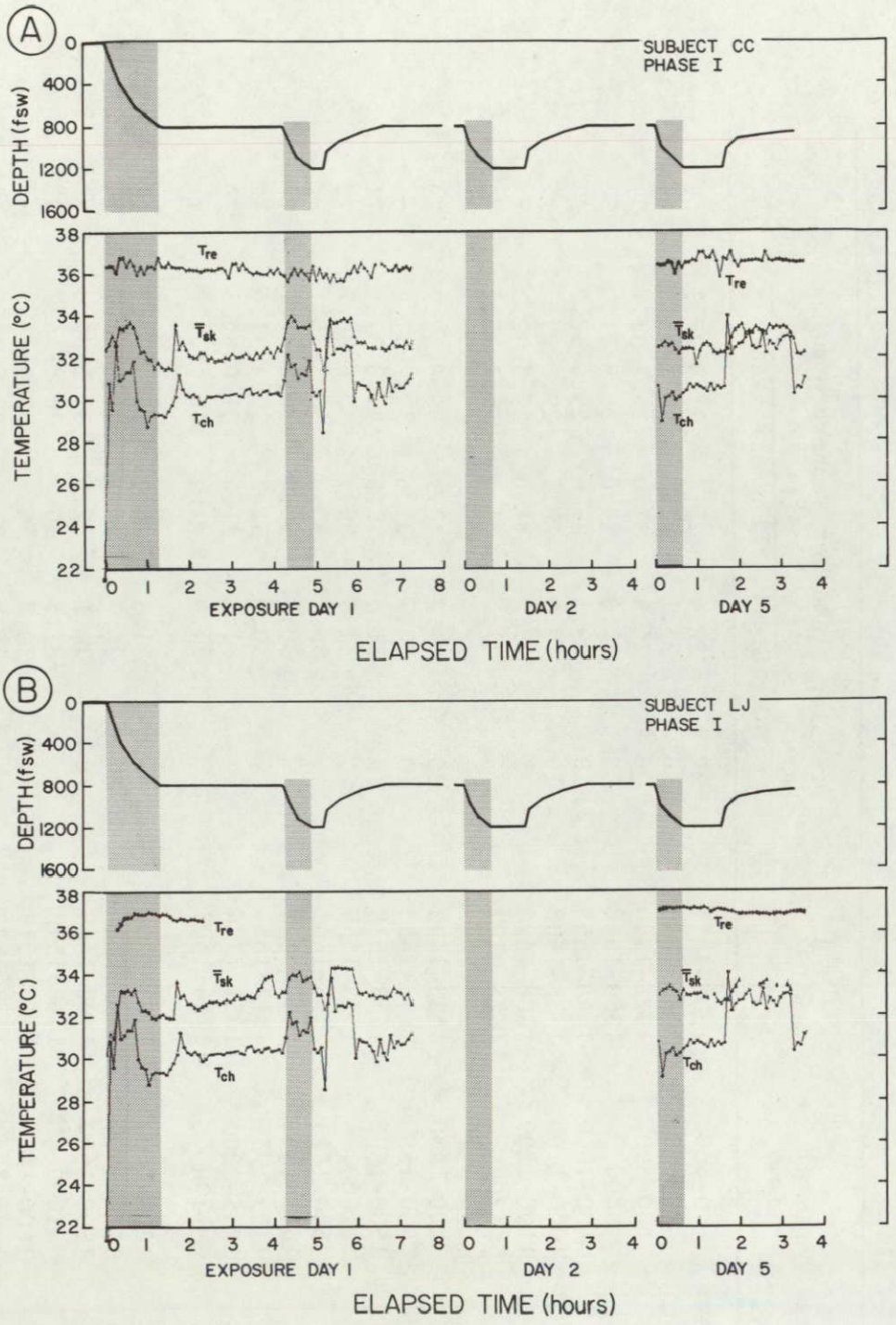

FIG. 1. Chamber and subject temperatures during experiment periods in Phase I for subjects CC and LJ. The data are plotted at five-minute intervals below the dive profile. Mean skin temperatures were calculated for subject CC using method #2, and for subject LJ using method #2 on exposure day 1 and method #1 on day 5. See text for explanation of fluctuations in T_{re} in subject CC. A, Subject CC; B, subject LJ.

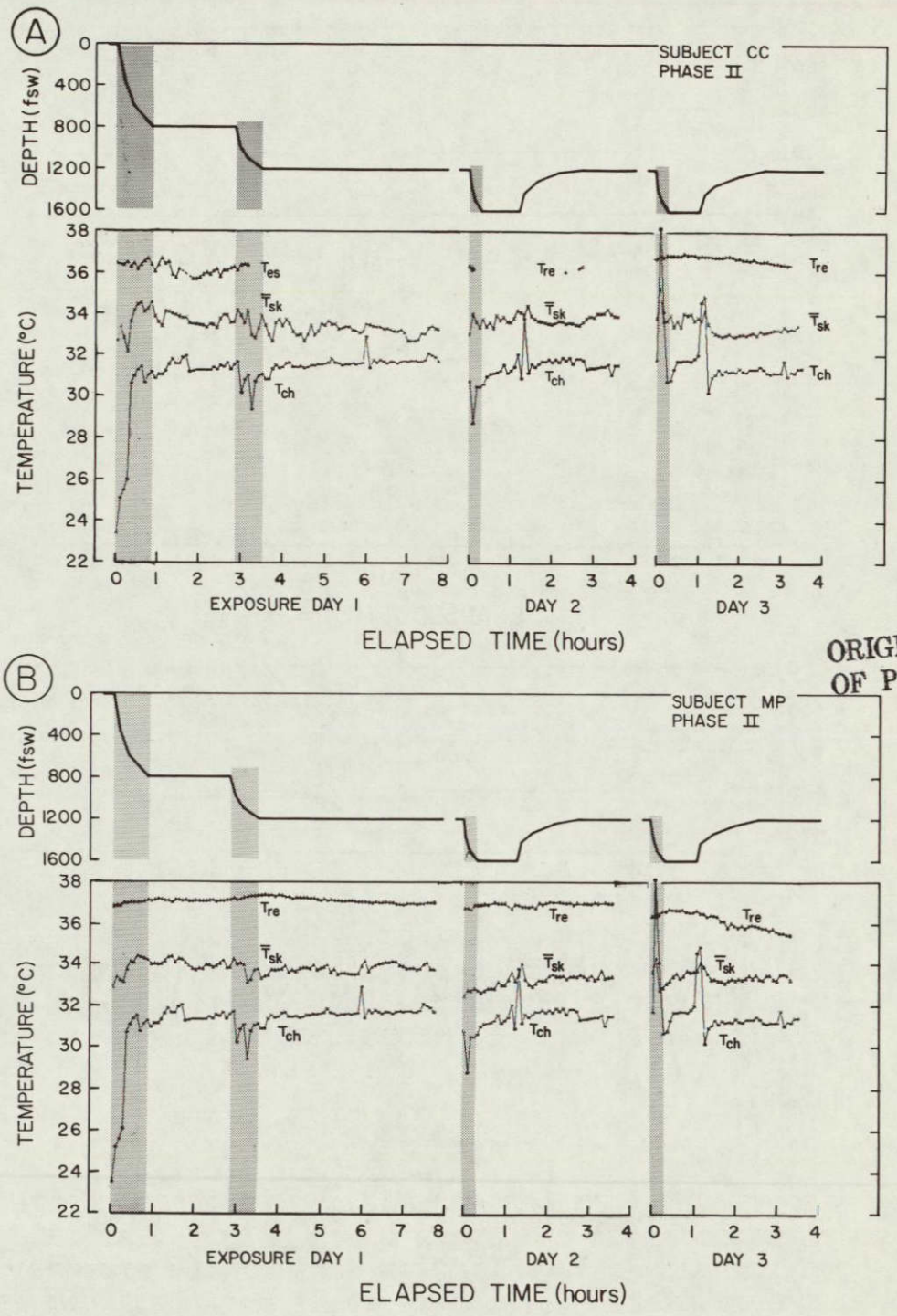

FIG. 2. Chamber and subject temperatures during experiment periods in Phase II for subjects CC and MP. The data are plotted at five-minute intervals below the dive profile. Mean skin temperatures were calculated for both subjects using method #2. A, Subject CC; B, subject MP.

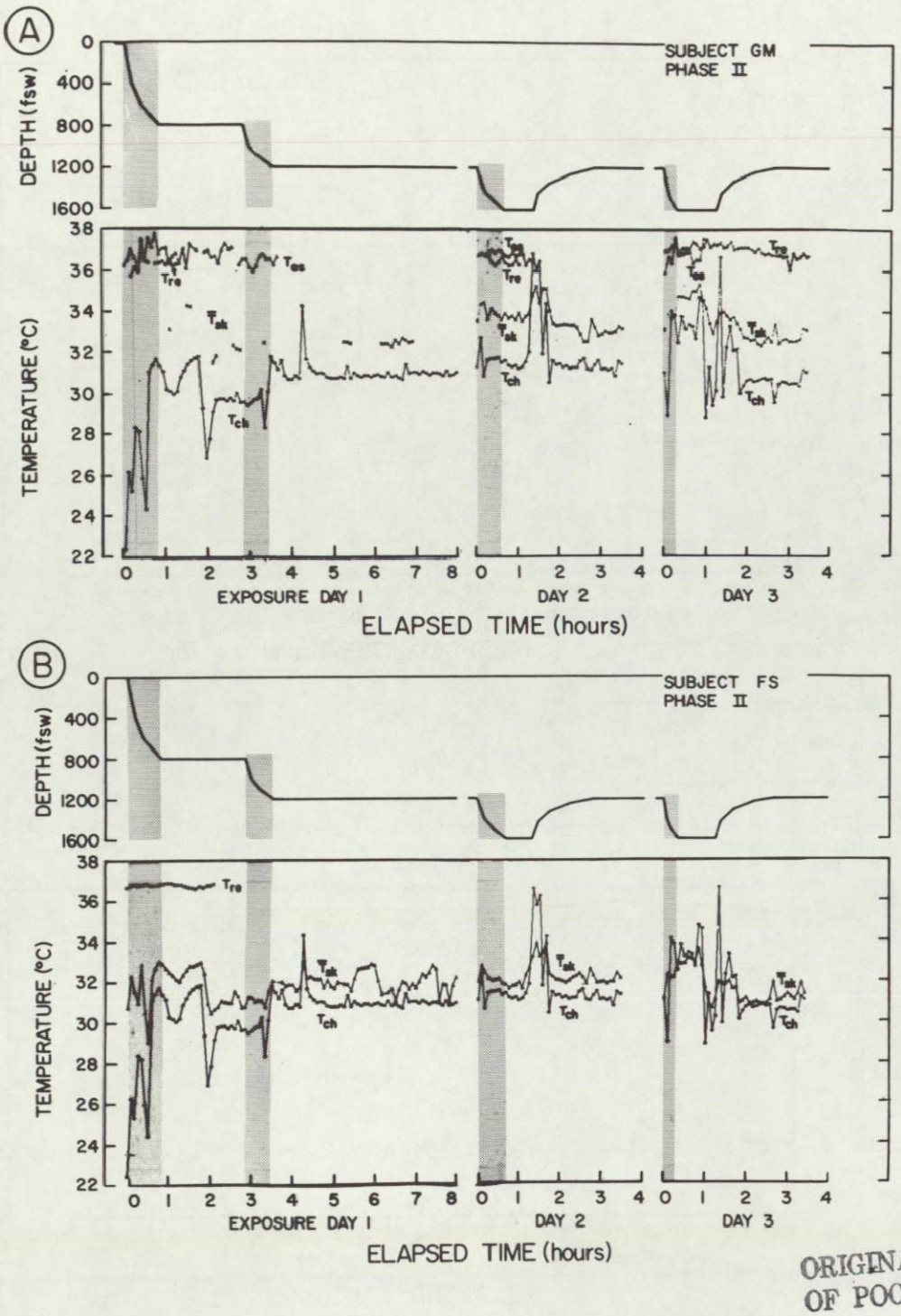

FIG. 3. Chamber and subject temperatures during experiment periods in Phase II for subjects GM and FS. The data are plotted at five-minute intervals below the dive profile. Mean skin temperatures were calculated for subject GM using method #2, and for subject FS using method #2 on exposure day 1 and method #1 on days 2 and 3. See text for explanation of fluctuations in T_{re} in subject GM. A, Subject GM; B, subject FS.

This led to momentary peak temperatures of 38.1°C and 37.1°C (Table III, exposure days 2 and 3, respectively).

Relative Humidity and Gas Velocity

Relative humidity during the experiments in Phase II ranged from a low of 57% to a high of 83% (Tables II and III). Relative humidities in Phase I were similar.

The horizontal component of gas velocity throughout the chamber was below the detection limit (0.1 m/sec) of the anemometer except at the carbon dioxide scrubber blower inlet (0.83 m/sec). The pattern of gas circulation from the heat exchange blowers located below the deck plates was oriented vertically at each end of the chamber.

THERMAL COMFORT RANGES

Table IV shows thermal comfort ranges as derived from subjects' comments recorded in the several dive logs and from the temperature data. The range at 1600 fsw has a low reliability since it was obtained during the relatively brief "bottom" times at this depth when the diver-subjects were preoccupied with experiments and with preparations for decompression.

TABLE IV. Thermal Comfort Ranges at Various Depths (Combined Data from Phases I and II)

Depth (fsw)	Low Limit (°C)	High Limit (°C)	Magnitude of Range (°C)
800	29.5	31.5	2.0
1200	30.8	32.5	1.7
1400	31.5	33.0	1.5
1600[a]	31.5	33.0	1.5

[a] Both subjects during a particular excursion at that depth were occupied with experiments for 29 of the 45 minutes at this depth.

SUBJECT TEMPERATURES DURING EXPERIMENTS

Figures 1-3 show that subject skin temperature generally closely paralleled chamber gas temperature, as expected. Rectal temperatures of the exercise subjects had brief elevations during the intermittent periods of exercise. Transient drops of rectal temperature to subnormal values in these subjects (Figs. 1A and 3A) are due to displacement of the probe (reported by CC and GM) and do not represent actual changes in deep body temperature.

Rectal temperatures usually increased slightly during compressions and were either stable or decreased somewhat during decompressions from deep excursions. While most changes were no more than several tenths of a degree C, changes of approximately 0.5°C occurred on some occasions, such as during compression from 0 to 800 fsw for rest subject MP, (Fig. 2B, exposure day 1), 800 to 1200 fsw for exercise subject CC (Fig. 1A, exposure day 5), 1200 to 1600 fsw for exercise subject GM (Fig. 3A, exposure day 3), and during decompressions from 1600 to 1200 fsw (Figs. 2B, 3A, exposure days 3). Esophageal temperatures only are shown on day 1 in Fig. 2A for subject CC in Phase II, since rectal temperatures on this subject were lost due to mechanical problems with the probe.

EARLY MORNING MEASUREMENTS

Data from early morning measurements are presented in Figs. 4 and 5 for Phase I and for Phase II in Figs. 6 and 7. The results of these measurements are as follows.

Rectal and Skin Temperatures

Core temperature showed no remarkable changes in either phase. The elevated temperature seen in subject WS on exposure day 6 in Phase I (Fig. 5A) was a febrile response to a severe dermatological infection.

The few measurements of skin temperature available in Phase I are all at elevated pressures. Mean skin temperatures are between 32.3 and 33.4°C for all subjects and show no consistent trends with exposure duration. In Phase II they are in the same range of values as in Phase I and the

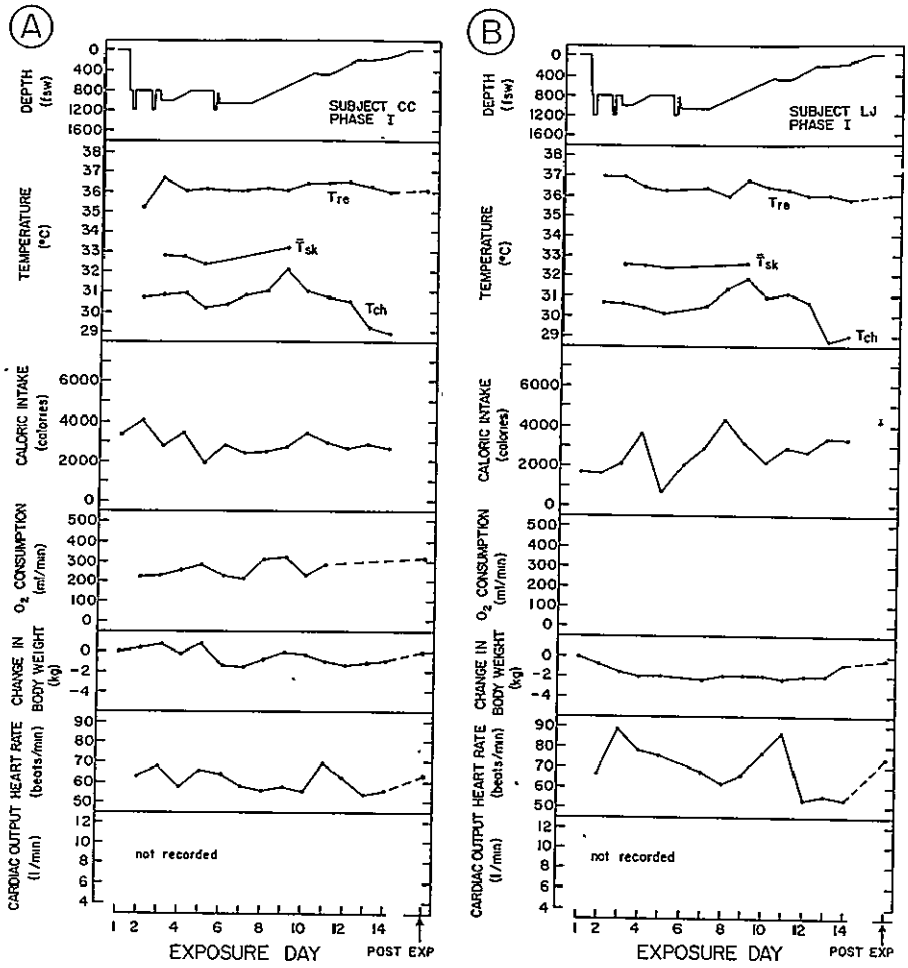

FIG. 4. Early morning thermal, metabolic and cardiac function measurements during Phase I for subjects CC and LJ. Dive profile is plotted as chamber pressure at 6 a.m. A, Subject CC; B, subject LJ.

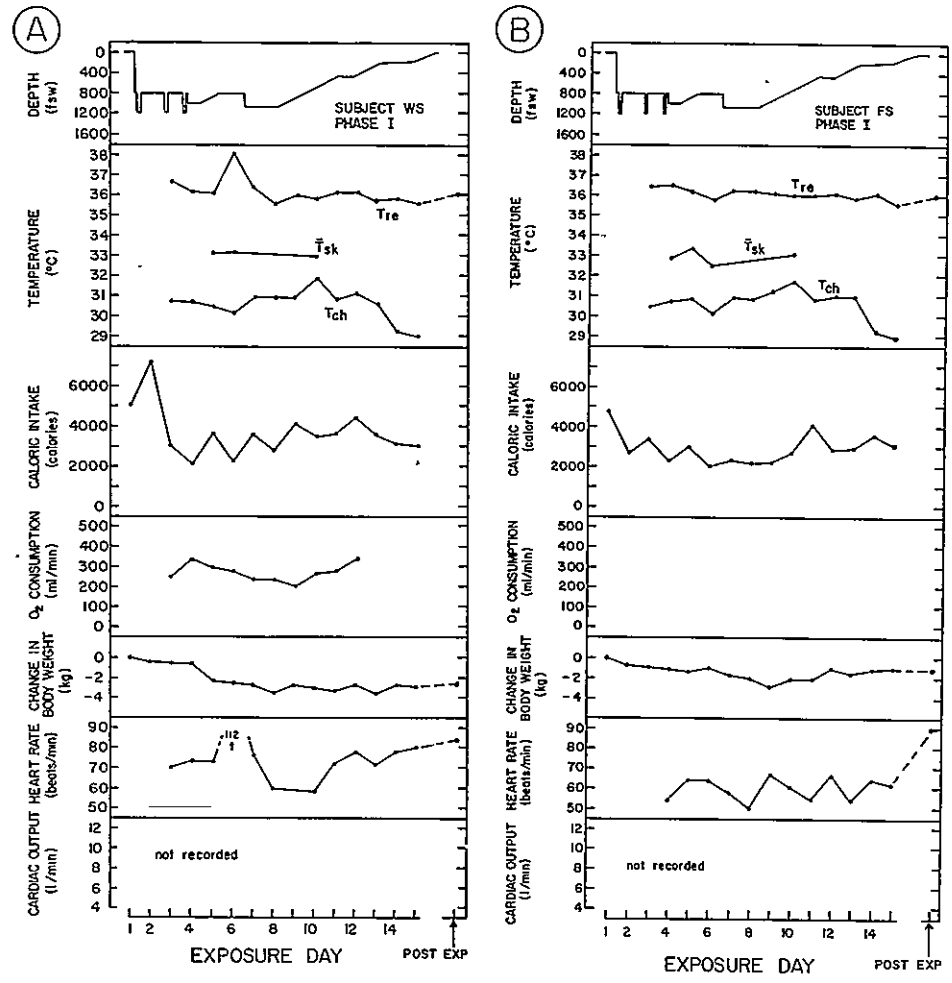

FIG. 5. Early morning thermal, metabolic and cardiac function measurements during Phase I for subjects WS and FS. Dive profile is plotted as chamber pressure at 6 a.m. A, Subject WS; B, subject FS.

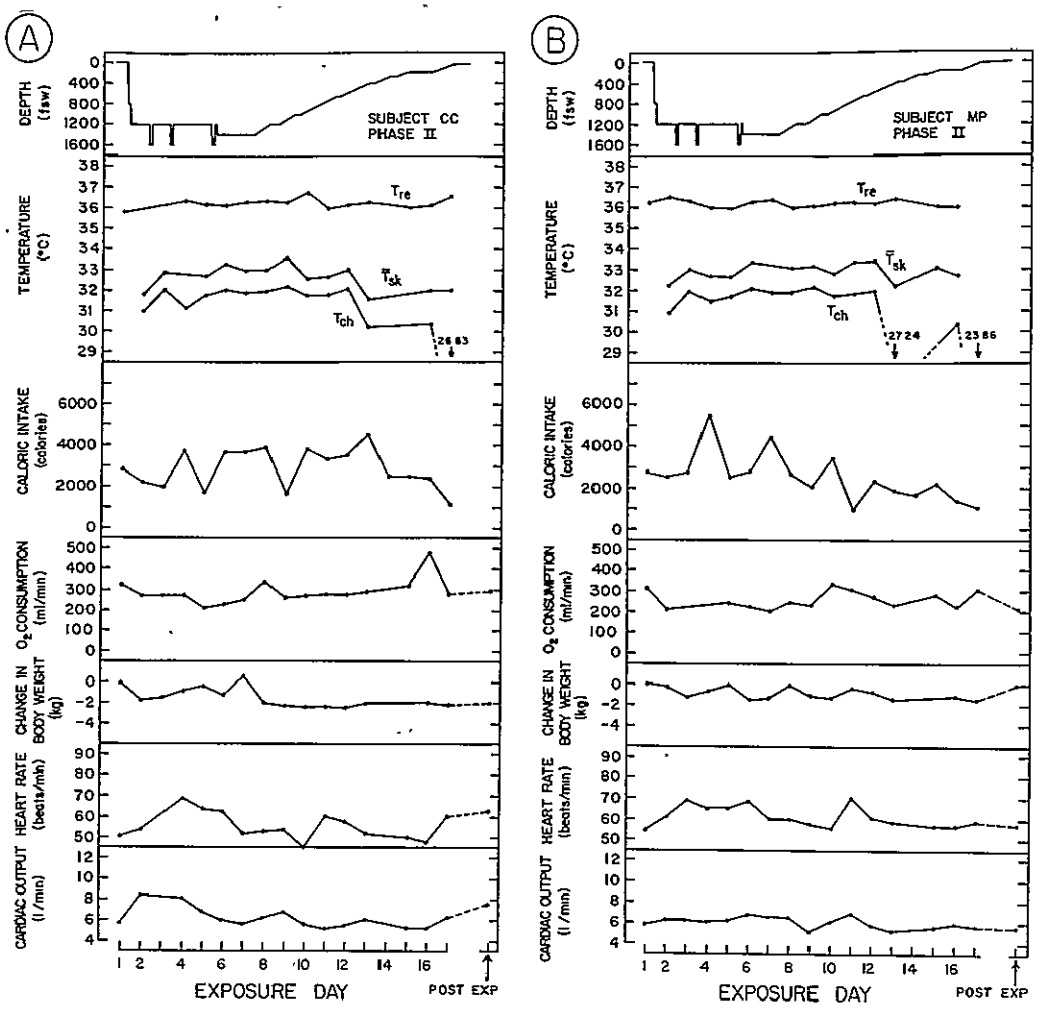

FIG. 6. Early morning thermal, metabolic and cardiac function measurements during Phase II for subjects CC and MP. Dive profile is plotted as chamber pressure at 6 a.m. A, Subject CC; B, subject MP.

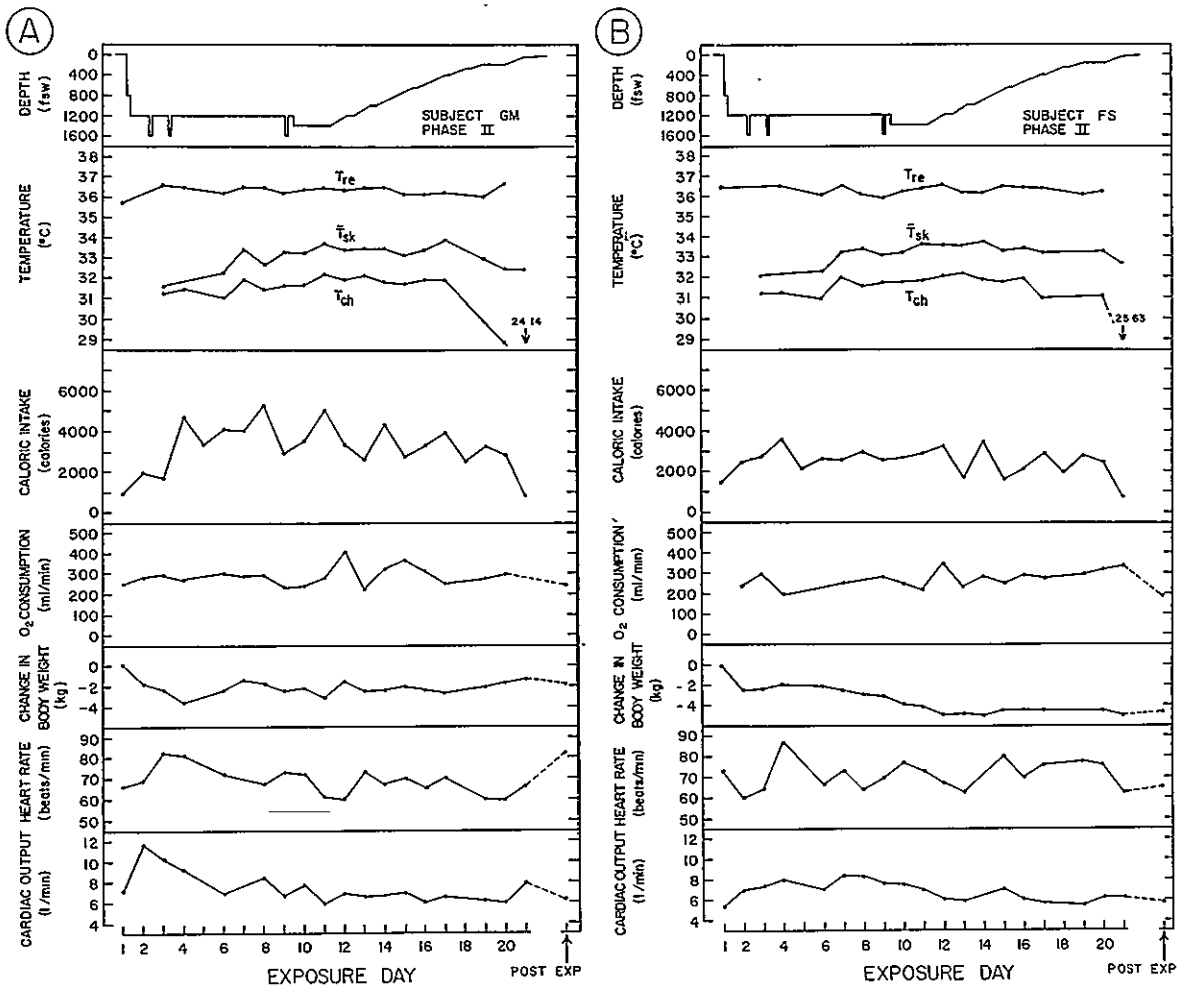

FIG. 7. Early morning thermal, metabolic and cardiac function measurements during Phase II for subjects GM and FS. Dive profile is plotted as chamber pressure at 6 a.m. A, Subject GM; B, subject FS.

TABLE V. Early Morning Thermal, Metabolic and Cardiac Function Measurements in Phase I (Subjects CC and LJ)

Exposure Day	Depth (fsw)	T_{ch} (°C)	T_{re} (°C)	$\bar{T}_{sk}$ [a] (°C)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (ml/min)	Caloric Intake[b]	Body Wt. (kg)	Body Wt. Change (kg)
Subject CC										
1	0	-	-	-	-	Not	-	3412	82.3	0
2	800	30.7	35.2	-	64	recorded	230	4084	82.6	+0.3
3	997	30.8	36.6	32.8	68	in this	232	2828	83.0	+0.7
4	821	30.9	36.1	32.7	58	phase	258	3474	82.1	-0.2
5	800	30.2	36.2	32.4	66		281	1928	83.1	+0.8
6	1050	30.4	36.0	-	64		233	2849	81.0	-1.3
7	1049	30.8	36.0	-	58		219	2444	80.8	-1.5
8	838	31.0	36.1	-	56		313	2495	81.5	-0.8
9	638	32.1	36.0	33.1	58		325	2721	82.3	0
10	453	31.0	36.3	-	56		241	3425	82.0	-0.3
11	387	30.7	36.4	-	70		287	2992	81.4	-0.9
12	233	30.5	36.5	-	62		-	2647	81.0	-1.3
13	200	29.2	36.2	-	54		-	2883	81.2	-1.1
14	110	28.9	35.9	-	56		-	2712	81.4	-0.9
Post-exp.	0	-	36 0	-	64		312	-	82.4	+0.1
Subject LJ										
1	0	-	-	-	-	Not	[c]	1695	72.4	0
2	800	30.7	37.0	-	67	recorded		1622	71.6	-0.8
3	990	30.7	37.0	32.6	89	in this		2166	70.9	-1.5
4	826	30.5	36.4	32.5	77	phase		3643	70.5	-1.9
5	800	30.2	36.3	32.3	76			717	70.5	-1.9
6	1046	-	-	-	-			2081	-	-
7	1046	30.5	36.4	-	68			2879	70.0	-2.4
8	841	31.3	36.0	-	62			4343	70.5	-1.9
9	635	31.8	36.8	32.6	66			3151	70.5	-1.9
10	451	30.9	36.5	-	76			2219	70.5	-1.9
11	385	31.1	36.3	-	87			2971	70.1	-2.3
12	231	30.7	36.0	-	54			2726	70.5	-1.9
13	200	28.7	36.0	-	56			3412	70.5	-1.9
14	111	29.0	35.8	-	54			3352	71.7	-0.7
Post-exp.	0	-	36.0	-	74			-	72.0	-0.4

[a] $\bar{T}_{sk}$ calculated by method #3 (see text).

[b] Caloric values shown in Tables V-VIII were calculated using the standard tables in: Composition of Foods· Raw--Processed--Prepared. U.S.D.A. Agriculture Handbook #8. Washington, D.C.: Government Printing Office, 1963.

[c] See text for explanation.

TABLE VI. Early Morning Thermal, Metabolic and Cardiac Function Measurements in Phase I (Subjects FS and WS)

Exposure Day	Depth (fsw)	T_{ch} (°C)	T_{re} (°C)	$\bar{T}_{sk}$ [a] (°C)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ [b] (ml/min)	Caloric Intake	Body Wt. (kg)	Body Wt. Change (kg)
Subject FS										
1	0	-	-	-	-	Not		4745	73.1	0
2	800	-	-	-	-	recorded		2639	72.3	-0.8
3	800	30.5	36.4	-	-	in this		3341	72.2	-1.0
4	994	30.7	36.5	32.9	54	phase		2234	72.0	-1.1
5	824	30.8	36.2	33.4	64			2954	71.8	-1.3
6	800	30.1	35.8	32.5	64			2013	72.1	-1.0
7	1050	30.9	36.2	-	58			2350	71.4	-1.7
8	1050	30.8	36.2	-	50			2198	71.1	-2.0
9	838	31.2	36.1	-	67			2255	70.3	-2.8
10	628	31.7	36.0	33.1	60			2687	70.9	-2.2
11	449	30.8	36.0	-	54			4028	70.9	-2.2
12	390	31.0	36.1	-	66			2818	72.0	-1.1
13	230	31.0	35.9	-	54			2920	71.5	-1.6
14	200	29.2	36.0	-	64			3542	71.9	-1.2
15	111	28.9	35.5	-	62			3057	72.0	-1.1
Post-exp.	0	-	36.0	-	90			-	71.8	-1.3
Subject WS										
1	0	-	-	-	-	Not	-	5175	85.6	0
2	800	-	-	-	-	recorded	-	7342	85.1	-0.5
3	800	30.7	36.6	-	70	in this	252	3019	85.1	-0.6
4	1000	30.7	36.2	-	73	phase	336	2192	85.0	-0.6
5	833	30.5	36.1	33.1	73		294	3702	83.3	-2.3
6	800	30.1	38.1	33.1	112		279	2276	83.0	-2.6
7	1050	30.9	36.2	-	76		234	3634	82.8	-2.8
8	1050	30.9	35.6	-	59		236	2806	82.0	-3.6
9	836	30.9	35.9	-	-		203	4190	82.8	-2.8
10	632	31.8	35.7	32.9	58		262	3557	82.6	-3.0
11	447	30.8	36.0	-	72		274	3659	82.3	-3.3
12	383	31.1	36.1	-	78		334	4473	82.9	-2.7
13	229	30.6	35.6	-	72		-	3642	82.0	-3.6
14	200	29.2	35.8	-	78		-	3149	82.9	-2.7
15	111	29.0	35.5	-	80		-	3070	82.7	-2.9
Post-exp.	0	-	36.0	-	84		-	-	82.9	-2.7

[a] $\bar{T}_{sk}$ calculated by method #3 (see text).

[b] See text for explanation.

TABLE VII. Early Morning Thermal, Metabolic and Cardiac Function Measurements in Phase II (Subjects CC and MP)

Exposure Day	Depth (fsw)	T_{ch} (°C)	T_{re} (°C)	$\bar{T}_{sk}$ [a] (°C)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (ml/min)	Caloric Intake	Body Wt. (kg)	Body Wt. Change (kg)
Subject CC										
1	0	-	35.8	-	51	5.7	325	2833	82.4	0
2	1200	31.0	-	31.9	54	8.5	276	2284	80.7	-1.7
3	1200	32.1	-	32.9	-	-	275	2062	81.0	-1.4
4	1200	31.2	36.3	-	69	8.1	276	3773	81.6	-0.8
5	1200	31.8	36.2	32.7	64	6.9	216	1707	82.1	-0.3
6	1400	32.1	36.1	33.3	63	6.1	229	3664	81.2	-1.2
7	1400	31.9	36.2	33.0	52	5.6	251	3699	83.0	+0.6
8	1224	32.0	36.4	33.0	53	6.2	334	3908	80.5	-1.9
9	1069	32.3	36.3	33.6	54	6.8	253	1693	80.2	-2.2
10	919	31.7	36.7	32.6	45	5.7	274	3818	80.1	-2.3
11	714	31.7	36.0	32.7	60	5.2	279	3375	80.1	-2.3
12	579	32.0	36.1	32.9	58	5.6	275	3542	80.0	-2.4
13	425	30.2	36.3	31.4	52	6.1	289	4495	80.4	-2.0
14	288	-	-	-	-	-	-	2554	-	-
15	200	-	36.0	-	50	5.3	323	2540	-	-
16	169	30.4	36.1	32.0	48	5.2	480	2425	80.5	-1.9
17	40	26.6	36.6	32.0	60	6.2	273	1154	80.3	-2.1
Post-exp.	0	-	-	-	63	7.6	294	-	80.5	-1.9
Subject MP										
1	0	-	36.3	-	55	5.9	318	2793	75.0	0
2	1200	31.0	36.5	32.3	61	6.3	217	2568	74.7	-0.3
3	1200	32.0	36.3	33.0	69	6.4	-	2573	73.8	-1.2
4	1200	31.5	36.0	32.7	65	6.1	-	5492	74.4	-0.6
5	1200	31.8	36.0	32.7	65	6.2	247	2532	75.0	0
6	1400	32.2	36.3	33.4	69	6.8	224	2795	73.6	-1.4
7	1400	32.0	36.4	32.9	60	6.6	205	4422	73.7	-1.3
8	1221	31.9	36.1	33.1	60	6.6	249	2684	74.9	-0.1
9	1066	32.2	36.2	33.2	58	5.2	236	2077	73.8	-1.2
10	921	31.8	36.2	32.9	56	6.1	336	3417	73.7	-1.3
11	711	31.8	36 3	33.4	70	6.9	305	992	74.7	-0.3
12	577	32.0	36.2	33.4	61	5.9	274	2344	74.3	-0.7
13	425	27.2	36.4	32.2	59	5.4	236	1967	73.6	-1.4
14	288	-	-	-	-	-	-	1716	-	-
15	200	-	36 1	33.1	57	5.6	282	2234	-	-
16	170	30.4	36.0	32.7	57	6.0	222	1395	73.9	-1.1
17	43	23.9	-	-	59	5.9	310	1090	73.5	-1.5
Post-exp.	0	-	-	-	58	5.6	218	-	75.0	0

[a] $\bar{T}_{sk}$ calculated by method 13 (see text).

TABLE VIII. Early Morning Thermal, Metabolic and Cardiac Function Measurements in Phase II (Subjects FS and GM)

Exposure Day	Depth (fsw)	T_{ch} (°C)	T_{re} (°C)	$\bar{T}_{sk}$ [a] (°C)	Heart Rate (beats/min)	Cardiac Output (l/min)	$\dot{V}_{O_2}$ (ml/min)	Caloric Intake	Body Wt. (kg)	Body Wt. Change (kg)
Subject FS										
1	0	-	36 4	-	73	5.4	-	1485	74.6	0
2	1200	-	-	-	60	7.0	242	2483	72.2	-2.4
3	1200	31.2	-	32.1	64	7.4	298	2718	72.3	-2.3
4	1200	31.3	36.5	-	87	8.1	206	3582	72.7	-1.9
5	1200	-	-	-	-	-	-	2135	-	-
6	1200	30.9	36.1	32.3	67	7.0	-	2661	72.6	-2.0
7	1200	32.0	36.5	33.2	73	8.5	255	2569	72.1	-2.5
8	1200	31.5	36.1	33.3	64	8.3	-	2965	71.7	-2.9
9	1200	31.7	35 8	33.1	69	7.6	279	2505	71.6	-3.0
10	1400	31.7	36.2	33.1	77	7.6	250	2626	70.7	-3.9
11	1400	31.8	36.4	33 6	73	7.1	218	2872	70.6	-4.0
12	1227	32.1	36.5	33.5	67	6.1	350	3127	69.8	-4.8
13	1075	32.1	36.1	33.5	63	6.0	232	1686	69.9	-4.7
14	926	31.8	36.1	33.7	-	-	285	3483	69.7	-4.9
15	718	31.8	36.4	33.2	80	7.2	257	1623	70 2	-4 4
16	581	31.9	36.4	33.4	70	6.0	288	2137	70.2	-4.4
17	425	30.9	36.3	33.2	76	5.8	275	2858	70.2	-4.4
18	288	-	-	-	-	-	-	1975	-	-
19	200	-	36.1	-	78	5.5	294	2754	-	-
20	171	31.6	36.2	33.3	76	6.3	317	2471	70.2	-4.4
21	40	25.6	-	32.6	63	6.4	336	798	69.6	-5.0
Post-exp.	0	-	-	-	65	5.8	187	-	70.0	-4.6
Subject GM										
1	0	-	35.8	-	66	7.2	252	926	79.2	0
2	1200	-	-	-	69	11.5	278	1923	77.4	-1.8
3	1200	31.2	36.6	31.5	82	10.2	293	1691	76.8	-2.4
4	1200	31.4	36 5	-	81	9.1	271	4741	75.6	-3 6
5	1200	-	-	-	-	-	-	3391	-	-
6	1200	31.0	36.2	32.3	72	6.9	301	4110	76.7	-2.5
7	1200	32.0	36.5	33.4	-	-	289	4077	77.8	-1.4
8	1200	31.5	36.5	32.7	67	8.5	291	5261	77.5	-1.7
9	1200	31 6	36.2	33.3	73	6.7	237	2943	76.7	-2.5
10	1400	31 7	36.4	33.2	72	7.7	240	3540	77.0	-2.2
11	1400	32.2	36.4	33.7	61	5.8	278	5061	76.0	-3.2
12	1230	32.0	36.3	33.4	60	6.8	405	3394	77.6	-1 6
13	1072	32.2	36.4	33.5	73	6.6	222	2637	76.7	-2.5
14	924	31.8	36.5	33.4	67	6.7	322	4345	76.8	-2.4
15	720	31.8	36.1	33.1	70	6.8	366	2756	77.1	-2.1
16	584	31.9	36.1	33.4	65	6.1	310	3305	76.8	-2.4
17	425	31.9	36.2	33.8	70	6.5	252	3964	76.5	-2.7
18	288	-	-	-	-	-	-	2555	-	-
19	200	-	36.0	-	60	6.2	277	3245	-	-
20	173	28.9	36.7	32.5	60	6.2	299	2861	77.5	-1.7
21	45	24.1	-	32.4	66	7.9	-	857	77.9	-1.3
Post-exp.	0	-	-	-	82	6.4	245	-	77.4	-1.8

[a] $\bar{T}_{sk}$ calculated by method #3 (see text).

direction of their changes generally parallel those in chamber ambient temperature.

Caloric Intake and Body Weight

Daily caloric intake during confinement in the chamber reflected maintenance of a healthy appetite. Pre-exposure data were not sufficient to establish a reliable baseline for comparison with data in the experimental period. During the Phase I pressure exposure, caloric intake generally increased with time in subject LJ, decreased in CC, and showed no overall trend in FS and WS (Tables V and VI, Figs. 4 and 5).

In Phase II, GM had an initial upward trend with a decline starting on exposure day 8 (Fig. 7A, Table VIII). Subjects CC, MP and FS showed no consistent trends except for a decline in MP's values after day 10 (Figs. 6 and 7B, Tables VII and VIII). Exercise subjects always had higher mean values than rest subjects. All subjects lost weight in both phases, with those exposed longest (FS and WS in Phase I; GM and FS in Phase II) losing most (Figs. 6 and 7, Tables VI and VIII).

Oxygen Consumption ($\dot{V}_{O_2}$)

Omissions in the $\dot{V}_{O_2}$ columns of Tables V and VI are due to technical difficulties with the gas exchange measurements. No data are presented for subjects FS and LJ and no sea level control data are available for WS in Phase I. In Phase II, using the mean of the pre- and post-exposure air environment values as control, subjects GM and FS had higher $\dot{V}_{O_2}$ values while exposed to helium-oxygen, subject CC had lower values, and MP had values approximately evenly divided above and below the control level.

Heart Rates

Pre-exposure measurements at 1 ata are available for the four subjects of Phase II (Tables VII and VIII, Figs. 6 and 7).

On the first day following compression from the surface, there was elevation of rate in three subjects (CC, GM, MP) and reduction in one (FS). During residence at depth, the subjects' heart rates fluctuated from day to day but four subjects (CC, LJ, GM, MP) had an overall decline over the exposure period while two had heart rates which increased (FS, WS). Those four subjects whose rates were elevated with compression also showed the decrease with time at depth. During decompression from saturation exposures, one subject (WS) had an increasing rate, two (LJ, GM) had decreasing rates and in three (CC, MP, FS) there were no consistent trends (Tables V-VIII, Figs. 4-7).

Cardiac Output

Three of the four subjects (CC, GM, FS) in Phase II had marked elevations in cardiac output beginning early in the pressure exposure, while the fourth (MP) had only a small increase. Cardiac output gradually returned to or toward the pre-exposure 1 ata level as the exposure progressed. Subjects CC and GM were at the control level within seven days, while cardiac output of FS was still above the control level at the conclusion of the pressure exposure.

DISCUSSION

CHAMBER ENVIRONMENT

The environment during both phases was moderately humid with generally comfortable temperature. The effort to minimize temperature change during compressions and decompressions was successful. A compression to 1600 fsw illustrates the extreme sensitivity of men to even small deviations from the comfort zone in the helium-oxygen environment. On his third exposure day in Phase II, subject FS had an abrupt onset of violent whole body shivering tremors when chamber temperature fell 3°C within five minutes (Fig. 3B). Within one minute of the start of tremor the chamber temperature was increased by about 5°C and the tremors subsided within 30 seconds. The subject said that he felt cold during this period and was shivering. This effect of the tight coupling of skin temperature to ambient temperature in the highly conductive helium

environment illustrates the need for a precise chamber temperature control system with high capacity and rapid response characteristics (see Section D). It also illustrates the importance of monitoring ambient and body temperatures, particularly during periods of tremor or psychomotor measurements, to prevent misinterpretation of temperature-related shivering as a hydrostatic pressure effect on the central nervous system.

Increased tremor in association with temperature elevation, as observed by others (1,2), was not apparent in these exposures (see also Section E-4). Thermal comfort ranges for our subjects did not vary greatly from those demonstrated elsewhere (10,12,14,16,19). Characteristically, the ranges progressively elevated and narrowed with increasing depth (Table IV).

Although the extremely low gas velocities at pressure minimized body heat loss via forced convection, this advantage was somewhat offset by the resultant non-uniform pattern of ambient temperatures in the chamber. Temperature differences of 2.9°C between different locations were observed. The higher areas in the chamber were warmer due to both natural convection of warm gas and local heating by the chamber lights. Thus a head-to-foot thermal gradient existed, which was most marked for the exercise subject who sat upright on the bicycle ergometer. The significance of this non-uniformity became apparent when the exercise subject complained of excessive heat; lowering the chamber ambient temperature resulted in a complaint from the rest subject, located at the other end of the chamber and at a lower position, that he felt cool. Adequate gas movement at low velocity is therefore necessary to achieve uniform temperature distribution and still avoid subject discomfort from convective heat loss.

POSSIBLE TEMPERATURE INFLUENCES ON HYDROSTATIC EFFECTS

The observation that the exercise subjects appeared to have more severe symptoms than the rest subjects (Section E-1) may be related in part to a combination of thermal stresses including the subjects' location in a warmer part of the chamber, their intermittent generation of heat by physical work, and the reduction in the effectiveness of evaporative heat loss from perspiration during exercise in

the humid, dense environment (20). These subjects complained of the heat prior to and during their compression-related symptomatic episodes and cooling the chamber seemed to aid in their recovery. Such apparent associations may be related to the lowering of convulsive threshold in rats by increased ambient temperature (3) and to the observations that tremors in man are associated with temperature rise during compression in "dry" dives but are less prevalent in "wet" dives where skin temperature does not rise (1,2). A relationship between hippocampal theta rhythm and cutaneous temperature in the rabbit at sea level in air has also been reported (8).

SUBJECT THERMAL BALANCE

Although deep body temperature was not completely protected from chamber temperature change during compressions and decompressions, severe thermal stresses were generally avoided. However, the subjects were in a state of "precarious thermal balance" (18) the effects of which could be minimized but not eliminated by control of chamber temperature.

Measurements of deep body temperature with both the esophageal and rectal probes were influenced by factors which caused temperatures observed at these sites to vary significantly (Fig. 3A). The high thermal conductivity of the He-O_2 environment apparently caused a respiratory influence on esophageal temperature, by both the inspired gas temperature and the level of ventilation. On the other hand, rectal temperatures of the exercise subjects were affected by the working leg muscles, the warmed venous blood of which passed close to the rectal probe.

The elevation of skin temperature during the high pressure He-O_2 exposures may have caused peripheral vasodilatation with an increase in cutaneous blood flow (17). Reflex responses to a concomitant reduction of total peripheral resistance could have induced the elevated heart rates found in four of the six subjects as well as the increased cardiac outputs observed in three of the four subjects studied.

An undesirable effect of increased cutaneous blood flow would be a concurrent increment in heat loss by augmentation

of the equivalent thermal conductivity of the body's boundary layer. Such an inappropriate response to the increased thermal conductivity of the helium environment would partially offset the beneficial effect of raising ambient temperature. With chamber temperature in the comfort zone, the deep tissue temperature sensors remain protected against thermal imbalance as evidenced by the small changes in early morning rectal temperatures. However, regional alterations in temperature may occur, such as in the extremities, as well as on the body surface. Local increases in oxygen consumption of warmed tissues may then be responsible for minor elevations in whole body oxygen uptake, providing a mechanism to support the hypothesis that a small but steady caloric drain occurs in helium-oxygen environments at pressure (14). Such increments would be difficult to substantiate, since even significant regional increases can be quite small compared with the total whole-body $\dot{V}_{O_2}$ and may be obscured by experimental error (14,16,18).

PROGRESSIVE WEIGHT LOSS

Despite the subjects' apparently adequate caloric intake, good appetite, and no significant overall change in $\dot{V}_{O_2}$, a consistent weight loss similar to that seen in several other exposures (12-14, 16) occurred. The degree of weight loss here appeared to be related to duration of exposure since, in both phases, the subjects exposed longest lost the most weight. The exercise subjects, with higher caloric intakes than the rest subjects, also lost weight.

The source of the weight loss remains unexplained despite detailed investigation (21). It may be partially explained by water losses (see Section E-18). Vomiting prevented the complete absorption of ingested food in subject GM on the first day of Phase II. The greatest weight loss occurred in subject FS who had the highest sweat output. Although all of these factors contribute to the loss of body weight and together may account for the entire loss, it is also possible that a small but persistent thermal-metabolic drain (14) may be involved. A small increment in $\dot{V}_{O_2}$ by such a means could easily go undetected by gas exchange measurements, since a sustained $\dot{V}_{O_2}$ elevation of only 10 cc/min would produce a weight loss of 0.2 pounds in 10 days.

SUMMARY AND CONCLUSIONS

Temperature of the ambient environment during compressions was well-controlled and subject comfort was usually maintained, despite the progressive increase in comfort temperature and narrowing of the comfort zone. Even small transient deviations of ambient gas temperature caused either shivering and tremor or complaints of excessive heat. Chamber temperatures were also well-controlled during the relatively rapid decompressions from excursions.

Gas velocity was low enough to avoid sensations of convective heat loss while at the same time the volume of circulating gas was sufficient to satisfy heat exchange requirements for control of chamber temperature. However, gas flow distribution was not adequate to prevent temperature gradients in the vertical cross section of the chamber.

Exercise subjects, exposed to warmer ambient temperatures and affected by heat generation from physical work, had more severe symptoms than rest subjects. They subjectively associated heat with symptomatic episodes and cooling with relief. Thus even a small temperature rise may exacerbate symptoms of rapid compression.

Elevation of ambient temperatures in the high pressure He-O_2 environment reduced body heat loss to levels consistent with subjective comfort and protection of deep body temperature sensors. However, elevated resting heart rates and cardiac outputs on the first exposure days in most of the subjects indicated the presence of a mild cardiovascular stimulus. One possible source of such an alteration is reduction in total peripheral resistance caused by cutaneous vasodilatation in response to an elevated skin temperature.

Elevation of ambient temperature is an appropriate compensatory response to the increased thermal conductivity of helium. If concomitant elevation in skin temperature causes increased cutaneous perfusion, the resultant increment in thermal conductivity will be an inappropriate response, which might produce regional temperature alterations in the extremities as well as on the body surface.

Body weight decreased progressively during the exposures to high pressure He-O_2 despite adequate caloric intake and unchanged whole body $\dot{V}_{O_2}$.

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E-16. VENTILATION AT REST DURING COMPRESSION AND AT STABLE HIGH PRESSURES

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The increased density of compressed nitrogen-oxygen mixtures at the moderately elevated ambient pressures of diving with compressed air has been associated with increased respiratory flow resistance, hypoventilation (7) and a reduced respiratory response to carbon dioxide (13). Effects of extremely high gas densities of respired gas on ventilation have been studied by using neon-helium-oxygen gas mixtures at step-wise elevations of pressures to 37 ata to achieve densities as high as 25 g/l (10). Such studies have made it possible to predict density-limited ventilatory capabilities during exercise (10), during voluntary hyperventilation (16), and during exposure to increased inspired P_{CO_2} (4,5) in helium-oxygen atmospheres of equivalent density (e.g., densities expected at depths as great as 5000 fsw). In the absence of conditions stimulating to respiration, ventilation at rest (and in sleep) is characterized by a balance between CO_2 production and ventilation (8). When metabolic rate is constant, any change in ventilation is opposed and minimized by a resultant change in arterial P_{CO_2} (Pa_{CO_2}) to a degree determined by an individual's respiratory sensitivity to the effects of CO_2 (9).

Sustained interference with CO_2 elimination results in elevated Pa_{CO_2} levels, while an increase in CO_2 elimination beyond metabolic need produces hypocapnia. Both of these can have significant sequelae. Due to the potential for

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CO_2 accumulation in chambers, habitats and breathing equipment, and due to the increased respiratory flow resistance induced by breathing equipment and dense gases, diving physiology (in contrast with altitude exposure) is more commonly concerned with the effects of hypercapnia than with excessive ventilation (11). Hypocapnia itself has caused problems in aviation (mental efficiency is impaired, and tetany can occur when $P_{a}CO_2$ is less than 25 mm Hg) (11), but it is usually not considered a problem in diving.

While neither hypo- nor hypercapnia was expected during compression in these stressful studies, it was possible that hyperventilation might result from general excitement, or from excitation of the central nervous system by hydrostatic pressure or other influences related to compression. The possibility that bulk movement of gas into the lungs during the relatively rapid compression might interfere with CO_2 elimination was also considered. It was recognized that possible consequences of altered brain CO_2 tension and hydrogen ion concentration might include exacerbation of the symptoms associated with compression and pressure. For these reasons end-tidal CO_2 partial pressure of subjects while at rest was examined to evaluate CO_2 homeostasis while they were exposed to the acute effects of compression and pressure.

An additional interest in CO_2 homeostasis related to the suggestion that hydrostatic pressure per se may cause CO_2 retention in resting man (19). Measurements of ventilation and end-tidal $P_{}CO_2$ at rest soon after awakening in the morning (ca. 6:30 A.M.) permitted evaluation of CO_2 balance over a range of stable hydrostatic pressures from 1 to 37 ata.

METHODS

APPARATUS

All resting measurements were taken as the subjects sat on a bicycle ergometer. The apparatus, conditions and procedures during compression and in the morning measurements are generally described in Sections E-14 and E-15, respectively.

Details relevant to the end-tidal P_{CO_2} ($P_{A_{CO_2}}$) measurements not included in Sections E-14 and E-15 are as follows. A sample of respired gas was continuously conducted via 1 mm inner diameter nylon tubing from the non-rebreathing valve to the chamber hull. Low dead-space, in-line isolation valves and stainless steel tubing carried the sample gas through the chamber hull to the infrared CO_2 analyzer outside the chamber. The entire path of gas flow was free of major dimensional discontinuities along its eight-foot length. The total flow withdrawn from the airway was approximately 4 standard liters/minute (l/min), with 0.5 l/min of this passing through the CO_2 analyzer. The excess flow bypassed the analyzer, reducing the transport lag from sample site to analyzer and helping to preserve the characteristic expired CO_2 wave form, which was recorded on a rapid response recorder (MFE model M-52, wide chart).

Three calibrating gases, appropriately selected for each ambient pressure condition studied, were employed to standardize the CO_2 analyzer. Their rated accuracy was such (Section E-14) that their contribution to error was less than ± 1 mm Hg. The calibration and measurement system was designed to permit frequent and rapid recalibration during periods of rapidly changing pressure.

MEASUREMENTS DURING COMPRESSION-PRESSURE EXPOSURES

In addition to ventilation and gas exchange, end-tidal gas tensions were measured on the two "exercise subjects" during the compression-pressure exposures of Phase II (Section E-14). End-tidal gas tension measurements were obtained on subject CC during his first three exposure days, involving compressions from 1 ata to the pressure equivalents of 800, 1200 and 1600 fsw. End-tidal gas sampling was performed on GM only during his 1200- to 1600-fsw excursions.

EARLY MORNING MEASUREMENTS

Early morning measurements including end-tidal gas tensions were made on the four subjects of Phase II (CC, GM, MP, FS). Data reported here relate to findings after two subjects (CC, MP) had been exposed to the high pressure, helium-oxygen environment at 37 ata for 8 days. The other

two subjects (GM, FS) had been exposed to this pressure for 12 days. All experiments at the nine elevated pressures examined during decompression were in a helium-oxygen filled chamber. The post-decompression measurements at 1 ata were in the same chamber, filled with air.

RESULTS

Some of the data summarized in Tables I and II also appear in Sections E-14 and E-15. Table I provides the values for the resting pre-exercise measurements during the acute phases of compression and exposure to high ambient pressure. Table II shows the morning measurements after multi-day adaptation to compression and pressure.

COMPRESSION AND INITIAL EXPOSURE TO INCREASED PRESSURE: VALUES OF $P_{A_{CO_2}}$, VENTILATION AND GAS EXCHANGE

Overall mean values of $P_{A_{CO_2}}$ while at rest during the acute phases of the compression-pressure exposures were 38.1 mm Hg and 36.0 mm Hg for CC and GM, respectively (Table I). Mean values before compression, during compression, at stable pressures after compression and after decompressions from excursions were as follows: CC - 36.9, 41.6, 39.4, 35.4 mm Hg; GM - 37.1, 37.5, 35.7, 35.3 mm Hg. Most ventilation ($\dot{V}_E$) and CO_2 production ($\dot{V}_{CO_2}$) values during the acute phase of exposure were greater than morning values in adapted states (Tables I and II; Fig. 2).

VENTILATION AND GAS EXCHANGE IN THE MORNING

Although the data graphed in Fig. 1 were obtained during decompression and therefore in order of the decreasing pressures of the decompression period, they will be described in terms of increasing pressure. Average $P_{A_{CO_2}}$ was was essentially constant from 1 to 28.9 ata (Table II). At the two highest pressures at which $P_{A_{CO_2}}$ measurements were made (33.4 and 38.0 ata), $P_{A_{CO_2}}$ was several mm Hg below the level of the other values. Average $\dot{V}_{CO_2}$ values were essentially constant with a small elevation near 7.1 ata. Mean $\dot{V}_E$ was less at lower pressures than at higher pressures.

TABLE I. Values of $P_{A_{CO_2}}$, $\dot{V}_E$ and $\dot{V}_{CO_2}$ at Rest During Compression-Pressure Modules in Phase II

Exposure Conditions	SUBJECT CC					SUBJECT GM				
	Module	Depth (fsw)	$P_{A_{CO_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{CO_2}$ (l/min STPD)	Module	Depth (fsw)	$P_{A_{CO_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{CO_2}$ (l/min STPD)
Exposure Day 1										
Pre-Compression	1	0	37.8	12.71	0.33			$P_{A_{CO_2}}$ not measured		
Compression	4	704	40.2	12.33	0.31					
Compression	10	1140	44.4	18.09	0.50					
Post-Compression	11	1200	41.3	14.60	0.33					
Post-Compression	13	1200	39.3	12.68	0.30					
Post-Compression	15	1200	39.8	12.03	0.28					
Exposure Day 2										
Pre-Compression	1	1200	38.0	13.11	0.34	1	1200	37.1	14.86	0.34
Compression	3	1460	41.5	14.53	0.37	3	1380	40.7	13.10	0.32
Compression		-	-	-	-	4	1540	33.1	13.32	0.30
Post-Compression	4	1600	37.0	14.23	0.34	5	1600	35.1	14.74	0.31
Post-Decompression	8	1200	30.3	13.09	0.27	9	1200	34.6	12.42	0.27
Post-Decompression	8	1200	35.9	13.04	0.30	9	1200	36.4	12.96	0.28
Post-Decompression	8	1200	33.8	11.30	-	9	1200	35.6	10.96	-
Exposure Day 3										
Pre-Compression	1	1200	35.0	14.56	0.34	-	-	-	-	-
Compression	3	1460	40.4	14.95	0.38	3	1460	38.6	15.74	0.37
Post-Compression	4	1600	39.4	13.92	0.32	4	1600	36.3	13.72	0.30
Post-Decompression	8	1200	37.4	10.96	0.24	8	1200	35.6	10.78	0.25
Post-Decompression	8	1200	37.3	9.95	0.22	8	1200	34.2	11.79	0.28
Post-Decompression	8	1200	37.6	10.00	-	8	1200	35.1	10.38	-
Overall Mean			38.1					36.0		
Pre-Compression Mean			36.9					37.1		
Compression Mean			41.6					37.5		
Post-Compression Mean			39.4					35.7		
Post-Decompression Mean			35.4					35.3		

TABLE II. Morning Rest Values of Ventilation and Gas Exchange During Decompression in Phase II

Pressure (ata)	Density (g/l)	Subject	$P_{A_{CO_2}}$ (mm Hg)	$\dot{V}_E$ (l/min BTPS)	f (br/min)	V_T (l/br)	$\dot{V}_{CO_2}$ (l/min STPD)	R	$P_{I_{O_2}}$ (mm Hg)	T_{re} (°C)
1.0	1.20	FS	38.1	6.39	5	1.28	0.20	1.07		-
		GM	34.4	10.05	14	0.72	0.24	0.99		-
		MP	31.8	9.85	12	0.82	0.24	1.08		-
		CC	37.9	10.68	14	0.76	0.28	0.96		-
		Mean	35.6	9.24	11	0.90	0.24		160	
		±SEM	1.5	0.97	2	0.13	0.02			
2.3	0.96	FS	37.5	7.79	8	0.97	0.24	0.70	-	-
		GM	36.4	9.41	16	0.59	0.27	0.97	-	-
		MP	33.7	10.04	15	0 67	0.23	0.75	-	-
		CC	35.7	8.47	14	0.61	0.20	0.73	-	36.6
		Mean	35.8	8.93	13	0.71	0.23		-	
		±SEM	0.8	0.50	2	0.09	0.01			
6.2	1.68	FS	41.1	6.87	6	1.15	0.23	0.72	396	36.2
		GM	38.7	10.49	14	0.75	0.29	0.96	392	36.7
		MP	35.2	8.02	10	0.80	0.23	1.04	394	36.0
		CC	36.4	10.28	17	0.60	0.27	0.56	377	36.1
		Mean	37.9	8.92	12	0.83	0.26		390	
		±SEM	1.3	0.88	2	0.12	0.02		4	
7.1	1.81	FS	41.0	8.14	13	0.63	0.26	0.88	390	36.1
		GM	36.5	9.44	13	0.73	0.27	0.96	394	36.0
		MP	31.2	8.96	12	0.75	0.25	0.88	409	36.1
		CC	37.9	10.87	16	0 68	0.29	0.91	406	36.0
		Mean	36.7	9.35	14	0.69	0.27		400	
		±SEM	2.0	0.57	1	0.03	0.01		5	
13.9	3.00	FS	41.1	7.06	7	1.01	0.22	0.80	399	36.3
		GM	39.4	9.93	15	0.66	0.25	0.98	399	36.2
		MP	32.9	8.50	10	0.85	0.20	0.85	399	36.4
		CC	35.6	10.66	16	0.67	0.26	0.89	396	36.3
		Mean	37.3	9.04	12	0.80	0.23		398	
		±SEM	1.8	0.80	2	0.08	0.01		1	
18.6	3.81	FS	42.6	7.87	10	0.79	0.23	0.81	394	36.4
		GM	35.4	11.35	14	0.81	0.24	0.77	397	36.1
		MP	32.9	10.54	12	0.88	0.22	0.80	388	36.2
		CC	39.1	10.72	15	0.72	0.25	0.90	391	36.1
		Mean	37.5	10.12	13	0.80	0.24		392	
		±SEM	2.1	0.77	1	0.03	0.01		2	
22.7	4.53	FS	39.2	8.47	12	0.70	0.22	0.88	380	36.4
		GM	35.4	12.66	15	0.84	0.27	0.74	389	36.1
		MP	31.1	11.86	12	0.99	0.24[a]	-	-	36.6
		CC	35.7	10.87	16	0.68	0.25	0.88	379	36.0
		Mean	35.3	10.96	14	0.80	0.24		383	
		±SEM	1.7	0.91	1	0.07	0.01		3	
28.9	5.60	FS	40.1	8.55	11	0.78	0.23	0.79	392	36.1
		GM	30.4	12.36	18	0.69	0.23	0.70	394	36.5
		MP	33.5	10.83	14	0.77	0.25	0.75	388	36.2
		CC	42.3	11.21	17	0.66	0.22	0.81	387	36.7
		Mean	36.6	10.74	15	0.72	0.23		390	
		±SEM	2.8	0.80	2	0.03	0.01		2	
33.4	6.38	FS	34.7	8.86	13	0.68	0.21	0.92	384	36.1
		GM	29.1	11.19	17	0.66	0.21	0.94	382	36.4
		MP	31.3	10.90	13	0.84	0.24	1.00	388	36.2
		CC	29.8	12.25	16	0.77	0.25	0.98	390	36.3
		Mean	31.2	10.80	15	0.74	0.23		386	
		±SEM	1.2	0.71	1	0.04	0.01		2	
38.0	7.20	FS	35.4	10.55	13	0.81	0.24	0.69	394	36.5
		GM	30.7	11.43	15	0.76	0.22	0.54	397	36.3
		MP	32.7	10.35	13	0.80	0.22	0.87	385	36.1
		CC	33.5	11.79	16	0.74	0.24	0.72	390	36.4
		Mean	33.1	11.03	14	0.78	0.23		392	
		±SEM	1.0	0.35	1	0.02	0.01		3	

[a]Average of 18.6 and 28.9 ata values.

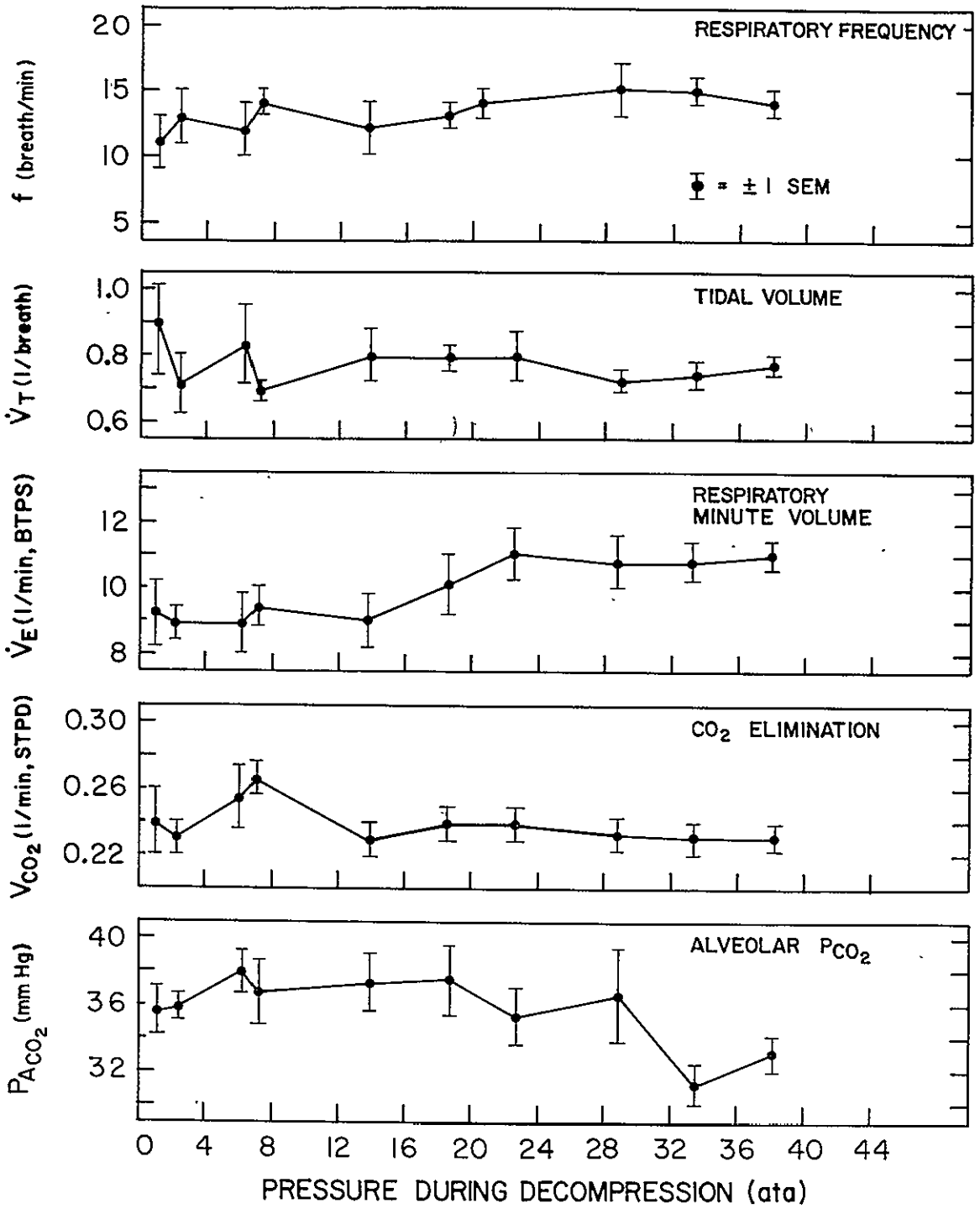

FIG. 1. Morning rest values of $P_{A_{CO_2}}$, $\dot{V}_{CO_2}$, $\dot{V}_E$, V_T and f plotted against hydrostatic pressure. Averages of four subjects of Phase II, ±1 SEM. One ata experiments were in an air-filled chamber, all others in a helium-oxygen filled chamber.

Both V_T and f fluctuated somewhat irregularly and inversely. Average values of $\dot{V}_E$ at the four highest pressures were significantly different ($p \leq 0.05$) from $\dot{V}_E$ at the lowest helium-oxygen pressure (2.3 ata). Respiratory frequency was tested in the same way and the differences were not statistically significant.

Paired values of $\dot{V}_E$ and $P_{A_{CO_2}}$ from Tables I and II are plotted in Fig. 2, along with the hyperbola which represents the locus of points at the constant metabolic rate of the subjects at 1 ata. This hyperbola satisfies the relationship[2] (numerical values are rounded):

$$(\dot{V}_E)\ (P_{A_{CO_2}}) = (9.2)\ (35.6) = 328$$

which forces it through the 1 ata point. When $\dot{V}_{CO_2}$ is in a stable state and if stable-state values of $\dot{V}_E$ and $P_{A_{CO_2}}$ are measured, all $\dot{V}_E$-$P_{A_{CO_2}}$ combinations must fall on this hyperbola (8). Hyperventilation above metabolic need, such as due to excitement, nervousness or other causes, moves points to the left along the hyperbola. Hypoventilation, such as due to increased respiratory resistance or drowsiness, moves points on the hyperbola to the right. $\dot{V}_E$ and $P_{A_{CO_2}}$ measured at increased $\dot{V}_{CO_2}$ would fall on hyperbolas which lie above the hyperbola shown in Fig. 2, while $\dot{V}_E$ and $P_{A_{CO_2}}$ measured at reduced $\dot{V}_{CO_2}$ would produce hyperbolas below the illustrated curve.

Five of the mean morning measurements made at elevated pressures (at 2.3, 6.2, 7.1, 13.9 and 33.4 ata) scatter along the hyperbola which represents $\dot{V}_{CO_2}$ at 1 ata, while the other four lie slightly above it. The measurements made during the compression modules, also shown in Fig. 2, lie in positions consistent with their $\dot{V}_{CO_2}$ values, most of which indicate a higher metabolic rate than the corresponding morning values.

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[2] The basic relationship is: $\dot{V}_{CO_2} = (\dot{V}_A)\ (F_{A_{CO_2}})$ where $\dot{V}_A$ is alveolar ventilation and $F_{A_{CO_2}}$ is alveolar CO_2 concentration (8). This relationship can be converted to: $\dot{V}_{CO_2} = (\dot{V}_E)\ (P_{A_{CO_2}}/P_B) - (\dot{V}_D)\ (P_{A_{CO_2}}/P_B)$ where P_B is barometric pressure and $\dot{V}_D$ is dead space ventilation. With $\dot{V}_D$ much smaller than $\dot{V}_E$, then: $\dot{V}_{CO_2} \simeq (\dot{V}_E)\ (P_{A_{CO_2}})/P_B)$. The product $(\dot{V}_E)\ (P_{A_{CO_2}})$ is a hyperbola which, for a small range of $P_{A_{CO_2}}$, is proportional to $\dot{V}_{CO_2}$.

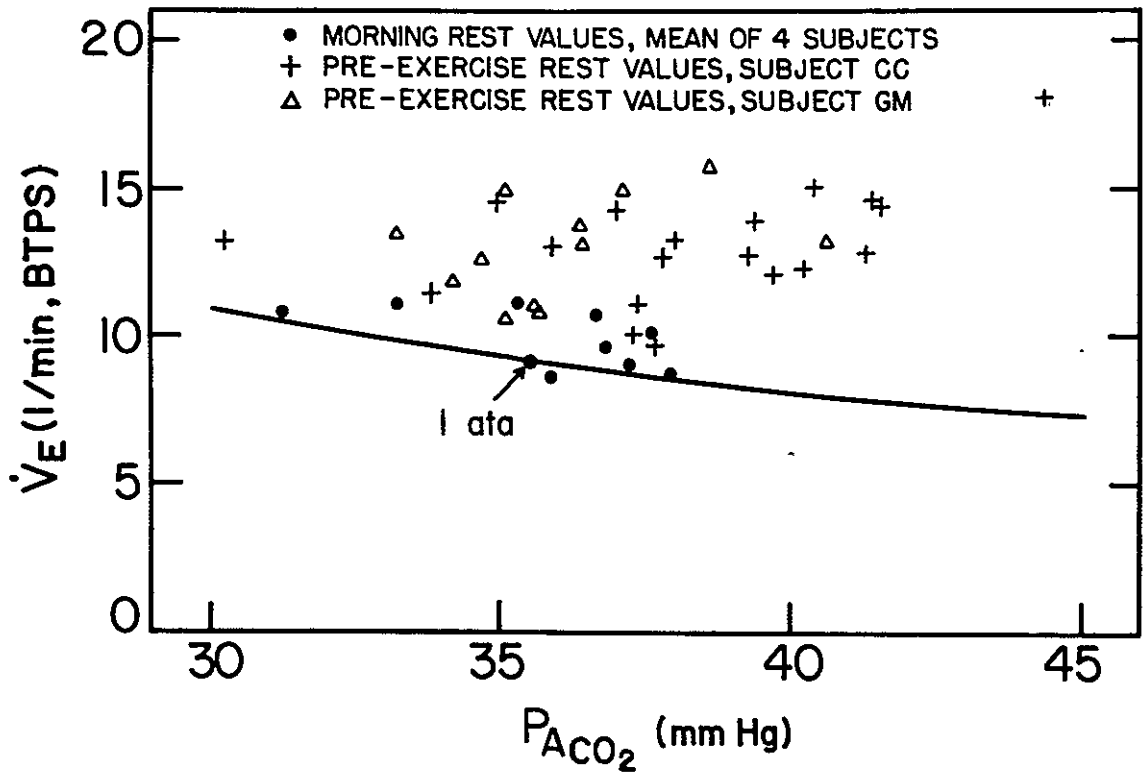

FIG. 2. Morning rest values of $\dot{V}_E$ plotted against $P_{A_{CO_2}}$, averages for the four subjects of Phase II. The data for the two exercise subjects obtained during the rest, pre-exercise phase of compression modules are also shown.

Individual $P_{A_{CO_2}}$ values (Table II) for FS are between 37.5 and 42.6 mm Hg except at 33.4 and 38.0 ata, when they are below 37.5 mm Hg. Subject CC's $P_{A_{CO_2}}$ values are above 35.5 mm Hg at all pressures, except at the two highest pressures. The $P_{A_{CO_2}}$ values of GM are equal to or above 34.4 mm Hg except at the three highest pressures. All of subject MP's $P_{A_{CO_2}}$ values are between 31.1 and 35.2 mm Hg. For all subjects, patterns of change in $P_{A_{CO_2}}$ are usually consistent with patterns of change in ventilation.

Resting ventilation was slightly elevated at the higher pressures in all four subjects. In one subject (FS), respiratory frequency (f) showed an overall increase at higher

pressures, while tidal volume (V_T) declined. In the other three subjects, f increased only slightly or not at all over the pressure range. Correspondingly, V_T showed little change in these three subjects.

DISCUSSION

Possible interactions between CO_2-mediated alterations in the acid-base status of excitable tissues with acute effects of compression and pressure motivated the specific evaluation of $P_{A_{CO_2}}$, $\dot{V}_E$ and $\dot{V}_{CO_2}$ relationships at rest, before exercise. These parameters are considered further in Section E-14, which relates rest to exercise. In contrast to these measurements, the early morning data evaluated here were obtained after the acute effects of compression, pressure and increased inspired gas density had time to stabilize or disappear; this provided the opportunity to examine stable effects of pressure on ventilation in resting man.

Changes in ventilation and gas exchange measured during the slow saturation-decompression over the 38 to 1 ata pressure range were small and not of magnitudes which would cause physiological or performance disruptions. It is usually not practical to attach significance to such small changes and to assign them causative factors. These parameters normally fluctuate from breath to breath (17) and cycle with periods ranging from minutes to hours (6). They are linked to metabolic rates which may themselves fluctuate with time, differ among subjects and depend on experimental circumstances. Metabolism may be more labile in helium-oxygen atmospheres than in air (20) and may vary with acclimatization to pressure and helium-oxygen exposure (18). Other factors which can affect ventilation and gas exchange include drowsiness and sleep (2), body temperature (21), oxygen tension (12) and individual differences. The discussion which follows emphasizes not the occurrence of gross effects but the tendency of resting ventilation to be unaffected even under the conditions of this study.

REST MEASUREMENTS DURING ACUTE COMPRESSION-PRESSURE EXPOSURES

The $P_{A_{CO_2}}$ and $\dot{V}_E$ values obtained in subjects CC and GM at rest, before beginning the pedaling exercise in the compression modules (Fig. 2), show that the subjects were in the normocapnic range while at rest throughout compression. Most of the $\dot{V}_E$ and $\dot{V}_{CO_2}$ values were elevated compared with the morning values (Tables I, II) but this is expected since, subsequent to the early morning measurements, the subjects had had food and had actively prepared for conducting experiments. In the absence of hypercapnia or hypocapnia, and with apparently normal cardiovascular function (Section E-13), CO_2-related alterations of cellular acid-base levels could not have contributed to or exacerbated the observed symptoms of rapid compression to high pressure (Section E-1). The small transient increase in $P_{A_{CO_2}}$ during compression (Table I) may have been the result of bulk movement of chamber gas into the airways and lungs, and consequent decrease in outward alveolar ventilation during rapid compression. These small and transitory changes, while of theoretical interest, were functionally unimportant.

EARLY MORNING RESTING MEASUREMENTS

Figures 1 and 2 show that sustained, multi-day exposure to helium-oxygen at hydrostatic pressures up to 38.0 ata did not result in CO_2 retention. The highest $P_{A_{CO_2}}$ observed in any single subject (FS) was 42.6 mm Hg at 18.6 ata (Table II). The mean $P_{A_{CO_2}}$ of 35 to 38 mm Hg over the pressure range of 1 to 28.9 ata is entirely similar to that observed in 33 normal subjects studied at 1 ata. (9). The associated mean $\dot{V}_E$ values in the range of 8.9-11.0 1/min are also similar to those of the 33 normal subjects (9). Comparable values have been found in a helium-oxygen exposure at 31.3.ata (15).

COMPARISON WITH OTHER EXPERIMENTS

Ventilation and pulmonary gas tensions have not previously been measured during acute exposure to compression

and pressure. Therefore, only the results of the measurements for rest at stable pressures can be compared with other studies.

Alveolar and Arterial P_{CO_2}

Previous measurements of $P_{a_{CO_2}}$ or $P_{A_{CO_2}}$ at rest in helium-oxygen environments have produced contradictory results. Elevated P_{CO_2} levels (1, 19), reduced levels (14), and elevation at lower densities with return toward 1 ata control levels at high densities (5) have been observed. The morning measurements in this predictive study show no change in $P_{A_{CO_2}}$ from the 1 ata level at pressures up to 28.9 ata and only slightly reduced $P_{A_{CO_2}}$ at 33.4 and 38.0 ata.

CO_2 Production

In several studies $\dot{V}_{CO_2}$ has been reported to increase in helium-oxygen environments, as compared with 1 ata controls in air (1, 14, 19). While these observations may be relevant to the peculiar thermal-metabolic state resulting from immersion in helium-oxygen atmospheres (20) (Section E-15), extremely close control of activity levels and diet both at 1 ata and during the pressure exposures is required to establish that a real difference related to helium exposure exists. The averaged results show that most measurements of $\dot{V}_{CO_2}$ during the pressure exposure are within 0.03 l/min of the 1 ata value (Fig. 1, Table II). If the 1 ata value had been only 0.01 l/min lower than observed, all the measurements at elevated pressures would have been higher than at 1 ata.

Minute Ventilation

Although the overall increase in mean $\dot{V}_E$ over the pressure range from 1 to 38 ata is small, it occurs despite a progressively increasing gas density which should cause $\dot{V}_E$ to change in the opposite direction. Elevated deep body temperature or reduced inspired oxygen tension cannot be responsible for the $\dot{V}_E$ change since these parameters did not change systematically with pressure (Table II).

Small increments in resting ventilation above the 1 ata control level have also been reported in several other helium-oxygen exposures (14, 19). While such changes may actually be due to incidental factors (14), the results of the present study, which involved measurements in four subjects at ten different pressures during the least stressful part of the pressure exposures, may possibly have resulted from a mild excitatory effect of hydrostatic pressure on the complex neural networks which establish the rate and depth of breathing.

An entirely different association between respiratory control and hydrostatic pressure has been suggested by others. From the results of acute exposures to helium-oxygen, neon-oxygen and nitrogen-oxygen in a helium-oxygen filled chamber at depths to 250 fsw, it was concluded that pressure per se causes CO_2 retention (19). However, that effort to separate effects of hydrostatic pressure from effects of increased gas density was based on statistical inference with data over only a small range of pressures (19). Furthermore, there is evidence that elevated nitrogen pressure can have separate effects on ventilation: at the same time that its increased density reduces ventilation, other influences proportional to its partial pressure lessen the degree of the density effect (4, 5). This influence may be no more than narcosis, releasing inhibitions on the respiratory function, as seems to occur with nitrous oxide at sea level (5). If hydrostatic pressure actually produces a mild excitation of respiratory neurons in resting man, the ability to maintain normal CO_2 levels during acute exposure to extremely high density atmospheres at very high pressures (5) may be facilitated.

CONCLUSIONS

Measurements of ventilation and respiratory gas exchange at rest during acute stages of rapid compression to high pressure showed that neither hypercapnia nor hypocapnia resulted from these exposures. There were therefore no important CO_2-related alterations in the acid-base status of excitable tissues to interact with compression-pressure effects on those tissues.

Measurements at rest in the morning soon after awakening over a pressure range from 38 to 1 ata showed that exposure to high hydrostatic pressure may cause a mild but tolerable stimulation of ventilation.

Acknowledgment

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E-17. BIOCHEMICAL, ENDOCRINOLOGICAL AND HEMATOLOGICAL STUDIES

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Previous studies have shown that prolonged exposure of men to increased ambient pressures produces changes in specific endocrine and biochemical parameters (2,10,15). To obtain objective and quantitative evaluations of the physiological stress incurred on "saturation-excursion" exposures to helium at pressures equivalent to 400-800-1200 and 1600 fsw, biochemical measurements were selected to assess the responses to compression as well as the adaptive changes which result from prolonged saturation at a stable increased pressure. This report contains results obtained in the two separate phases of the study, the one involving compression to a maximum of 1200 fsw (Phase I), and the other to a maximum of 1600 fsw (Phase II).

METHODS

Six subjects (CC, LJ, GM, MP, FS, WS) participated in Phases I and II of the study, with two subjects (CC and FS) common to both phases.

Twenty milliliters of venous blood were drawn from each subject three times prior to compression for baseline values

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(pre-exposure days 5, 6 and 7, or 6, 7 and 8 for Phase I; pre-exposure days 1, 11 and 13, or 2, 7 and 9 for Phase II), and three or four times after exposure (days 0, 1, 2 and 14 for Phase I; days 0, 2 and 8 for Phase II). The blood samples drawn on Phase I post-exposure day 14 were also used as the first Phase II pre-exposure samples in subjects CC and FS who participated in both exposures. In addition, one blood sample was drawn from each subject on exposure day 3 in Phase II after decompression to 1200 fsw from the second excursion to 1600 fsw. All samples were drawn from fasting subjects between 7 and 8 A.M. except those drawn at pressure and immediately upon emerging from the chamber.

The biochemical-endocrine measurements on blood plasma or serum included cortisol, aldosterone, angiotensin I, growth hormone, insulin, thyroxine (T_4), dopamine-β-hydroxylase (DβH), adrenocorticotropic hormone (ACTH), serum glutamic oxalic transaminase (SGOT), calcium, magnesium, creatine phosphokinase (CPK), lactic acid dehydrogenase (total LDH), osmolality, sodium, potassium, chloride, creatinine and triglycerides.

In addition to the biochemical-endocrine analyses, which were performed at the Lyndon B. Johnson Space Center, hematological measurements performed at the Hospital of the University of Pennsylvania included hemoglobin and hematocrit, counts of erythrocytes, reticulocytes, leucocytes and platelets, and calculations of mean corpuscular volume, hemoglobin and hemoglobin content.

Twenty-four hour urine samples were collected from each subject beginning on the first blood collection day, throughout the exposure, and post-compression for either three or seven days. The urine was analyzed for these biochemical-endocrine parameters: cortisol, aldosterone, antidiuretic hormone (ADH), epinephrine, norepinephrine, specific gravity, osmolality, sodium, potassium, chloride, calcium, magnesium, inorganic phosphate, uric acid and creatinine. Methods for these analyses have been described elsewhere (2,10). The subjects were given a general diet during the entire study; fluids were available ad libitum.

Analysis of variance for repeated measures (20) was performed on data from both phases separately. Data on four subjects were averaged for all days when measurements were

available; in each phase these means were compared with the 1 ata control mean for that phase. Differences were considered significant if $p \leq 0.05$.

RESULTS

Average data obtained in both phases and the results of statistical testing are summarized in Tables I-V. Individual values are provided in the Appendix tables. Since the experiment design allowed for full tracking of initial compression on each subject, exposures were started on different days for each subject pair; data were averaged with reference to the days representing start and end of the subject exposure rather than the calendar day of collection. Averages were not calculated for the days on which the exposure duration of one subject pair differed from that of the other.

CIRCULATING ENDOCRINE FACTORS

Plasma endocrine data are summarized in Table I. Plasma cortisol concentrations showed a consistent pattern of initial decline followed by progressive elevation throughout both post-exposure periods. The 11.6 µg/dl average value upon "surfacing" from the Phase I exposure was slightly below the control level and the value obtained on post-exposure day 15 was higher than the control. In Phase II the value obtained on exposure day 3 was decreased significantly and the value on post-exposure day 9 had returned nearly to the control level.

Aldosterone and angiotensin showed a similar pattern of reduction or little change during and immediately after the pressure exposure, followed by progressive elevation over the post-exposure period.

Thyroxine levels were significantly increased during the post-Phase I exposure period and upon surfacing from the Phase II exposure.

Average levels of growth hormone were generally similar to the control values except for a significant elevation immediately after the Phase I exposure.

There were no significant or consistent patterns of change in the plasma levels of insulin, dopamine-beta-hydroxylase or adrenocorticotropic hormone.

PLASMA ELECTROLYTES AND ENZYMES

Table II contains average values of serum osmolality, electrolytes and enzymes. Serum osmolality was significantly increased throughout the Phase I post-exposure period. It decreased on Phase II exposure day 3, was at or above the control level on post-exposure days 1 and 3, and was elevated on post-exposure day 9.

Changes in serum sodium concentration were generally parallel to those of serum osmolality.

Serum chloride concentration was significantly increased during the Phase I post-exposure period and was decreased on exposure day 3 of Phase II.

Changes in serum potassium, calcium, magnesium and creatinine were generally small in magnitude and not statistically significant.

Average serum levels of LDH, CPK, and SGOT increased after the Phase I exposure and the LDH elevation was statistically significant on post-exposure day 15. Serum LDH also increased significantly immediately after the Phase II exposure, while the mean levels of CPK and SGOT were less than their respective control values throughout the post-exposure period.

Changes in serum triglyceride concentrations appeared to have no consistent pattern. Elevations found immediately after the Phase I exposure and on exposure day 3 of Phase II were probably caused by the fact that the subjects were not fasting at these times.

URINE VOLUME AND ENDOCRINE FACTORS

Average daily, 24-hour urine volumes and corresponding endocrine data are summarized in Table III. With the exception of exposure day 2 of Phase I, average urine volumes

were consistently elevated throughout both exposure periods. Volumes were also increased during at least the early parts of both decompression periods. Average values of cortisol output were elevated throughout both exposure periods. Aldosterone excretion was also elevated during both exposures and during the post-exposure period of Phase I. As compared with a pre-exposure control value which was above the normal range, urine ADH output in Phase I decreased from the second exposure day throughout the decompression and post-exposure periods.

In Phase II the pre-exposure control value of ADH was in the upper range of normal. Subsequent average values scattered randomly about the control value during the saturation exposure but were consistently reduced during the decompression and post-exposure periods. There were no consistent changes in urine epinephrine outputs, while the norepinephrine outputs were elevated following the Phase I exposure and during the Phase II exposure.

URINE ELECTROLYTES

Average values of urine osmolality and electrolyte outputs are summarized in Table IV. With the exception of exposure day 2 of Phase I, average values of urine osmolality consistently decreased throughout both exposure periods and during all (Phase I) or part (Phase II) of the decompression period.

Average 24-hour outputs of potassium, uric acid and creatinine increased during the Phase I exposure, while the outputs of sodium, chloride, calcium and magnesium decreased. Sodium, chloride and calcium outputs also decreased after the exposure.

During the Phase II exposure, average potassium output increased, while sodium, chloride, calcium and magnesium outputs all decreased.

Post-exposure there were average decrements in the outputs of sodium, potassium, chloride, magnesium, inorganic phosphate and uric acid. Most of these deviations from average control values were small in magnitude and only a few were statistically significant.

TABLE I. Effects on Plasma Endocrine Parameters of Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw (Mean Values in Four Subjects in Each Phase)

MEASUREMENT		PHASE I[a]					PHASE II[a]				
		PRE-EXP CONTROL	POST-EXPOSURE				PRE-EXP CONTROL	EXPOSURE	POST-EXPOSURE		
			7/15[b] Day 1	7/16 Day 2	7/17 Day 3	7/29 Day 15		Day 3 at 1200 fsw	8/27[b] Day 1	8/29 Day 3	9/4 Day 9
Cortisol (μg/dl)	Mean	13.4	11.6	16.4	15.4	21.6	19.4	7.0*	12.7	15.8	18.9
	±SEM	1.8	2.8	2.5	3.3	3.6	2.3	2.4	2.0	2.7	3.7
Aldosterone (pg/dl)	Mean	180	111	184	208	247	210	113	149	184	249
	±SEM	34	27	24	32	61	45	13	26	17	59
Angiotensin I (ng/ml/hr)	Mean	0.21	0.24	0.61	0.88	1.52*	1.25	0.99	0.96	1.71	1.46
	±SEM	0.03	0.06	0.20	0.23	0.44	0.23	0.12	0.31	0.16	0.50
Growth Hormone (ng/ml)	Mean	6.0	8.4	6.2	4.6	5.2	4.0	4.0	3.6	3.6	5.2
	±SEM	0.6	0	0.4	0.4	1.2	1.1	1.1	0.8	0.6	1.2
Insulin (μU/ml)	Mean	18	(21)	24	18	14	18	(30)	21	(17)	25
	±SEM	2	2	6	1	2	1	4	7	2	6
Thyroxine (T_4) (μg/dl)	Mean	7.2	9.0	9.6*	9.6*	9.9*	7.2	(7.0)	10.0*	9.1	8.5
	±SEM	0.1	0.9	0.3	0.5	0.8	0.8	0.5	1.3	1.5	0.5
DβH (IU)	Mean	63.4	62.8	63.0	57.0	56.0	43.1	42.5	48.4	42.3	45.0
	±SEM	7.8	10.0	7.4	6.2	2.8	6.2	5.9	6.7	5.9	8.4
ACTH (pg/ml)	Mean	9.8	43.3	13.2	(13.3)	37.9	8.8	-	10.0	7.7	(18.3)
	±SEM	2.5	34.2	2.4	2.2	26.9	1.0		4.0	2.9	8.9

[a] Phase I subjects: FS, CC, LJ, WS; Phase II subjects: FS, CC, MP, GM.

[b] Reach surface.

* Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects; not used in statistics.

TABLE II. Effects on Serum Biochemical Parameters of Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw (Mean Values in Four Subjects in Each Phase)

MEASUREMENT		PHASE I[a]					PHASE II[a]				
		PRE-EXP CONTROL	POST-EXPOSURE				PRE-EXP CONTROL	EXPOSURE	POST-EXPOSURE		
			7/15[b] Day 1	7/16 Day 2	7/17 Day 3	7/29 Day 15		Day 3 at 1200 fsw	8/27[b] Day 1	8/29 Day 3	9/4 Day 9
Osmolality (mOsmo)	Mean	293	303*	310*	307*	304*	308	297	313	308	322
	±SEM	4	2	3	2	3	2	3	2	4	6
Sodium (mEq/l)	Mean	145	146	150*	151*	148	151	145	152	149	156
	±SEM	1	1	1	1	1	1	0.5	2	1	3
Potassium (mEq/l)	Mean	4.0	3.8	4.0	4.2	4.2	4.4	4.0	4.0	4.3	4.4
	±SEM	0.05	0.2	0.05	0.2	0.2	0.1	0.2	0.1	0.2	0.1
Chloride (mEq/l)	Mean	102	102	106*	108*	106*	109	99*	108	108	112
	±SEM	2	1	1	1	1	1	1	1	2	2
Creatinine (mg/dl)	Mean	1.4	1.5	1.5	1.5	1.6	1.6	1.5	1.6	1.5	1.6
	±SEM	0.2	0.2	0.2	0.2	0.2	0.1	0.1	0.2	0.2	0.2
SGOT (mU/ml)	Mean	11	-	16	16	15	16	15	15	15	15
	±SEM	1		4	4	3	3	2	1	1	2
Calcium (mg/dl)	Mean	9.4	10.0	10.0	9.9	9.7	10.0	10.3	10.2	10.1	10.3
	±SEM	0.1	0.2	0.2	0.3	0.4	0.1	0.1	0.1	0.05	0.1
Magnesium (mg/dl)	Mean	2.0	2.2	2.1	2.2	2.2	2.3	2.4	2.3	2.1*	2.4
	±SEM	0.05	0.1	0.1	0.05	0.1	0.05	0.03	0.03	0.05	0.05
CPK (mU/ml)	Mean	48	57	74	76	70	117	166	76	74	75
	±SEM	11	32	34	12	29	32	44	15	11	14
LDH (mU/ml)	Mean	129	137	167	159	205*	226	227	260*	238	254
	±SEM	18	12	12	24	20	30	23	32	20	29
Triglycerides (mg/dl)	Mean	177	293*	168	221	140	128	172	101	106	159
	±SEM	36	17	19	48	14	21	66	11	12	42

[a]Phase I subjects: FS, CC, LJ, WS; Phase II subjects: FS, CC, MP, GM.

[b]Reach surface.

*Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects; not used in statistics.

TABLE IIIA. Effects on Urine Endocrine Parameters of Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw (Mean Values on Four Subjects in Each Phase) (Phase I)

MEASUREMENT		PRE-EXP CONTROL	EXPOSURE						DECOMPRESSION							POST-EXPOSURE			
			Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14	Day 1[c] 7/15	Day 2 7/16	Day 3 7/17	Day 15 7/29
Cortisol	Mean	47.0	126 2	88.4	74.7	50.3	128.4	71.6	43.1	62.5	91.5	59.3	61.8	49.8	55.1	31.5	(24.6)	31.3	35.8
(μg/TV)	±SEM	7.7	34.7	38.3	9.8	11.2	71.9	25 2	13.6	25.6	25.4	17.2	16.4	16 1	14.1	5.9	5.2	6.0	3.6
Aldosterone	Mean	8.3	14.0	16 9	10.1	7.8	8.7	12.1	10 0	9.4	11.6	9.8	7.8	5 8	6.2	(8.0)	15.8	16.9	9.3
(μg/TV)	±SEM	1.0	1.6	5.3	2.9	1.2	2.5	2.2	2.0	1.5	2.7	2.9	1.7	1.9	1.9	2.2	4.1	3.2	2.2
ADH	Mean	55.7	66.1	41.3	39.0	35.8	34.8	27 3*	21.3*	32.6	25.5*	22.4*	26.4*	18.5*	30.4*	35 4	34.8	29.5*	28.2*
(mU/TV)	±SEM	14.9	14.7	1.8	9.8	8.5	14.2	5.2	3.0	7.0	7.0	3.1	2.7	3.2	4.7	12.3	4.7	14.6	9.2
Epinephrine	Mean	32.4	(61.6)	(20.1)	-	(14.0)	19.0	13.6	10.9	(16.1)	(7.9)	(13.1)	(5.7)	-	(2.8)	9.2	(5.7)	-	(12.4)
(μg/TV)	±SEM	15.1	35.3	12.3		2.3	9.2	5.2	4.7	3.1	3.7	3.3	2.9		1.8	2.1	1.9		6.9
Norepinephrine	Mean	42.1	(32.6)	60.1	-	40.8	(58.0)	46.0	36.3	35.9	31.8	32.7	21.4	29.0	29.5	37.0	59.8	95.1	42.4
(μg/TV)	±SEM	7 0	12.3	14.1		10.4	23.6	18.0	6.6	14.0	3.3	5.9	6.9	7.9	5.2	3.8	11 9	39.1	15.1
Volume	Mean	1459	1902	986	2375*	1525	1750	1562	1515	1798	1730	1908	1465	1375	1368	1308	1750	880	1178
(ml/24 hr)	±SEM	62	311	246	520	282	428	316	298	388	385	270	284	168	176	174	463	208	266
Fluid Intake	Mean	1887	1510	1613	2161	1730	1939	1428	1972	2090	1436	2190	1565	1434	1684	1554	-	-	(2045)
(ml/24 hr)	±SEM	436	176	311	178	260	638	161	350	371	234	524	54	318	240	175			242

[a] Exposure day 1 was 7/1 for subjects WS and FS, 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

* Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects; not used in statistics.

TABLE IIIB. Effects on Urine Endocrine Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 faw (Mean Values in Four Subjects in Each Phase) (Phase II)

MEASUREMENT		PRE-EXP CONTROL	EXPOSURE						DECOMPRESSION										POST-EXPOSURE							
			Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26	Day 1[c] 8/27	Day 2 8/28	Day 3 8/29	Day 4 8/30	Day 5 8/31	Day 6 9/1	Day 7 9/2	Day 8 9/3
Cortisol (μg/TV)	Mean	51 1	65.4	53.8	76 3	90.4	66 6	68 5	55.7	78 4	(48.1)	51 7	75 5	(52.0)	53 1	29.4	60.8	47.7	(88.6)	28.0	-	(57.5)	49 6	31.2	58.5	30.0
	±SEM	10.0	17.8	11.3	21.1	30 9	24.6	18.7	13 7	29.4	28 7	20.4	33 3	21.5	18.5	7 8	27.4	11 3	54.9	10 9		22.9	16.8	9 2	27 5	8.4
Aldosterone (μg/TV)	Mean	9.0	5 6	7 1	20.8*	19.1*	8 2	12.8	6.8	8.9	(9 1)	9.4	7.6	(10 9)	9.0	5.8	7 4	5.0	(7 8)	3.8	-	(7 8)	3.8	3.7	5.0	3.6
	±SEM	0.8	1 1	1.1	6.3	2 6	1 7	3.4	2 1	1.7	0 9	2.9	2 8	1.9	4.0	2.2	2.6	1.4	4 6	1.1		2 4	1.6	0 9	1 1	0.8
ADH (mU/TV)	Mean	29.4	36.6	22.8	39.7	33.9	20 8	24.6	32 2	44.8	(20 0)	22 6	21.9	(21.2)	26 0	15.0	14 6	18.9	(19 7)	16.2	-	(16.5)	19 3	25.7	16.6	17 6
	±SEM	3.9	9 9	2.4	11.3	9 5	2 4	3 1	12 2	20.6	6 6	4.9	4 7	3.8	4.1	3 8	5.3	4 9	9.1	4 7		1 5	4.1	4.0	1 8	2.5
Epinephrine (μg/TV)	Mean	13 5	-	-	-	-	-	(11.3)	-	-	-	-	-	-	-	-	-	-	-	(15.5)	-	-	-	-	-	-
	±SEM	2.5						5.4												10 8						
Norepinephrine (μg/TV)	Mean	43.9	42 2	49.6	104 0	77.5	45 2	(33 3)	(29.6)	(15 7)	-	-	-	-	-	-	-	-	-	49.6	-	(37.9)	-	-	-	-
	±SEM	9.9	13 0	14.3	38 6	7.6	10.3	3.4	15.1	6.8										23.4		11.2				
Volume (ml/24 hr)	Mean	1224	1390	1630	1465	1482	1365	2282*	1312	2002	(1370)	1235	2114*	(1070)	1370	1020	1545	1432	(2527)	868	-	(1090)	1197	1302	1262	1258
	±SEM	200	220	158	97	168	461	188	213	467	448	167	350	299	228	73	388	155	693	182		368	363	271	262	292
Fluid Intake (ml/24 hr)	Mean	2330	1076	1742	1624	1256	1243	1354	1700	1964	1531	1648	1602	1404	1516	1230	1534	1497	-	-	(2635)	-	-	-	-	-
	±SEM	212	95	302	186	178	229	196	208	392	50	45	349	86	92	155	243	338			227					

[a] Exposure day 1 was 8/7 for subjects GM and FS, 8/11 for subjects MP and CC

[b] Start of decompression for all subjects.

[c] Reach surface

* Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects, not used in statistics.

TABLE IVA. Effects on Urine Biochemical Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Mean Values in Four Subjects in Each Phase) (Phase I)

MEASUREMENT		PRE-EXP CONTROL	EXPOSURE						DECOMPRESSION							POST-EXPOSURE			
			Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14	Day 1[c] 7/15	Day 2 7/16	Day 3 7/17	Day 15 7/29
Osmolality (mOsmo)	Mean	695	569	853	468	591	496	658	518	636	527	523	608	602	648	666	428*	738	804
	±SEM	57	33	81	74	64	40	101	48	100	53	37	85	50	26	29	56	94	83
Sodium (mEq/TV)	Mean	225	166	90*	143	162	174	161	124*	223	148	188	192	178	194	154	109*	64*	162
	±SEM	22	32	4	27	24	46	34	221	45	30	30	24	28	32	16	47	8	56
Potassium (mEq/TV)	Mean	78	113	93	117*	83	82	109	68	96	66	67	68	52	61	81	65	72	75
	±SEM	6	16	18	22	13	22	15	12	18	13	7	10	6	7	8	17	10	13
Chloride (mEq/TV)	Mean	209	189	108	138	149	181	160	94*	195	140	165	168	142	157	145	105*	58*	157
	±SEM	18	36	4	21	29	52	31	19	41	29	26	25	24	24	17	44	12	54
Calcium (mEq/TV)	Mean	15.1	15.3	10.7	14.4	12.0	11.1	8.1*	9.0*	12.6	15.0	14.3	12.4	11.8	14.8	12 2	11.3	9.4*	14.4
	±SEM	1.6	3.2	1.9	1.9	2.5	3.1	1.1	2.0	1.6	3.0	2.1	0.8	2.9	2.4	2.5	3.0	1.8	3.3
Magnesium (mEq/TV)	Mean	8.6	7.7	7.4	10.4	10.3	7.2	5.6	8.0	7.4	12.2	9.2	7.0	9.9	9.7	7.9	8.4	9 1	5.0
	±SEM	0.5	1.0	1.0	0.8	1.9	2.4	0.5	1.0	2.0	3.0	2.3	1.4	2.2	1.6	2.2	1.1	3 0	1.8
IPO4 (mg/TV)	Mean	1075	1188	1140	1336	1051	813	974	1001	1027	1026	1008	932	1263	1017	962	1040	1131	1376
	±SEM	121	260	87	123	177	215	133	105	70	245	157	88	270	156	189	236	341	200
Uric Acid (mg/TV)	Mean	764	809	1030	842	706	656	825	636	832	774	751	705	675	677	658	738	794	916
	±SEM	81	78	276	56	127	173	161	89	101	171	105	79	159	87	101	153	108	187
Creatinine (mg/TV)	Mean	1701	1942	1838	2050	1576	1588	1975	1738	1736	1689	1761	1609	1683	1631	1537	1693	1648	1942
	±SEM	125	257	222	176	269	413	372	174	160	366	232	144	294	250	278	394	284	252

[a] Exposure day 1 was 7/1 for subjects WS and FS, 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

* Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects; not used in statistics.

TABLE IVB. Effects on Urine Biochemical Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Mean Values in Four Subjects in Each Phase) (Phase II)

MEASUREMENT		PRE-EXP CONTROL	EXPOSURE						DECOMPRESSION										POST-EXPOSURE							
			Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26	Day 1[c] 8/27	Day 2 8/28	Day 3 8/29	Day 4 8/30	Day 5 8/31	Day 6 9/1	Day 7 9/2	Day 8 9/3
Osmolality (mOsmo)	Mean	779	477*	586	655	699	630	573	680	696	(744)	839	606	(798)	898	896	806	668	(575)	920	-	(820)	742	671	630	718
	±SEM	89	55	37	30	32	65	21	85	122	173	62	54	139	14	86	80	54	199	116		249	124	64	91	94
Sodium (mEq/TV)	Mean	176	126	136	143	144	143	194	147	201	(152)	108	180	(92)	181	148	200	197	(221)	100	-	(121)	163	180	153	139
	±SEM	17	40	29	29	38	27	9	32	43	54	27	34	16	28	21	26	31	15	18		22	20	26	32	32
Potassium (mEq/TV)	Mean	76	76	96	112	120	84	114	70	90	(69)	66	108	(85)	92	71	81	76	(105)	44	-	(63)	50	52	57	52
	±SEM	12	23	18	10	3	16	8	11	12	14	12	12	12	10	4	10	12	27	9		15	14	10	11	12
Chloride (mEq/TV)	Mean	171	125	154	162	134	128	181	166	188	(134)	128	191	(95)	148	129	177	178	(197)	66	-	(108)	152	172	171	127
	±SEM	10	43	30	37	37	23	8	55	42	50	21	28	20	26	15	31	22	23	9		24	33	25	28	38
Calcium (mEq/TV)	Mean	12.2	7.4	11 9	10 7	11.0	7.7	14.2	10.4	13.2	(10 6)	12.3	13.7	(7 6)	15.2	12 5	12.2	9.0	(12 3)	9.1	-	(7 4)	12 7	9.8	7.8	12.0
	±SEM	1 6	1.2	2.3	1 3	2 5	1.6	1.6	2.5	2.0	2 4	1.9	1.2	0.6	6.9	3.2	2 3	1 1	1.0	2.0		1.0	2.6	1 3	1 3	3 6
Magnesium (mEq/TV)	Mean	9 7	3.8	5.6	9 7	9.8	4.0	10 9	7.9	6.9	(8 5)	10.0	6.7	(6 7)	7.0	4 9	6 9	4.0	(6 0)	5 9	-	(5 6)	3 0	3.7	1.7	7.5
	±SEM	0 8	1 7	1.7	1.0	2 0	1 3	3.8	3 1	2.4	0.06	3 7	2.4	0.4	2 6	1.8	2 2	1 4	3.2	2 6		3.2	1 9	1 6	1 4	4 9
IPO4 (mg/TV)	Mean	1144	788	929	1260	1686	1084	1458	1127	1410	(1005)	1248	1151	(1143)	1291	1000	1264	1168	(1037)	1086	-	(1149)	744	816	525	876
	±SEM	58	256	125	216	84	255	245	229	127	47	144	120	21	62	109	78	216	318	166		137	157	146	40	147
Uric Acid (mg/TV)	Mean	986	735	564	984	1023	910	1078	939	1347	(907)	1716	1278	(1093)	1152	711	949	712	(933)	900	-	(1118)	637	762	729	770
	±SEM	25	281	122	88	62	270	98	170	293	139	388	242	241	231	31	161	94	274	164		348	63	167	48	82
Creatinine (mg/TV)	Mean	1869	1549	1655	2307	2264	1613	2426	1745	2172	(1843)	2209	2280	(1866)	2307	1854	2080	1906	(2612)	1860	-	(1984)	1773	1916	1416	1716
	±SEM	145	286	254	120	118	344	152	280	73	279	228	130	155	209	36	87	300	738	256		239	265	285	139	140

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC

[b] Start of decompression for all subjects.

[c] Reach surface.

* Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects, not used in statistics.

TABLE V. Effects on Hematological Parameters of Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw (Mean Values in Four Subjects in Each Phase)

MEASUREMENT		PHASE I[a]					PHASE II[a]				
		PRE-EXP CONTROL	POST-EXPOSURE				PRE-EXP CONTROL	EXPOSURE	POST-EXPOSURE		
			7/15[b] Day 1	7/16 Day 2	7/17 Day 3	7/29 Day 15		Day 3 at 1200 fsw	8/27[b] Day 1	8/29 Day 3	9/4 Day 9
Mean Corpuscular Volume (cu micron)	Mean	88.1	89.8	90.5*	89.5	84.0*	90.0	-	84.0*	85.2*	-
	±SEM	1.9	1.7	2.3	1.8	1.7	1.7		1.8	1.9	
Mean Corpuscular Hemoglobin (gamma gamma)	Mean	29.4	29.2	29.2	29.8	29.5	29.0	-	27.2*	28.2*	-
	±SEM	0.6	0.5	0.8	0.8	0.9	0.6		0.5	0.5	
Mean Corpuscular Hb Content (%)	Mean	32.9	33.5	33.5	32.1	34.8*	32.5	-	32.0	32.5	-
	±SEM	0.3	0.3	0.3	0.4	0.3	0.3		0.4	0.3	
Hematocrit (%)	Mean	44.1	46.5	45.8	44.0	42.8	47.2	-	44.8	44.8	42.5*
	±SEM	1.3	1.2	1.5	0.7	1.9	2.2		1.6	1.7	1.7
Hemoglobin (g/100 ml)	Mean	14.7	15.2	15.1	14.4	14.6	15.1	-	14.8	14.9	14.6
	±SEM	0.4	0.5	0.6	0.3	0.7	0.8		0.7	0.5	0.6
Erythrocytes (x 10^6/cu mm)	Mean	5.0	5.2	5.2	4.8	4.8	5.2	-	(5.4)	-	-
	±SEM	0.05	0.2	0.2	0.1	0.1	0.2		0.2		
Reticulocytes (%)	Mean	2.0	1.2	0.4*	0.6	2.3	2.2	3.4	2.2	1.6	2.6
	±SEM	0.3	0.3	0.1	0.2	0.8	0.9	0.5	0.6	0.6	0.6
Platelets (x 10^3/cu mm)	Mean	267	210*	196*	231	206*	196	244	254	250	324*
	±SEM	30	24	25	31	18	17	30	29	17	18
Leucocytes (x 10^3/cu mm)	Mean	5.8	8.3*	7.5*	7.0	5.7	6.5	-	9.2*	5.9	6.8
	±SEM	0.4	0.6	0.8	0.5	0.5	0.9		0.8	0.7	1.6

[a]Phase I subjects: FS, CC, LJ, WS; Phase II subjects: FS, CC, MP, GM.

[b]Reach surface.

*Mean value indicated by asterisk significantly different from its respective pre-exposure control at $p \leq 0.05$.

( ) Indicates data on only three subjects; not used in statistics.

BLOOD CELLULAR COMPOSITION

Table V shows average values of the measured hematological parameters. Mean corpuscular volume increased slightly but significantly on post-exposure day 2 of Phase I and decreased significantly on post-exposure day 15; it also decreased following the Phase II exposure. Mean corpuscular hemoglobin was not changed after Phase I, but it decreased significantly after Phase II. Hemoglobin and hematocrit also decreased following Phase II. Average platelet levels significantly decreased after the Phase I exposure but increased during and after the Phase II exposure. Leucocyte counts increased significantly during the early part of both post-exposure periods.

DISCUSSION

Surveys of hormonal and biochemical effects of exposure to mild or even severe environmental stresses are generally difficult to interpret because many of the observed responses are nonspecific and can be induced by any one or a combination of a variety of conditions. Interpretation of results is also complicated by individual variability of response to any given stress or combination of stresses.

In addition to effects of compression, prolonged exposure to increased hydrostatic pressure, and decompression, the stresses of an experimental hyperbaric exposure include factors such as the potential of serious accident, living in cramped and crowded quarters, thermal discomfort, exercise and long hours of tedious work. Some of the subjects in the two phases of Predictive Studies IV also experienced decompression sickness and one had a transient bacterial skin infection. Since it was impractical to isolate responses to a specific stress by appropriate timing of blood and urine samples, the observed changes should be regarded as indicating the degree of responses to the composite of many stresses.

Because of the impracticality of separating specific forms of stress in a hyperbaric exposure, the existence of individual variability and the usual logistic requirement to limit study to relatively small numbers of subjects, the importance of a hormonal or biochemical change that is

found repeatedly in different hyperbaric exposures is obviously greater than that of a comparable change in any single exposure.

In Table VI the results obtained in both phases of Predictive Studies IV are compared with similar data from a previous $He-O_2$ saturation exposure at 1200 fsw (Predictive Studies III) (1,9), a saturation N_2-O_2 exposure at 100 fsw (Predictive Studies II) (2,10), and a non-saturation N_2-O_2 exposure at 200 fsw (also Predictive Studies III) (1,9). Although the hormonal and biochemical profiles were not identical for these exposures, a large number of measurements were common to all five surveys and all experiments and analyses were done by the same environmental and analytical laboratories. The related findings of the several major studies will be considered together in the discussion which follows.

HORMONAL RESPONSES TO INCREASED AMBIENT PRESSURE

Urine endocrine data from both phases of Predictive Studies IV are generally consistent with nonspecific stress responses. Total 24-hour outputs of cortisol were significantly elevated during both exposures (Tables III and VI). Norepinephrine outputs were elevated intermittently throughout the exposure and post-exposure periods of Phases I and II (Table III) and were also increased during previous saturation exposures to nitrogen at 100 fsw and to helium at 1200 fsw (Table VI). Aldosterone outputs appeared to be elevated during the three high pressure $He-O_2$ saturation exposures included in Table VI, but not in the saturation N_2-O_2 exposure at 100 fsw or the non-saturation exposure at 200 fsw (1,10). Total outputs of antidiuretic hormone decreased during and after the shallow N_2-O_2 saturation exposure and also appeared to be decreased following the deep $He-O_2$ saturation exposures of Phases I and II (Table VI).

Some of the changes in plasma hormone concentrations which occurred in Predictive Studies IV were also found in previous exposures and others were not (Table VI). It is interesting that plasma cortisol was significantly decreased after a N_2-O_2 saturation exposure at 100 fsw (10) as well as during and after the much deeper $He-O_2$ saturation-excursion exposure (Phase II of the present study). In the case of the Phase II

TABLE VI. Biochemical and Endocrine Changes during and after Exposure to Increased Ambient Pressures

	Predictive Studies II		Predictive Studies III		Predictive Studies IV Phase I		Predictive Studies IV Phase II		Predictive Studies II	
Breathing gas	N_2-O_2		He-O_2		He-O_2		He-O_2		N_2-O_2	
Saturation depth	100 fsw		1200 fsw		800 fsw		1200 fsw		Non-saturation dive to 200 fsw	
Excursion depth	-		-		1200 fsw		1600 fsw		-	
	During Exposure	Post Exposure	During Exposure	Post[a] Exposure	During Exposure	Post Exposure	During Exposure	Post Exposure	During Exposure	Post Exposure
PLASMA ENDOCRINE DATA										
Cortisol	↔	↓*	↔	↔	-	↔	↓*	↓	↓*	↓
Aldosterone	-	-	-	-	-	↓	↓	↓	-	-
Angiotensin I	↑*	↔	↔	↔	-	↑*	↔	↔	↔	↔
Growth Hormone	-	-	↔	↔	-	↑*	↔	↔	-	-
Insulin	↑*	↑*	↔	↑	-	↔	↔	↔	↑	↑
Thyroxine	↔	↔	-	-	-	↑*	↔	↑*	-	-
DHA	-	-	-	-	-	↔	↔	↔	-	-
ACTH	-	-	-	-	-	↔	↔	↔	↓*	↓*
SERUM BIOCHEMICAL DATA										
Osmolality	↑*	↔	↑	↑	-	↑*	↓	↔	↓	↔
Sodium	↑*	↔	↔	↑*	-	↑*	↓	↔	↓*	↔
Potassium	↑*	↔	↔	↔	-	↔	↓	↔	↔	↔
Chloride	↔	↑*	↔	↔	-	↑*	↓*	↔	↓*	↔
Creatinine	↔	↔	↔	↔	-	↔	↔	↔	↔	↔
SGOT	↑	↔	↑	↔	-	↑	↔	↔	↔	↔
Calcium	↔	↔	-	-	-	↑	↔	↔	↔	↔
Magnesium	↔	↔	-	-	-	↔	↔	↔	↔	↔
CPK	↔	↔	↓	↓	-	↑	↑	↓	↔	↔
LDH	↑	↔	↑*	↔	-	↑*	↔	↑*	↑	↓
Triglycerides	↔	↔	-	-	-	↑*	↔	↔	↑	↑
URINE ENDOCRINE DATA										
Volume	↑*	↔	↑	↑	↑	↔	↑	↔	↓*	↓
Cortisol	↔	↔	↔	↑	↑	↔	↑	↔	↓	↓
Aldosterone	↔	↓	↑	↑	↑	↑	↑*	↓	-	-
ADH	↓*	↓	↓	↔	↓*	↓*	↔	↓	-	-
Epinephrine	↔	↔	↔	↔	↔	↔	↔	↔	↔	↔
Norepinephrine	↑*	↔	↔	↑	↔	↑	↑	↔	↔	↓
URINE BIOCHEMICAL DATA										
Osmolality	↔	↔	-	-	↓	↓*	↓*	↔	↑*	↑
Sodium	↑*	↔	↓	↓	↓*	↓*	↓	↓	↓	↔
Potassium	↔	↔	↑	↑	↑	↔	↑	↓	↔	↔
Chloride	↑*	↓	↓	↓	↓	↓*	↓	↓	↔	↔
Calcium	↔	↔	-	-	↓*	↓*	↓	↔	↑*	↔
Magnesium	↔	↔	-	-	↓	↔	↓	↓	↑	↔
Inorg. Phosphate	↔	↔	↑	↑	↔	↔	↔	↓	↑	↔
Uric Acid	↑*	↔	↔	↑	↑	↔	↔	↓	↓	↔
Creatinine	↔	↔	↔	↑	↑	↔	↔	↔	↑	↔

[a] Urine was collected only for a 12-hour period.

* Change significant.

- Not measured.

exposure, the association of increased urine cortisol outputs with decreased plasma levels (Tables III and VI) suggests the possibility of diminished adrenal stores of this hormone in response to chronic stress.

There were similar associations of increased urine outputs and decreased plasma levels of aldosterone after the Phase I and during the Phase II exposures (Table VI). However, the blood samples obtained during and immediately after the Phase II exposure were collected during the late afternoon and early evening while control samples were drawn in the early morning. Thus diurnal variation of plasma hormone concentrations could have contributed to the observed results (6,12).

Increased plasma concentrations of insulin were found in four of the five studies the results of which are summarized in Table VI. This may indicate some metabolic influence induced by chronic stress or other effect of exposure to increased hydrostatic pressure. However, the elevated plasma insulin level found during the Phase II exposure was in a nonfasting sample. In contrast with the repeated observation of increased insulin levels, the significant post-exposure increments in growth hormone and thyroxine that occurred in Predictive Studies IV were not found in any of the other four surveys (Table VI).

When the hormonal changes that occurred in Predictive Studies IV are evaluated in the context of prolonged exposure to a variety of stresses, they appear to be generally appropriate in nature and only moderate in degree. Neither the individual nor the total composite of responses can be considered to be extreme or detrimental. The hormonal responses of the subjects were similar to those found previously in saturation exposures to increased ambient pressures (1,7,10) as well as during space flight (11). These results provide no indication that the more rapid and higher pressure compressions and the repeated excursions employed in Predictive Studies IV were significantly more stressful than the slower compressions and less frequent excursions studied previously.

BIOCHEMICAL AND ELECTROLYTE RESPONSES TO INCREASED AMBIENT PRESSURE

Although there were many apparent alterations in the pattern of electrolyte excretion during both phases of Predictive Studies IV (Table VI), most of these were small in magnitude. They cannot definitely be attributed to changes in renal function since the corresponding electrolyte intakes were not measured. Sodium excretion and chloride excretion significantly increased during a N_2-O_2 saturation exposure at 100 fsw (2), while they consistently decreased in Phases I and II of the present study. The decreased sodium and increased potassium excretion rates found in both phases are consistent with a renal action of aldosterone which had an increased excretion rate during both exposures. Decreased excretion of sodium, chloride, calcium and magnesium in conjunction with increased potassium excretion were also found in previous deep He-O_2 saturation exposures (3,4,5).

The alterations in serum enzyme concentrations were generally small in magnitude and nearly all of them remained within normal ranges even when changes were statistically significant. Serum SGOT was significantly elevated after Phase I, with no important change throughout Phase II. Serum CPK had a similar pattern in Phase I except for a transient, slight elevation and then a decrease in average concentration during the Phase II exposure. Serum CPK also decreased during and after a previous He-O_2 saturation exposure at 1200 fsw (Table VI).

Of the three serum enzymes monitored in these studies, only LDH had consistently elevated values following both exposures. Average values of serum LDH also increased in the three previous pressure exposures (Table VI). For all four saturation exposures included in Table VI, the elevations of serum LDH concentration were accompanied by increases in plasma osmolality. Furthermore, the response patterns of serum LDH and plasma osmolality were nearly parallel throughout both phases of the present study. These similar response patterns may indicate that the increments in serum LDH concentration were at least partly caused passively by hemoconcentration, whether physiological or in sample handling.

RELATIONS OF BIOCHEMICAL, HORMONAL, ELECTROLYTE AND WATER BALANCE CHANGES

Of all the observations of blood composition made in association with the composite study, three related parameters are considered questionable: serum sodium, chloride and osmolality. These factors in electrolyte and water balance are normally maintained under precise physiological control. While the measurement methods employed (18) are extremely reliable, and prior studies have shown some hemoconcentration, some individual subject values tended to fall at the upper limit of or even above accepted normal ranges before, as well as during and after exposure to increased pressure. Extensive tracking has led to the conclusion that the sum of factors in the sampling, separation, storage and shipment of samples for analysis led to loss of between two and four percent of the water content of blood, resulting in equivalent increases in values separately measured for sodium, chloride and osmolality. The subject still deserves investigation and is reported here with technical critique to stimulate such study.

While some physiological hemoconcentration may have occurred, it is not considered likely that there were prominent changes in serum osmolality for several reasons. First, the subjects were queried concerning thirst and indicated that it was not unusual. The urine ADH output was lowered rather than elevated in association with the apparent hemoconcentration. Although urine output was significantly increased on only one day of the Phase I exposure, average 24-hour urine outputs were higher than control values throughout most of the two phases (Table III).

With the exception of an elevation on exposure day 2 of Phase I, average urine osmolality was decreased throughout the Phase I exposure as well as during the saturation and early decompression periods of Phase II (Table IV). An increased volume of dilute urine has been found previously during saturation exposures to increased ambient pressures (8,14,16,17,19), with a concurrent decrement in fluid intake and a state of negative water balance. Although water balance was not measured, daily fluid intakes were recorded by the subjects during both phases. On all but one of the exposure days, average fluid intake exceeded urine output

by less than 500 ml/day and on some days was less than urine volume (Table III). Addition of oxidation water to the fluid intake-urine output values and subtraction of insensible and fecal water losses would probably yield negative values for overall water balance on most exposure days.

Progressive loss of body fluids has been implicated as the cause of the weight loss usually found during saturation exposures to increased pressures (13,16). The subjects in the present study had average weight losses of 1.1 and 2.5 kg in Phases I and II, respectively (E-15).

If progressive loss of body fluids had caused both the weight loss and hemoconcentration found in the subjects of Phase I, concurrent reversal of these changes during the post-exposure period might be expected. However, subjects CC and FS who participated in both exposures still had increased values of plasma osmolality and serum sodium concentration at the start of Phase II (Appendix Table IIB) when body weight had returned to normal. Furthermore, the control values of plasma osmolality and serum sodium concentration were even higher in subjects GM and MP who were not exposed to increased ambient pressures in Phase I. Thus, the basis for the high control values for sodium, chloride and osmolality in these subjects is uncertain and requires detailed further study.

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TABLE IA. Effects on Plasma Endocrine Parameters of Exposure to Pressures Equivalent to 800 and 1200 fsw (Phase I)

Subject	Pre-Exposure Control				Post-Exposure			
	6/24	6/25	6/26	Mean ± SD	Day 1 7/15	Day 2 7/16	Day 3 7/17	Day 15 7/29
Cortisol (µg/dl)								
WS	13.5	8.7	10.8	11.0 ± 2.4	5.2	16.3	11.5	15.1
LJ	15.0	19.9	18.5	17.8 ± 2.6	15.3	9.4	10.2	24.9
FS	10.8	14.0	19.0	14.6 ± 4.2	17.0	20.3	25.0	30.0
CC	8.7	10.8	10.4	10.0 ± 1.0	8.7	19.5	15.0	16.2
Mean				13.4	11.6	16.4	15.4	21.6
±SD				3.6	5.5	5.0	6.7	17.1
±SEM				1.8	2.8	2.5	3.3	3.6
Aldosterone (pg/dl)								
WS	181	67	136	128 ± 57	51	180	197	134
LJ	212	397	219	276 ± 106	136	128	160	209
FS	132	169	224	175 ± 47	174	246	300	419
CC	144	158	118	140 ± 21	83	181	175	227
Mean				180	111	184	208	247
±SD				67	55	48	63	121
±SEM				34	27	24	32	61
Angiotensin I (ng/ml/hour)								
WS	0.22	0.22	0.07	0.17 ± 0.09	0.12	1.16	1.17	0.23
LJ	0.45	0.22	0.16	0.28 ± 0.16	0.24	0.50	0.34	2.07
FS	0.55	0.07	0.14	0.25 ± 0.26	0.18	0.21	1.36	2.10
CC	0.18	0.15	0.05	0.13 ± 0.07	0.40	0.56	0.67	1.69
Mean				0.21	0.24	0.61	0.88	1.52
±SD				0.07	0.12	0.40	0.47	0.88
±SEM				0.03	0.06	0.20	0.23	0.44
Growth Hormone (ng/ml)								
WS	5.0	8.4	6.6	6.7 ± 1.7	8.4	6.6	3.6	2.3
LJ	5.0	10.4	6.6	7.3 ± 2.8	8.4	6.6	5.0	8.4
FS	5.0	5.0	5.0	5.0 0	8.4	6.6	5.0	5.0
CC	6.6	2.3	6.6	5.2 ± 2.4	8.4	5.0	5.0	5.0
Mean				6.0	8.4	6.2	4.6	5.2
±SD				1.1	0	0.8	0.7	2.5
±SEM				0.6	0	0.4	0.4	1.2
Insulin (µU/ml)								
WS	15	15	21	17 ± 3	23	42	20	14
LJ	17	15	26	19 ± 5	23	20	17	17
FS	18	14	14	15 ± 2	-	15	14	18
CC	11	15	42	23 ± 17	17	17	20	9
Mean				18	21	24	18	14
±SD				3	3	12	3	4
±SEM				2	2	6	1	2
Thyroxine (T_4) (µg/dl)								
WS	7.7	6.8	7.8	7.4 ± 0.5	11.3	10.5	10.5	12.0
LJ	6.8	7.1	7.1	7.0 ± 0.2	9.0	9.0	8.5	8.5
FS	7.4	6.8	7.4	7.2 ± 0.3	7.1	9.5	10.5	10.5
CC	6.8	7.1	7.4	7.1 ± 0.3	8.5	9.5	9.0	8.5
Mean				7.2	9.0	9.6	9.6	9.9
±SD				0.2	1.7	0.6	1.0	1.7
±SEM				0.1	0.9	0.3	0.5	0.8
DβH (IU)								
WS	91.2	84.9	83.9	86.7 ± 4.0	90.0	85.1	74.7	63.8
LJ	55.9	63.7	46.4	55.3 ± 8.7	42.0	53.0	46.7	51.7
FS	49.5	59.0	52.2	53.6 ± 4.8	61.1	56.3	56.5	56.9
CC	54.3	62.5	57.6	58.1 ± 4.2	58.0	57.7	50.2	51.8
Mean				63.4	62.8	63.0	57.0	56.0
±SD				15.6	20.0	14.8	12.5	5.7
±SEM				7.8	10.0	7.4	6.2	2.8
ACTH (pg/ml)								
WS	10.9	22.2	-	16.6 -	145.6	12.0	10.4	10.4
LJ	1.3	8.1	9.1	6.2 ± 4.3	15.4	20.0	17.7	118.7
FS	9.9	16.4	3.7	10.0 ± 6.4	8.3	8.9	-	13.2
CC	7.9	8.1	2.5	6.2 ± 3.1	4.0	11.9	11.7	9.3
Mean				9.8	43.3	13.2	13.3	37.9
±SD				4.9	68.3	4.8	3.9	53.9
±SEM				2.5	34.2	2.4	2.2	26.9

TABLE IB. Effects on Plasma Endocrine Parameters of Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

Subject	Pre-Exposure Control			Exposure	Post-Exposure		
	7/31	Day 1[a]	Mean	Day 3	Day 1 8/27	Day 3 8/29	Day 9 9/4
Cortisol (µg/dl)							
MP	13.0	13.2	13.1	2.2	9.5	20.0	18.5
GM	22.0	21.2	21.6	7.6	18.5	16.7	20.2
FS	23.1	25.0	24.0	5.0	10.7	18.5	27.5
CC	16.7	21.0	18.9	13.3	12.0	7.8	9.5
Mean			19.4	7.0	12.7	15.8	18.9
±SD			4.7	4.7	4.0	5.5	7.4
±SEM			2.3	2.4	2.0	2.7	3.7
Aldosterone (pg/dl)							
MP	154	123	138	76	76	190	151
GM	132	244	188	121	174	228	214
FS	352	333	342	115	197	175	422
CC	150	191	170	139	150	144	209
Mean			210	113	149	184	249
±SD			91	27	52	35	119
±SEM			45	13	26	17	59
Angiotensin I (ng/ml/hour)							
MP	1.91	0.59	1.25	1.17	0.36	1.89	1.37
GM	1.44	0.78	1.11	0.63	0.71	1.43	0.54
FS	2.75	0.96	1.86	1.09	1.80	1.44	2.87
CC	1.00	0.55	0.78	1.07	0.98	2.09	1.08
Mean			1.25	0.99	0.96	1.71	1.46
±SD			0.45	0.24	0.61	0.33	1.00
±SEM			0.23	0.12	0.31	0.16	0.50
Growth Hormone (ng/ml)							
MP	0.9	8.4	4.6	5.0	2.2	5.0	2.3
GM	2.3	2.3	2.3	2.3	2.3	3.6	5.0
FS	5.0	8.4	6.7	6.6	5.0	3.6	8.4
CC	0.9	3.6	2.2	2.3	5.0	2.3	5.0
Mean			4.0	4.0	3.6	3.6	5.2
±SD			2.1	2.1	1.6	1.1	2.5
±SEM			1.1	1.1	0.8	0.6	1.2
Insulin (µU/ml)							
MP	11	21	16	26	15	-	17
GM	14	18	16	-	13	20	23
FS	17	20	18	25	14	17	42
CC	17	23	20	38	42	14	17
Mean			18	30	21	17	25
±SD			2	7	14	3	12
±SEM			1	4	7	2	6
Thyroxine (T_4) (µg/dl)							
MP	6.3	6.0	6.2	6.0	8.5	5.1	8.1
GM	5.7	6.0	5.8	-	7.8	8.5	7.4
FS	10.5	8.1	9.3	7.8	13.7	11.3	9.5
CC	8.5	6.3	7.4	7.1	10.0	11.5	9.0
Mean			7.2	7.0	10.0	9.1	8.5
±SD			1.6	0.9	2.6	3.0	0.9
±SEM			0.8	0.5	1.3	1.5	0.5
DSH (IU)							
MP	47.9	41.6	44.8	47.1	44.4	49.3	42.2
GM	25.8	25.8	25.8	25.8	31.3	24.7	22.6
FS	53.4	57.6	55.5	53.6	59.5	48.4	60.6
CC	51.1	41.6	46.4	43.5	58.5	46.8	54.7
Mean			43.1	42.5	48.4	42.3	45.0
±SD			12.5	11.9	13.3	11.8	16.8
±SEM			6.2	5.9	6.7	5.9	8.4
ACTH (pg/ml)							
MP	1.8	16.4	9.1	11.7	4.3	16.1	12.5
GM	0.08	14.9	7.5	-	21.6	3.2	-
FS	5.2	8.6	6.9	-	5.3	6.5	35.8
CC	2.4	20.7	11.6	4.7	8.8	5.0	6.7
Mean			8.8	8.2	10.0	7.7	18.3
±SD			2.1		8.0	5.8	15.4
±SEM			1.0		4.0	2.9	8.9

[a] Blood sample drawn at sea level before start of compression on exposure day 1. 8/7 for subjects FS, GM; 8/11 for subjects CC, MP.

TABLE IIA. Effects on Serum Biochemical Parameters of Exposures to Pressures Equivalent to 800 and 1200 fsw (Phase I)

Subject	Pre-Exposure Control 6/24	6/25	6/26	Mean ± SD	Post-Exposure Day 1 7/15	Day 2 7/16	Day 3 7/17	Day 15 7/29
Osmolality (mOsmo)								
WS	306	293	311	303 ± 9	308	316	305	306
LJ	284	302	289	292 ± 9	299	302	306	297
FS	255	303	295	284 ± 26	301	309	312	309
CC	289	296	294	293 ± 3	305	311	306	303
Mean				293	303	310	307	304
±SD				8	4	6	3	5
±SEM				4	2	3	2	3
Sodium (mEq/l)								
WS	146	145	152	148 ± 3	147	150	149	148
LJ	141	149	145	145 ± 3	142	147	150	145
FS	132	150	145	142 ± 9	148	152	154	151
CC	142	146	145	144 ± 2	147	151	150	148
Mean				145	146	150	151	148
±SD				2	3	2	2	2
±SEM				1	1	1	1	1
Potassium (mEq/l)								
WS	3.7	4.0	4.6	4.1 ± 0.5	3.6	4.0	3.8	3.8
LJ	4.1	4.2	3.6	4 0 ± 0.3	4.0	4.0	4.6	4.6
FS	3.4	4.3	4.1	3.9 ± 0.5	4.3	4.2	4.6	4.4
CC	4.0	4.4	3.9	4.1 ± 0.3	3.5	4.0	3.8	4.1
Mean				4.0	3.8	4.0	4.2	4.2
±SD				0.1	0.4	0.1	0.5	0.4
±SEM				0.05	0.2	0.05	0.2	0.2
Chloride (mEq/l)								
WS	107	104	110	107 ± 3	103	107	111	107
LJ	97	102	100	100 ± 2	104	104	106	104
FS	96	107	103	102 ± 5	100	103	108	104
CC	100	105	103	103 ± 2	102	108	105	107
Mean				102	102	106	108	106
±SD				4	2	2	3	2
±SEM				2	1	1	1	1
Creatinine (mg/dl)								
WS	1.1	1.0	1.3	1.1 ± 0.2	1.2	1.2	1.1	1.1
LJ	1.5	1.6	1.7	1.6 ± 0 2	1.5	1.8	1.8	2.0
FS	1.4	1.7	1.9	1.7 ± 0.2	1.9	1.7	1.9	2.1
CC	1.0	1.2	1.0	1.1 ± 0.2	1.3	1.3	1 2	1.3
Mean				1.4	1.5	1.5	1.5	1.6
±SD				0.3	0 3	0.4	0.4	0.5
±SEM				0.2	0.2	0.2	0.2	0.2
SGOT (mU/ml)								
WS	10	8	8	9 ± 2	-	12	11	9
LJ	-	13	12	12 ± 2	-	12	13	19
FS	12	11	17	13 ± 3	-	27	26	20
CC	8	8	10	9 ± 2	12	14	13	12
Mean				11	-	16	16	15
±SD				2		7	7	5
±SEM				1		4	4	3
Calcium (mg/dl)								
WS	9.2	9.1	9.4	9.2 ± 0.2	10.0	9.6	9.3	9.0
LJ	9.6	10.3	9.4	9.8 ± 0.5	9.6	9.8	9.9	9.5
FS	8.8	9.8	9 4	9.3 ± 0.5	10.6	10.7	10.6	10.7
CC	9.4	9.4	9.3	9.4 ± 0	9.9	10.1	9 9	9.5
Mean				9.4	10.0	10.0	9.9	9.7
±SD				0.3	0.4	0.5	0 5	0.7
±SEM				0.1	0.2	0.2	0.3	0.4
Magnesium (mg/dl)								
WS	2.0	2.0	2.3	2.1 ± 0.2	2.4	1.8	2.1	2.3
LJ	2.1	2.1	2 0	2.1 ± 0	2.2	2.2	2.3	2.3
FS	1.7	2.0	2.1	1.9 ± 0.2	2.1	2.2	2.3	2.0
CC	2.1	2 1	2.1	2.1 ± 0	2.2	2.3	2.2	2.1
Mean				2 0	2.2	2.1	2.2	2.2
±SD				0.1	0.1	0.2	0.1	0.2
±SEM				0.05	0 1	0.1	0.05	0.1
CPK (mU/ml)*								
WS	<2	33	23	28 ± 9	<2	27	49	52
LJ	35	32	32	33 ± 2	<2	<2	65	<2
FS	95	74	61	77 ± 17	126	131	85	83
CC	50	55	55	53 ± 3	98	134	105	141
Mean				48	57	74	76	70
±SD				22	65	69	24	58
±SEM				11	32	34	12	29
LDH (mU/ml)								
WS	116	77	90	94 ± 19	108	179	112	172
LJ	125	133	116	125 ± 9	159	138	215	188
FS	219	168	146	178 ± 38	151	159	181	262
CC	103	99	151	118 ± 29	129	193	129	198
Mean				129	137	167	159	205
±SD				35	23	24	47	40
±SEM				18	12	12	24	20
Triglycerides (mg/dl)								
WS	209	150	152	170 ± 33	324	213	320	137
LJ	261	189	239	230 ± 36	316	184	287	139
FS	290	228	172	230 ± 59	251	129	144	177
CC	74	81	81	79 ± 3	282	145	133	108
Mean				177	293	168	221	140
±SD				71	34	38	96	28
±SEM				36	17	19	48	14

*For CPK values <2 assumed equal to 2.

TABLE IIB. Effects on Serum Biochemical Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

Subject	Pre-Exposure Control			Exposure	Post-Exposure		
	7/31	Day 1[a]	Mean	Day 3	Day 1 8/27	Day 3 8/29	Day 9 9/4
Osmolality (mOsmo)							
MP	310	315	312	296	307	310	317
GM	316	298	307	300	311	300	332
FS	311	295	303	289	315	316	330
CC	307	312	310	302	318	304	307
Mean			308	297	313	308	322
±SD			4	6	5	7	12
±SEM			2	3	2	4	6
Sodium (mEq/l)							
MP	152	154	153	145	147	150	155
GM	154	146	150	144	153	148	162
FS	152	147	150	145	156	151	160
CC	151	152	152	146	152	147	149
Mean			151	145	152	149	156
±SD			2	1	4	2	6
±SEM			1	0.5	2	1	3
Potassium (mEq/l)							
MP	4.2	4.4	4.3	3.7	3 9	4.3	4 5
GM	4.3	4.1	4.2	4 3	4 2	4.8	4.4
FS	4 8	4.3	4.6	4.1	4.2	4.1	4.4
CC	4.2	4.4	4.3	3.7	3.9	3.9	4.2
Mean			4.4	4.0	4.0	4 3	4 4
±SD			0.2	0 3	0 2	0.4	0.1
±SEM			0.1	0.2	0.1	0.2	0.1
Chloride (mEq/l)							
MP	108	113	110	100	106	111	110
GM	110	106	108	97	108	105	115
FS	108	105	106	98	109	109	113
CC	110	110	110	100	107	105	108
Mean			109	99	108	108	112
±SD			2	2	1	3	3
±SEM			1	1	1	2	2
Creatinine (mg/dl)							
MP	1.6	1.7	1.7	1.5	1.6	1.6	1 8
GM	1.5	1 4	1 5	1.6	1.5	1.5	1.6
FS	1.9	1.9	1.9	1.8	2.0	1.9	1.9
CC	1.2	1.1	1.2	1.2	1.2	1.0	1 1
Mean			1.6	1.5	1.6	1.5	1 6
±SD			0 3	0.2	0.3	0.4	0.4
±SEM			0.1	0.1	0.2	0.2	0.2
SGOT (mU/ml)							
MP	18	9	14	13	16	17	15
GM	14	14	14	15	15	14	12
FS	25	22	24	20	16	16	22
CC	12	9	10	11	12	14	12
Mean			16	15	15	15	15
±SD			6	4	2	2	5
±SEM			3	2	1	1	2
Calcium (mg/dl)							
MP	10.2	9.8	10 0	10 4	9.9	10 1	10 5
GM	10 2	10.1	10.2	10.6	10.1	10 0	10.4
FS	10.6	9.6	10.1	10.2	10.5	10.0	10.3
CC	9.8	9.4	9.6	10 0	10.3	10.2	10.0
Mean			10.0	10 3	10.2	10 1	10.3
±SD			0.3	0.3	0.3	0 1	0.2
±SEM			0.1	0.1	0 1	0.05	0.1
Magnesium (mg/dl)							
MP	2.4	2 2	2 3	2 4	2.3	2.1	2.4
GM	2.4	2.2	2 3	2.4	2.3	2 0	2.5
FS	2.1	2.1	2 1	2.3	2 2	2.2	2.3
CC	2 3	2 3	2.3	2.4	2 3	2.2	2 3
Mean			2 3	2.4	2.3	2 1	2.4
±SD			0.1	0 05	0.05	0 1	0.1
±SEM			0 05	0.03	0.03	0 05	0.05
CPK (mU/ml)							
MP	62	51	56	99	50	53	51
GM	256	126	191	141	81	88	103
FS	212	92	152	297	115	97	94
CC	88	51	70	127	56	58	53
Mean			117	166	76	74	75
±SD			65	89	30	22	27
±SEM			32	44	15	11	14
LDH (mU/ml)							
MP	181	160	170	168	183	189	206
GM	258	245	252	259	288	264	289
FS	344	251	298	267	331	275	317
CC	189	176	182	215	236	224	202
Mean			226	227	260	238	254
±SD			60	46	64	39	58
±SEM			30	23	32	20	29
Triglycerides (mg/dl)							
MP	112	264	188	363	103	123	108
GM	78	103	90	60	82	70	129
FS	128	114	121	119	87	121	284
CC	85	138	112	145	131	112	114
Mean			128	172	101	106	159
±SD			42	132	22	25	84
±SEM			21	66	11	12	42

[a]Blood sample drawn at sea level before start of compression on exposure day 1. 8/7 for subjects FS, GM; 8/11 for subjects CC, MP

TABLE IIIA. Effects on Urine Endocrine Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase I)

Urine Cortisol (μg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	93.5	62.5	33.5	37.9	-	22.6	25.4	-	45.9 ± 27.1
LJ	19.8	40.0	5.5	22.8	61.9	25.9	19.9	21.4	27.2 ± 17.0
FS	-	49.9	37.7	61.8	66.1	-	36.5	-	50.4 ± 13.4
CC	60.3	74.0	50.4	49.4	-	-	67.2	86.6	64.6 ± 14.5
Mean									47.0
±SD									15.4
±SEM									7.7

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	82.5	34.9	66.3	68.8	341.0	126.8	12.2
LJ	125.5	104.2	51.0	30.8	36.2	16.4	-
FS	72.4	24.4	93.5	31.1	45.2	44.2	29.2
CC	224.4	190.3	87.9	70.5	91.0	99.0	-
Mean	126.2	88.4	74.7	50.3	128.4	71.6	-
±SD	69.4	76.6	19.7	22.4	143.8	50.3	
±SEM	34.7	38.3	9.8	11.2	71.9	25.2	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	57.8	39.8	123.5	96.8	72.0	81.6	45.1
LJ	23.7	40.9	33.2	58.8	47.8	15.9	36.0
FS	17.3	30.3	66.0	13.7	25.5	29.4	42.5
CC	73.7	139.1	143.4	67.8	101.9	72.5	96.9
Mean	43.1	62.5	91.5	59.3	61.8	49.8	55.1
±SD	27.0	51.3	50.9	34.4	32.8	32.1	28.1
±SEM	13.6	25.6	25.4	17.2	16.4	16.1	14.1

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	25.2	27.4	46.5	43.5
LJ	18.5	14.5	21.7	26.3
FS	37.7	32.0	21.6	35.7
CC	44.5	-	35.3	37.5
Mean	31.5	24.6	31.3	35.8
±SD	11.8	9.1	12.0	7.1
±SEM	5.9	5.2	6.0	3.6

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIA (cont'd). Phase I

Urine Aldosterone (μg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	8.9	8.5	6.4	-	-	2.0	8.8	-	6.9 ± 2.9
LJ	4.8	10.5	3.0	4.6	11.3	10.8	10.1	4.1	7.4 ± 3.7
FS	-	6.9	6.6	10.5	8.4	-	6.6	-	7.8 ± 1.8
CC	16.0	6.7	6.0	0.5	-	-	18.3	19.0	11.1 ± 7.6
Mean									8.3
±SD									1.9
±SEM									1.0

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	14.4	10.6	12.4	8.7	7.3	15.4	9.3
LJ	10.0	18.3	9.6	6.6	2.7	8.1	-
FS	13.5	7.5	2.4	5.3	10.3	8.6	4.6
CC	18.0	31.2	16.0	10.7	14.6	16.2	-
Mean	14.0	16.9	10.1	7.8	8.7	12.1	-
±SD	3.3	10.6	5.8	2.4	5.0	4.3	
±SEM	1.6	5.3	2.9	1.2	2.5	2.2	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	12.4	9.2	10.1	5.3	3.4	3.6	3.3
LJ	5.0	5.4	5.5	7.7	8.3	3.1	5.4
FS	8.5	10.9	12.2	8.0	7.6	5.2	4.4
CC	14.0	12.2	18.7	18.4	11.7	11.4	11.8
Mean	10.0	9.4	11.6	9.8	7.8	5.8	6.2
±SD	4.0	3.0	5.5	5.8	3.4	3.8	3.8
±SEM	2.0	1.5	2.7	2.9	1.7	1.9	1.9

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	7.9	25.2	21.8	3.1
LJ	4.2	5.8	13.1	10.1
FS	-	13.7	22.8	13.6
CC	12.0	18.3	9.9	10.3
Mean	8.0	15.8	16.9	9.3
±SD	3.9	8.1	6.4	4.4
±SEM	2.2	4.1	3.2	2.2

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIA (cont'd). Phase I

Urine ADH (mU/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	134.2	123.5	89.4	91.0	-	54.0	70.0	-	93.7 ± 30.6
LJ	11.2	40.7	4.6	12.9	23.9	50.8	36.7	31.4	26.5 ± 16.1
FS	-	89.3	81.2	41.3	66.0	-	41.6	-	63.9 ± 22.1
CC	44.4	47.6	41.9	26.9	-	-	32.5	39.0	38.7 ± 7.8
Mean									55.7
±SD									29.7
±SEM									14.9

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	70.6	45.1	68.1	57.2	76.4	42.9	12.3
LJ	27.5	37.0	33.3	22.0	12.5	23.8	-
FS	67.0	39.8	26.9	22.3	23.9	21.0	18.2
CC	99.2	43.3	27.7	41.8	26.3	21.4	-
Mean	66.1	41.3	39.0	35.8	34.8	27.3	-
±SD	29.5	3.6	19.6	17.0	28.4	10.5	
±SEM	14.7	1.8	9.8	8.5	14.2	5.2	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	29.4	53.2	41.9	30.0	30.4	22.6	39.6
LJ	14.7	27.3	7.8	24.8	31.7	10.0	17.1
FS	21.0	21.6	25.0	16.2	21.0	17.4	32.9
CC	20.0	28.4	27.2	18.4	22.6	23.9	31.9
Mean	21.3	32.6	25.5	22.4	26.4	18.5	30.4
±SD	6.1	14.0	14.0	6.3	5.4	6.3	9.5
±SEM	3.0	7.0	7.0	3.1	2.7	3.2	4.7

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	69.2	42.9	72.0	51.7
LJ	10.5	26.4	7.5	7.2
FS	27.8	42.8	14.0	23.7
CC	34.2	26.9	24.5	30.3
Mean	35.4	34.8	29.5	28.2
±SD	24.6	9.4	29.2	18.4
±SEM	12.3	4.7	14.6	9.2

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIA (cont'd). Phase I

Urine Epinephrine (mg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	-	23.1	-	-	-	-	-	-	23.1 ± -
LJ	12.3	-	4.4	12.0	13.8	4.1	21.5	7.7	10.8 ± 6.1
FS	-	8.8	14.7	19.3	22.8	-	28.9	-	18.9 ± 7.6
CC	38.6	80.7	67.5	67.7	-	-	68.1	139.1	77.0 ± 33.6
Mean									32.4
±SD									30.1
±SEM									15.1

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	-	5.4	-	18.6	7.2	11.9	10.2
LJ	30.3	44.6	41.5	12.1	45.9	28.7	-
FS	22.6	10.2	11.5	11.3	15.1	8.1	7.4
CC	132.0	-	-	-	7.7	5.9	-
Mean	61.6	20.1	-	14.0	19.0	13.6	-
±SD	61.1	21.4		4.0	18.3	10.3	
±SEM	35.3	12.3		2.3	9.2	5.2	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	8.3	19.8	15.0	19.2	2.7	16.0	1.3
LJ	24.5	-	-	12.1	2.8	14.6	0.6
FS	7.6	10.0	5.7	7.9	11.5	-	6.4
CC	3.2	18.4	2.9	-	-	-	-
Mean	10.9	16.1	7.9	13.1	5.7	-	2.8
±SD	9.3	5.3	6.3	5.7	5.1		3.2
±SEM	4.7	3.1	3.7	3.3	2.9		1.8

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	4.8	8.5	3.4	-
LJ	7.7	2.1	17.1	6.6
FS	9.3	6.5	-	26.2
CC	14.8	-	-	4.5
Mean	9.2	5.7	-	12.4
±SD	4.2	3.3		12.0
±SEM	2.1	1.9		6.9

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIA (cont'd). Phase I

Urine Norepinephrine (μg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	81.7	49.6	60.1	92.1	-	28.1	-	-	62.3 ± 25.5
LJ	14.1	64.3	13.3	31.6	55.2	56.4	40.5	20.4	37.0 ± 20.1
FS	-	50.7	36.5	39.5	38.2	-	29.9	-	39.0 ± 7.6
CC	30.2	-	-	-	-	-	-	-	30.2 ± -
Mean									42.1
±SD									14.0
±SEM									7.0

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	56.1	37.3	-	47.0	101.7	97.0	14.6
LJ	26.9	59.4	25.1	21.9	-	21.1	-
FS	14.8	43.6	63.0	26.9	20.9	20.5	12.7
CC	-	100.2	-	67.5	51.4	45.4	-
Mean	32.6	60.1	-	40.8	58.0	46.0	-
±SD	21.2	28.3		20.8	40.8	35.9	
±SEM	12.3	14.1		10.4	23.6	18.0	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	40.3	22.4	40.9	28.0	25.4	37.7	34.8
LJ	20.9	77.4	27.1	21.5	38.4	6.5	24.2
FS	32.0	16.2	32.8	32.1	15.3	29.9	17.8
CC	52.0	27.5	26.5	49.3	6.4	41.7	41.1
Mean	36.3	35.9	31.8	32.7	21.4	29.0	29.5
±SD	13.1	28.1	6.7	11.9	13.8	15.7	10.5
±SEM	6.6	14.0	3.3	5.9	6.9	7.9	5.2

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	41.8	73.1	92.1	73.4
LJ	26.6	24.5	40.6	62.3
FS	36.2	74.5	206.7	11.0
CC	43.4	66.9	41.0	22.8
Mean	37.0	59.8	95.1	42.4
±SD	7.6	23.7	78.2	30.2
±SEM	3.8	11.9	39.1	15.1

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIA (cont'd). Phase I

Volume (ml/24 hour)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	1290	2200	1290	1850	-	1100	1495	-	1538 ± 414
LJ	640	1600	840	1300	1820	1620	1530	840	1274 ± 441
FS	-	1160	1480	2290	1130	-	1490	-	1510 ± 467
CC	1800	1000	1400	960	-	-	1680	2250	1515 ± 497
Mean									1459
±SD									124
±SEM									62

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	2200	820	3400	2150	2200	1950	740
LJ	1000	840	1020	820	640	620	-
FS	2010	580	2150	1390	1560	1880	1390
CC	2400	1703	2930	1740	2600	1800	-
Mean	1902	986	2375	1525	1750	1562	-
±SD	622	492	1040	563	855	631	
±SEM	311	246	520	282	428	316	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	1630	1990	1900	2150	1200	1700	1700
LJ	1480	1860	840	1200	920	910	890
FS	750	740	1500	1820	1500	1510	1330
CC	2200	2600	2680	2460	2240	1380	1550
Mean	1515	1798	1730	1908	1465	1375	1368
±SD	597	775	770	539	568	337	353
±SEM	298	388	385	270	284	168	176

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	1360	2380	1500	1850
LJ	860	500	700	650
FS	1300	1600	600	870
CC	1710	2520	720	1340
Mean	1308	1750	880	1178
±SD	349	926	417	533
±SEM	174	463	208	266

[a] Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IIIA (cont'd). Phase I

Fluid Intake (ml/24 hour)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	3451	2773	1947	2457	3659	3481	-	-	2961 ± 680
LJ	-	-	-	1539	-	-	-	1658	1598 ± -
FS	2891	2106	2451	1598	1425	2736	1490	-	2100 ± 610
CC	-	-	-	1066	-	-	-	710	888 ± -
Mean									1887
±SD									872
±SEM									436

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	1569	1894	1865	1635	3818	1510	2383
LJ	1066	1273	2073	1318	1125	1169	-
FS	1924	947	2028	1480	1658	1184	1110
CC	1480	2338	2679	2486	1154	1850	-
Mean	1510	1613	2161	1730	1939	1428	-
±SD	353	623	356	521	1276	322	
±SEM	176	311	178	260	638	161	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	2679	1362	1569	2102	1495	1954	2324
LJ	2131	1643	829	901	1658	524	1362
FS	1006	2338	1954	3463	1658	1776	1776
CC	2072	3019	1391	2294	1450	1480	1273
Mean	1972	2090	1436	2190	1565	1434	1684
±SD	700	743	468	1049	109	637	480
±SEM	350	371	234	524	54	318	240

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	2013	2921	1416	1593
LJ	1598	-	-	-
FS	1184	2679	2907	2419
CC	1421	-	-	2124
Mean	1554			2045
±SD	350			419
±SEM	175			242

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIB. Effects on Urine Endocrine Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

Urine Cortisol (μg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	30.5	31.8	14.4	41.3	19.0	15.6	-	22.7	25.0 ± 9.8
GM	70.1	88.7	62.1	69.8	-	-	-	-	72.7 ± 11.4
FS	39.4	50.4	69.1	36.2	-	-	-	-	48.8 ± 14.8
CC	42.5	67.6	47.4	52.5	63.9	51.8	48.2	89.3	57.9 ± 15.3
Mean									51.1
±SD									20.0
±SEM									10.0

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	50.5	35.5	21.3	20.7	46.9	30.2	-	-	-	-
GM	117.5	81.9	124.0	171.0	57.8	120.0	66.5	140.4	58.5	58.5
FS	56.6	35.6	79.3	87.0	24.3	60.6	49.9	50.7	69.1	45.3
CC	37.2	62.3	80.5	82.9	137.5	63.3	-	-	-	-
Mean	65.4	53.8	76.3	90.4	66.6	68.5	-	-	-	-
±SD	35.6	22.6	42.1	61.7	49.3	37.5				
±SEM	17.8	11.3	21.1	30.9	24.6	18.7				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	44.0	12.4	19.5	20.0	19.0	19.0	11.8	17.0	17.5	31.9
GM	96.0	150.0	105.5	111.0	171.3	92.5	78.0	43.5	140.1	77.1
FS	34.4	54.5	19.4	44.4	65.6	44.5	90.0	42.3	51.9	53.6
CC	48.4	96.6	-	31.5	46.1	-	32.5	15.0	33.6	28.1
Mean	55.7	78.4	48.1	51.7	75.5	52.0	53.1	29.4	60.8	47.7
±SD	27.5	58.8	49.7	40.8	66.7	37.3	37.0	15.6	54.7	22.6
±SEM	13.7	29.4	28.7	20.4	33.3	21.5	18.5	7.8	27.4	11.3

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	37.9	15.2	39.9	27.9	18.6	19.5	19.4	21.3
GM	198.3	60.3	147.3	102.6	85.0	42.3	139.8	55.3
FS	-	21.3	-	41.9	23.4	12.2	41.0	21.7
CC	29.7	15.1	-	-	71.5	51.0	33.8	21.6
Mean	88.6	28.0	-	57.5	49.6	31.2	58.5	30.0
±SD	95.1	21.7		39.7	33.6	18.4	54.9	16.9
±SEM	54.9	10.9		22.9	16.8	9.2	27.5	8.4

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIB (cont'd). Phase II

Urine Aldosterone (μg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	7.8	6.6	7.0	8.9	8.6	5.3	-	3.9	6.9 ± 1.9
GM	11.3	15.5	4.2	6.9	-	-	-	-	9.5 ± 5.0
FS	12.7	11.0	6.7	4.6	-	-	-	-	8.8 ± 3.8
CC	4.5	19.8	13.3	13.3	6.6	7.0	8.4	13.9	10.8 ± 5.1
Mean									9.0
±SD									1.6
±SEM									0.8

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	5.8	7.3	8.7	12.4	6.1	8.3	-	-	-	-
GM	8.5	4.7	34.9	18.4	8.5	19.5	17.2	38.0	9.2	11.5
FS	4.5	6.4	12.0	20.1	5.2	17.8	9.9	7.6	5.3	6.7
CC	3.4	9.8	27.6	25.0	13.0	5.8	-	-	-	-
Mean	5.6	7.1	20.8	19.0	8.2	12.8	-	-	-	-
±SD	2.2	2.1	12.5	5.2	3.5	6.8				
±SEM	1.1	1.1	6.3	2.6	1.7	3.4				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	5.9	6.5	7.3	8.1	6.4	7.3	3.9	3.5	3.4	3.3
GM	12.6	11.5	10.1	15.8	15.7	13.5	20.6	10.8	13.8	8.3
FS	5.9	12.0	9.9	11.8	3.2	12.0	8.4	7.8	9.5	6.0
CC	2.7	5.6	-	2.0	5.1	-	3.3	1.1	2.9	2.2
Mean	6.8	8.9	9.1	9.4	7.6	10.9	9.0	5.8	7.4	5.0
±SD	4.2	3.3	1.6	5.9	5.6	3.2	8.0	4.3	5.2	2.7
±SEM	2.1	1.7	0.9	2.9	2.8	1.9	4.0	2.2	2.6	1.4

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	3.1	4.5	7.7	4.4	3.1	1.0	2.3	1.9
GM	17.0	6.5	16.9	12.4	8.4	4.7	7.4	3.8
FS	-	2.3	-	6.5	2.9	4.8	4.8	5.7
CC	3.2	2.0	-	-	0.9	4.3	5.6	3.1
Mean	7.8	3.8	-	7.8	3.8	3.7	5.0	3.6
±SD	8.0	2.1		4.1	3.2	1.8	2.1	1.6
±SEM	4.6	1.1		2.4	1.6	0.9	1.1	0.8

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IIIB (cont'd). Phase II

Urine ADH (mU/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	34.9	35.6	35.3	26.9	45.4	34.2	-	39.9	36.0 ± 5.6
GM	19.4	42.0	26.8	54.0	-	-	-	-	35.6 ± 15.4
FS	18.0	25.9	28.2	32.2	-	-	-	-	26.1 ± 6.0
CC	13.1	9.8	17.4	30.4	23.7	12.7	27.0	26.4	20.1 ± 7.6
Mean									29.4
±SD									7.7
±SEM									3.9

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	40.5	29.0	34.9	28.4	24.9	30.2	-	-	-	-
GM	24.8	18.3	72.8	62.2	24.6	28.8	30.2	46.4	22.8	12.0
FS	62.6	24.1	25.1	22.4	15.2	22.8	21.8	27.3	18.3	17.3
CC	18.3	19.6	26.0	22.5	18.5	16.5	-	-	-	-
Mean	36.6	22.8	39.7	33.9	20.8	24.6	-	-	-	-
±SD	19.7	4.9	22.5	19.1	4.8	6.3				
±SEM	9.9	2.4	11.3	9.5	2.4	3.1				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	61.4	57.2	19.3	24.6	20.5	28.1	27.7	23.4	26.6	20.1
GM	42.8	4.8	31.8	35.2	35.1	20.8	36.5	5.0	2.5	31.6
FS	13.2	20.2	9.0	12.7	18.7	14.8	16.8	16.3	19.3	15.6
CC	11.5	97.2	-	17.8	13.3	-	23.2	15.4	9.9	8.3
Mean	32.2	44.8	20.0	22.6	21.9	21.2	26.0	15.0	14.6	18.9
±SD	24.2	41.2	11.4	9.7	9.3	6.7	8.3	7.6	10.6	9.8
±SEM	12.2	20.6	6.6	4.9	4.7	3.8	4.1	3.8	5.3	4.9

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	5.4	7.0	15.3	13.7	13.1	21.0	14.8	17.0
GM	36.5	15.6	27.7	16.8	30.8	37.6	15.1	17.7
FS	-	13.2	-	19.0	13.7	21.9	14.7	24.0
CC	17.2	29.1	-	-	19.7	22.2	22.0	11.9
Mean	19.7	16.2	-	16.5	19.3	25.7	16.6	17.6
±SD	15.7	9.3		2.7	8.2	8.0	3.6	5.0
±SEM	9.1	4.7		1.5	4.1	4.0	1.8	2.5

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IIIB (cont'd). Phase II

Urine Epinephrine (μg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	22.3	32.3	21.3	3.5	15.6	11.0	-	13.0	17.0 ± 9.3
GM	18.6	-	0.4	-	-	-	-	-	9.5 -
FS	9.5	15.7	6.0	3.9	-	-	-	-	8.8 ± 5.2
CC	6.3	39.9	-	-	31.2	12.1	3.8	-	18.7 ± 16.1
Mean									13.5
±SD									5.1
±SEM									2.5

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP			6.6	2.5	6.4	5.3	-	-	-	-
GM	-	-	-	-	-	-	-	-	-	-
FS	6.8	12.5	6.4	8.5	-	22.1	4.7	6.5	1.8	10.0
CC	8.2	14.2	-	-	0.9	6.4	-	-	-	-
Mean	-	-	-	-	-	11.3	-	-	-	-
±SD						9.4				
±SEM						5.4				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	2.5	25.8	-	-	-	-	-	-	-	-
GM	102.9	-	-	-	-	-	-	-	-	-
FS	-	3.5	0.9	0.8	-	-	-	69.2	-	-
CC	-	-	-	2.8	-	-	-	2.8	4.4	1.2
Mean										
±SD										
±SEM										

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	-	37.0	-	-	-	22.8	-	-
GM	-	-	-	-	-	-	7.8	2.1
FS	-	6.3	-	-	8.1	-	-	-
CC	-	3.1	-	-	6.1	7.4	14.7	9.5
Mean		15.5						
±SD		18.7						
±SEM		10.8						

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IIIB (cont'd). Phase II

Urine Norepinephrine (μg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	51.1	43.8	51.0	70.9	49.6	43.5	-	47.5	51.1 ± 9.3
GM	87.4	33.5	53.3	67.2	-	-	-	-	60.4 ± 22.8
FS	25.5	7.2	16.3	11.2	-	-	-	-	15.0 ± 7.8
CC	34.6	91.6	57.7	75.3	65.3	32.4	13.2	21.9	49.0 ± 27.7
Mean									43.9
±SD									19.9
±SEM									9.9

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	73.1	88.8	84.6	58.8	43.1	38.6	-	-	-	-
GM	54.4	34.3	202.0	76.0	50.7	-	-	48.6	60.5	-
FS	22.3	23.4	15.5	79.3	18.8	34.3	48.2	8.3	25.0	2.4
CC	19.1	52.0	113.8	96.0	68.4	27.0	-	-	-	-
Mean	42.2	49.6	104.0	77.5	45.2	33.3	-	-	-	-
±SD	26.0	28.7	77.3	15.2	20.6	5.9				
±SEM	13.0	14.3	38.6	7.6	10.3	3.4				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	59.8	19.5	-	-	-	-	-	32.1	-	-
GM	-	-	-	-	-	-	-	-	-	-
FS	13.5	2.5	12.8	7.0	32.8	24.5	-	-	-	-
CC	15.4	25.2	-	34.9	23.6	-	22.7	17.2	11.7	7.7
Mean	29.6	15.7	-	-	-	-	-	-	-	-
±SD	26.2	11.8								
±SEM	15.1	6.8								

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	-	15.8	56.9	19.9	-	-	-	-
GM	-	110.6	-	58.4	107.6	76.0	29.4	39.6
FS	-	62.2	-	35.3	-	-	-	-
CC	19.3	10.0	-	-	37.8	9.0	3.1	18.6
Mean	-	49.6		37.9	-	-	-	-
±SD		46.9		19.4				
±SEM		23.4		11.2				

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IIIB (cont'd). Phase II

Volume (ml/24 hour)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	1450	2050	1600	1920	1150	1600	-	1420	1599 ± 307
GM	850	900	600	900	-	-	-	-	812 ± 144
FS	750	1050	960	1050	-	-	-	-	952 ± 142
CC	680	1610	930	1420	2780	1420	1060	2380	1535 ± 718
Mean									1224
±SD									400
±SEM									200

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	2020	1820	1640	1220	1340	1890	-	-	-	-
GM	1000	1950	1600	1900	850	2400	1000	2400	1000	1500
FS	1300	1250	1220	1200	600	2090	1560	1370	940	1710
CC	1240	1500	1400	1610	2670	2750	-	-	-	-
Mean	1390	1630	1465	1482	1365	2282	-	-	-	-
±SD	440	316	194	336	923	376				
±SEM	220	158	97	168	461	188				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	1240	2150	1730	1600	2320	1620	1150	850	2000	1750
GM	1600	2400	1900	1200	2500	1000	1300	1208	950	1400
FS	740	660	480	800	1075	590	1000	1020	830	1020
CC	1670	2800	-	1340	2560	-	2030	1000	2400	1560
Mean	1312	2002	1370	1235	2114	1070	1370	1020	1545	1432
±SD	426	934	775	334	700	519	457	147	775	310
±SEM	213	467	448	167	350	299	228	73	388	155

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	3700	600	1100	1800	1200	1500	1850	1940
GM	1300	900	950	900	850	600	650	850
FS	-	600	-	570	520	1220	1050	700
CC	2580	1370	-	-	2200	1890	1500	1540
Mean	2527	868	-	1090	1192	1302	1262	1258
±SD	1201	364		637	727	543	523	584
±SEM	693	182		368	363	271	262	292

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IIIB (cont'd). Phase II

Fluid Intake (ml/24 hour)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	3127	2242	2537	1770	2478	2006	2419	1888	2308 ± 376
GM	2537	2301	2507	3304	-	-	-	-	2662 ± 538
FS	1652	3127	2950	2714	-	-	-	-	2611 ± 807
CC	1239	2773	1947	1003	-	-	-	-	1740 ± 972
Mean									2330
±SD									423
±SEM									212

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	1298	1006	1865	1065	1273	1066	-	-	-	-
GM	918	1569	1376	1770	1317	1598	2301	1184	1835	1416
FS	1169	1968	1243	1213	636	1776	651	1302	1361	1670
CC	918	2427	2013	977	1746	977	-	-	-	-
Mean	1076	1742	1624	1256	1243	1354	-	-	-	-
±SD	190	603	372	356	458	393				
±SEM	95	302	186	178	229	196				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	1362	1243	1658	1569	888	1421	1243	844	1302	1066
GM	2301	2419	1416	1770	2539	1180	1652	1593	1243	2353
FS	1628	2832	1539	1593	1652	1416	1598	1180	2260	1711
CC	1510	1362	1509	1658	1332	1598	1569	1302	1332	858
Mean	1700	1964	1531	1648	1602	1404	1516	1230	1534	1497
±SD	415	784	100	90	698	172	185	310	485	676
±SEM	208	392	50	45	349	86	92	155	243	338

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	-	-	2537	2360	-	-		
GM	-	2419	2301	-	-	-		
FS	1593	2360	3068	3658	2242	2478		
CC	-	-	-	-	-	-		
Mean	-	-	2635	-	-	-		
±SD			393					
±SEM			227					

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IVA. Effect on Urine Biochemical Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase I)

Urine Uric Acid (mg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	955	836	851	962	-	594	927	-	854 ± 137
LJ	269	768	185	338	910	745	643	403	533 ± 269
FS	-	882	888	916	768	-	1013	-	893 ± 87
CC	972	620	700	576	-	-	840	945	776 ± 169
Mean									764
±SD									161
±SEM									81

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	792	672	952	903	1012	1248	370
LJ	600	1831	694	426	218	471	-
FS	884	661	903	556	562	752	500
CC	960	954	820	940	832	828	-
Mean	809	1030	842	706	656	825	-
±SD	155	551	113	255	346	321	
±SEM	78	276	56	127	173	161	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	782	955	1102	946	816	952	782
LJ	444	595	302	480	644	328	427
FS	525	740	780	692	510	483	692
CC	792	1040	911	886	851	938	806
Mean	636	832	774	751	705	675	677
±SD	178	202	341	211	158	318	174
±SEM	89	101	171	105	79	159	87

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	734	714	1020	1184
LJ	413	360	686	468
FS	598	768	924	1263
CC	889	1109	547	750
Mean	658	738	794	916
±SD	202	307	216	374
±SEM	101	153	108	187

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVA (cont'd). Phase I

Urine Osmolality (mOsmo)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	925	579	988	780	-	690	827	-	798 ± 149
LJ	531	586	294	382	641	668	563	682	543 ± 139
FS	-	951	858	528	705	-	785	-	765 ± 161
CC	671	848	719	648	-	-	579	568	672 ± 103
Mean									695
±SD									114
±SEM									57

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	633	887	371	471	598	557	560
LJ	593	937	673	736	404	961	-
FS	574	971	481	498	508	566	444
CC	476	617	349	660	473	549	-
Mean	569	853	468	591	496	658	-
±SD	67	161	148	128	81	202	
±SEM	33	81	74	64	40	101	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	544	653	641	603	651	713	622
LJ	454	518	451	564	831	580	600
FS	640	911	592	487	480	478	721
CC	432	461	424	437	471	637	651
Mean	518	636	527	523	608	602	648
±SD	95	200	106	75	170	99	53
±SEM	48	100	53	37	85	50	26

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	752	281	606	704
LJ	639	402	645	674
FS	630	519	1016	1041
CC	643	510	687	795
Mean	666	428	738	804
±SD	58	112	188	166
±SEM	29	56	94	83

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVA (cont'd). Phase I

Urine Sodium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	227	308	297	307	-	162	238	-	256 ± 59
LJ	79	170	54	125	280	230	174	252	170 ± 82
FS	-	249	323	289	170	-	298	-	266 ± 60
CC	265	205	263	123	-	-	168	227	208 ± 56
Mean									225
±SD									45
±SEM									22

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	229	80	197	161	253	135	64
LJ	79	92	71	119	54	92	-
FS	187	92	140	139	148	253	136
CC	168	97	164	231	241	164	-
Mean	166	90	143	162	174	161	
±SD	63	7	53	49	93	68	
±SEM	32	4	27	24	46	34	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	150	304	200	275	238	250	241
LJ	124	244	68	144	144	116	110
FS	64	93	134	160	158	159	246
CC	156	250	190	172	228	185	177
Mean	124	223	148	188	192	178	194
±SD	42	91	61	59	48	56	64
±SEM	21	45	30	30	24	28	32

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	177	58	77	309
LJ	106	19	59	38
FS	166	126	45	131
CC	169	232	77	169
Mean	154	109	64	162
±SD	33	93	16	112
±SEM	16	47	8	56

[a] Exposure day 1 was 7/1 for subjects WS and FS: 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IVA (cont'd). Phase I

Urine Potassium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	117	99	81	111	-	33	102	-	90 ± 32
LJ	36	61	24	46	122	55	77	74	62 ± 31
FS	-	72	71	87	63	-	94	-	77 ± 13
CC	95	65	62	61	-	-	81	122	81 ± 24
Mean									78
±SD									12
±SEM									6

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	147	93	160	112	92	96	49
LJ	72	72	63	60	27	78	-
FS	121	64	146	60	78	117	63
CC	113	143	100	99	133	146	-
Mean	113	93	117	83	82	109	-
±SD	31	36	44	27	44	29	
±SEM	16	18	22	13	22	15	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	80	139	82	77	62	61	61
LJ	49	78	37	68	70	40	43
FS	48	56	53	46	45	45	68
CC	95	112	91	76	94	62	73
Mean	68	96	66	67	68	52	61
±SD	23	37	25	14	20	11	13
±SEM	12	18	13	7	10	6	7

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	92	67	99	104
LJ	59	17	60	42
FS	81	80	70	69
CC	92	96	58	86
Mean	81	65	72	75
±SD	16	34	19	26
±SEM	8	17	10	13

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVA (cont'd). Phase I

Urine Chloride (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	217	268	250	279	-	83	223	-	220 ± 71
LJ	73	162	60	125	322	167	151	201	158 ± 82
FS	-	229	297	268	164	-	270	-	246 ± 51
CC	270	212	265	131	-	-	173	221	212 ± 54
Mean									209
±SD									37
±SEM									18

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	279	114	173	163	266	127	19
LJ	107	107	79	120	48	102	-
FS	205	96	140	90	145	246	108
CC	166	114	161	223	263	164	-
Mean	189	108	138	149	181	160	-
±SD	72	8	42	58	105	63	
±SEM	36	4	21	29	52	31	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	86	267	196	241	200	202	185
LJ	107	195	69	122	129	90	89
FS	47	81	116	137	122	122	194
CC	136	237	180	160	222	155	161
Mean	94	195	140	165	168	142	157
±SD	37	82	59	53	50	48	48
±SEM	19	41	29	26	25	24	24

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	162	64	75	285
LJ	95	15	45	28
FS	159	120	32	125
CC	164	222	81	190
Mean	145	105	58	157
±SD	33	89	24	108
±SEM	17	44	12	54

[a] Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IVA (cont'd). Phase I

Urine Calcium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	16.5	18.4	21.3	19.3	-	12.6	18.7	-	17.8 ± 2.9
LJ	6.4	18.6	5.4	6.7	15.6	15.8	10.9	5.7	10.6 ± 5.4
FS	-	17.4	21.1	17.3	12.1	-	15.3	-	10.6 ± 3.4
CC	16.6	13.2	15.5	8.1	-	-	16.4	22.0	15.3 ± 4.7
Mean									15.1
±SD									3.2
±SEM									1.6

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	19.1	8.5	16.8	14.0	14.9	7.3	3.4
LJ	7.4	11.7	10.7	6.0	2.7	5.3	-
FS	12.8	6.9	11.6	10.5	9.9	9.5	6.1
CC	21.8	15.6	18.6	17.6	16.7	10.2	-
Mean	15.3	10.7	14.4	12.0	11.1	8.1	-
±SD	6.5	3.8	3.9	5.0	6.3	2.2	
±SEM	3.2	1.9	1.9	2.5	3.1	1.1	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	10.2	14.3	18.1	16.5	13.5	18.1	16.4
LJ	6.2	11.1	6.3	8.1	11.0	4.3	8.0
FS	5.4	8.9	15.7	15.9	11.0	11.0	16.1
CC	14.1	16.3	19.7	16.6	14.0	13.9	18.9
Mean	9.0	12.6	15.0	14.3	12.4	11.8	14.8
±SD	4.0	3.3	6.0	14.1	1.6	5.8	4.7
±SEM	2.0	1.6	3.0	2.1	0.8	2.9	2.4

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	14.9	11.1	13.8	19.9
LJ	6.6	4.1	5.3	6.0
FS	9.9	11.5	8.3	12.5
CC	17.6	18.6	10.0	19.4
Mean	12.2	11.3	9.4	14.4
±SD	4.9	5.9	3.6	6.6
±SEM	2.5	3.0	1.8	3.3

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVA (cont'd). Phase I

Urine Magnesium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	7.9	9.2	13.6	9.9	-	7.8	10.5	-	9.8 ± 2.2
LJ	1.3	12.5	2.2	3.3	8.8	16.4	9.8	14.5	8.6 ± 5.7
FS	-	8.5	11.0	6.9	8.3	-	8.9	-	8.7 ± 1.6
CC	8.9	6.2	6.0	3.9	-	-	9.1	9.9	7.3 ± 2.2
Mean									8.6
±SD									1.0
±SEM									0.5

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	9.3	6.5	11.4	14.7	10.9	4.8	1.1
LJ	5.4	10.3	12.0	5.4	0.5	5.9	-
FS	6.7	6.2	10.0	11.4	10.2	6.9	6.5
CC	9.5	6.4	8.3	9.6	7.1	5.0	-
Mean	7.7	7.4	10.4	10.3	7.2	5.6	-
±SD	2.0	2.0	1.6	3.9	4.7	1.0	
±SEM	1.0	1.0	0.8	1.9	2.4	0.5	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	5.1	3.9	17.0	11.8	9.2	16.0	11.1
LJ	8.6	5.2	3.6	2.3	5.2	5.6	5.3
FS	8.7	12.9	14.2	11.4	9.5	9.6	12.5
CC	9.8	7.4	14.2	11.1	4.0	8.4	9.9
Mean	8.0	7.4	12.2	9.2	7.0	9.9	9.7
±SD	2.0	4.0	5.9	4.6	2.8	4.4	3.1
±SEM	1.0	2.0	3.0	2.3	1.4	2.2	1.6

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	13.5	8.7	9.7	0.1
LJ	3.2	5.5	4.6	4.3
FS	6.0	10.7	17.3	7.4
CC	8.9	8.6	4.8	8.1
Mean	7.9	8.4	9.1	5.0
±SD	4.4	2.2	6.0	3.6
±SEM	2.2	1.1	3.0	1.8

[a]Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVA (cont'd). Phase I

Urine IPO_4 (mg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	1290	1364	1496	1665	-	1034	1465	-	1386 ± 216
LJ	166	992	84	442	582	2106	1163	823	795 ± 651
FS	-	928	1154	1191	701	-	1192	-	1033 ± 215
CC	1224	880	896	653	-	-	1277	1575	1084 ± 336
Mean									1075
±SD									243
±SEM									121

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	1540	1148	1632	1247	880	975	548
LJ	460	1260	1061	640	218	645	-
FS	1166	893	1419	890	905	978	723
CC	1584	1260	1231	1427	1248	1296	-
Mean	1188	1140	1336	1051	813	974	-
±SD	520	173	246	353	431	266	
±SEM	260	87	123	177	215	133	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	1206	1154	1406	1290	1128	2006	1190
LJ	977	893	336	576	699	783	552
FS	720	918	1020	983	960	966	1117
CC	1100	1144	1340	1181	941	1297	1209
Mean	1001	1027	1026	1008	932	1263	1017
±SD	209	141	490	314	177	539	312
±SEM	105	70	245	157	88	270	156

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	1251	1238	1650	1887
LJ	516	370	420	1066
FS	780	1088	1776	1050
CC	1300	1462	677	1501
Mean	962	1040	1131	1376
±SD	378	472	682	400
±SEM	189	236	341	200

[a] Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IVA (cont'd). Phase I

Urine Creatinine (mg/TV)

PRE-EXPOSURE CONTROL

Subject	6/24	6/25	6/26	6/27	6/28	6/29	6/30	7/1	Mean ± SD
WS	1858	2156	1987	1998	-	1254	2063	-	1886 ± 326
LJ	678	1696	370	832	1820	2356	1775	1142	1334 ± 682
FS	-	1833	1776	1878	1627	-	1937	-	1810 ± 119
CC	2124	1440	1456	1229	-	-	2016	2385	1775 ± 461
Mean									1701
±SD									249
±SEM									125

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WS	2244	1542	2244	1935	2332	2925	844
LJ	1180	2150	1612	918	474	1116	-
FS	2090	1380	2408	1362	1466	1842	1362
CC	2256	2282	1934	2088	2080	2016	-
Mean	1942	1838	2050	1576	1588	1975	-
±SD	514	441	352	538	827	744	
±SEM	257	222	176	269	413	372	

DECOMPRESSION

Subject	7/8[b]	7/9	7/10	7/11	7/12	7/13	7/14
WS	1891	1751	2052	2150	1728	2278	1734
LJ	1420	1488	622	1176	1251	928	926
FS	1485	1524	1830	1602	1530	1540	1756
CC	2156	2184	2251	2116	1926	1987	2108
Mean	1738	1736	1689	1761	1609	1683	1631
±SD	348	320	732	464	288	588	500
±SEM	174	160	366	232	144	294	250

POST-EXPOSURE

Subject	7/15[c]	7/16	7/17	7/29
WS	1850	1523	2130	2442
LJ	791	680	1162	1300
FS	1456	2048	2148	1804
CC	2052	2520	1152	2224
Mean	1537	1693	1648	1942
±SD	556	788	567	504
±SEM	278	394	284	252

[a] Exposure day 1 was 7/1 for subjects WS and FS; 7/2 for subjects LJ and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IVB. Effects on Urine Biochemical Parameters of Exposure to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

Urine Osmolality (mOsmo)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	730	533	528	483	724	687	-	672	622 ± 103
GM	988	928	909	849	-	-	-	-	918 ± 58
FS	1095	932	830	931	-	-	-	-	947 ± 110
CC	728	573	664	691	439	503	846	581	628 ± 130
Mean									779
±SD									178
±SEM									89

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	621	652	634	759	637	626	-	-	-	-
GM	375	649	719	676	659	562	712	546	770	555
FS	505	523	685	740	770	579	558	602	519	534
CC	408	522	582	620	455	525	-	-	-	-
Mean	477	586	655	699	630	573	-	-	-	-
±SD	111	74	60	63	131	42				
±SEM	55	37	30	32	65	21				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	901	581	559	697	491	538	878	1028	631	574
GM	705	508	583	865	573	845	931	753	900	812
FS	612	1052	1091	800	752	1011	869	742	976	684
CC	502	645	-	993	608	-	912	1062	715	600
Mean	680	696	744	839	606	798	898	896	806	668
±SD	169	244	300	124	109	240	29	172	160	107
±SEM	85	122	173	62	54	139	14	86	80	54

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	225	924	862	336	574	633	461	492
GM	914	877	870	965	860	861	885	839
FS	-	1221	-	1160	1034	605	610	903
CC	586	658	-	-	500	584	562	640
Mean	575	920	-	820	742	671	630	718
±SD	345	232		431	249	128	181	188
±SEM	199	116		249	124	64	91	94

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVB (cont'd). Phase II

Urine Sodium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	309	267	179	198	169	237	-	203	223 ± 50
GM	145	157	108	162	-	-	-	-	143 ± 24
FS	120	172	163	245	-	-	-	-	175 ± 52
CC	96	135	95	135	275	145	140	267	161 ± 71
Mean									176
±SD									34
±SEM									17

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	240	209	185	134	162	202	-	-	-	-
GM	55	156	195	255	122	211	116	216	139	168
FS	104	86	120	94	81	171	125	129	71	171
CC	105	95	73	93	208	193	-	-	-	-
Mean	126	136	143	144	143	194	-	-	-	-
±SD	80	57	57	76	54	17				
±SEM	40	29	29	38	27	9				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	197	230	161	58	181	97	122	115	242	215
GM	189	252	241	163	270	117	225	210	190	276
FS	58	73	53	66	108	61	144	137	131	150
CC	144	249	-	146	159	-	233	130	235	147
Mean	147	201	152	108	180	92	181	148	200	197
±SD	64	86	94	54	68	28	56	42	51	61
±SEM	32	43	54	27	34	16	28	21	26	31

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	192	56	100	79	155	210	246	201
GM	231	142	200	153	149	105	120	125
FS	-	101	-	131	128	181	109	55
CC	240	101	-	-	220	223	138	176
Mean	221	100	-	121	163	180	153	139
±SD	26	35		38	40	53	63	64
±SEM	15	18		22	20	26	32	32

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

TABLE IVB (cont'd). Phase II

Urine Potassium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	73	107	72	106	109	82	-	80	90 ± 16
GM	43	58	29	52	-	-	-	-	46 ± 12
FS	53	95	50	67	-	-	-	-	66 ± 20
CC	52	135	80	121	122	78	100	131	102 ± 31
Mean									76
±SD									25
±SEM									12

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	127	142	108	111	94	102	-	-	-	-
GM	27	58	141	124	75	125	80	137	60	74
FS	101	80	104	122	45	132	89	69	38	62
CC	47	105	94	122	123	99	-	-	-	-
Mean	76	96	112	120	84	114	-	-	-	-
±SD	46	36	20	6	33	16				
±SEM	23	18	10	3	16	8				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	97	99	87	34	125	104	99	77	92	95
GM	75	79	78	82	130	89	90	79	70	98
FS	42	64	41	62	81	62	66	65	60	51
CC	67	120	-	88	97	-	114	64	101	58
Mean	70	90	69	66	108	85	92	71	81	76
±SD	23	24	24	24	23	21	20	8	19	24
±SEM	11	12	14	12	12	12	10	4	10	12

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	63	37	76	47	48	63	52	81
GM	98	29	74	93	39	28	36	20
FS	-	42	-	48	22	41	53	56
CC	155	70	-	-	90	74	87	52
Mean	105	44	-	63	50	52	57	52
±SD	46	18		26	29	21	22	25
±SEM	27	9		15	14	10	11	12

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVB (cont'd). Phase II

Urine Chloride (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	189	242	171	188	156	216	-	195	194 ± 29
GM	148	172	112	143	-	-	-	-	144 ± 24
FS	99	198	162	247	-	-	-	-	176 ± 62
CC	88	169	100	152	245	145	157	302	170 ± 71
Mean									171
±SD									21
±SEM									10

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	255	242	162	115	135	185	-	-	-	-
GM	73	133	261	243	137	175	119	199	119	154
FS	91	106	143	89	64	161	112	121	70	166
CC	81	134	84	90	176	201	-	-	-	-
Mean	125	154	162	134	128	181	-	-	-	-
±SD	87	60	74	73	47	17				
±SEM	43	30	37	37	23	8				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	160	202	135	144	186	91	94	91	224	198
GM	317	211	219	149	263	131	166	164	134	227
FS	57	72	47	66	128	63	119	132	115	125
CC	132	269	-	155	187	-	211	128	235	162
Mean	166	188	134	128	191	95	148	129	177	178
±SD	109	83	86	42	55	34	52	30	61	44
±SEM	55	42	50	21	28	20	26	15	31	22

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	163	41	113	65	132	210	242	211
GM	189	70	203	148	146	104	124	68
FS	-	79	-	112	85	163	128	59
CC	240	75	-	-	244	212	189	169
Mean	197	66	-	108	152	172	171	127
±SD	39	17		42	67	51	56	75
±SEM	23	9		24	33	25	28	38

[a] Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b] Start of decompression for all subjects.

[c] Reach surface.

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TABLE IVB (cont'd). Phase II

Urine Calcium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	12.2	11.9	12.3	8.8	4.6	12.4	-	9.2	10.2 ± 2.9
GM	9.9	8.5	6.0	11.3	-	-	-	-	8.9 ± 2.2
FS	15.7	11.2	17.7	17.7	-	-	-	-	15.6 ± 3.0
CC	8.9	13.4	9.8	14.7	19.0	12.9	13.6	20.4	14.1 ± 4.0
Mean									12.2
±SD									3.2
±SEM									1.6

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	8.2	10.8	10.4	6.3	7.1	10.8	-	-	-	-
GM	9.7	18.6	13.1	16.6	6.3	18.3	8.3	12.8	8.9	7.0
FS	4.1	8.3	7.2	7.3	5.0	14.9	10.5	10.0	5.9	13.8
CC	7.6	9.9	12.1	13.7	12.5	12.6	-	-	-	-
Mean	7.4	11.9	10.7	11.0	7.7	14.2	-	-	-	-
±SD	2.4	4.6	2.6	5.0	3.3	3.2				
±SEM	1.2	2.3	1.3	2.5	1.6	1.6				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	10.0	10.6	7.0	12.0	15.9	8.8	8.7	6.4	10.5	6.6
GM	17.5	18.3	15.1	17.7	15.6	6.9	35.4	21.4	7.3	11.5
FS	6.3	9.8	9.6	10.8	11.0	7.2	11.7	11.0	12.8	9.9
CC	7.6	14.3	-	8.8	12.3	-	5.0	11.2	18.3	7.8
Mean	10.4	13.2	10.6	12.3	13.7	7.6	15.2	12.5	12.2	9.0
±SD	5.0	3.9	4.1	3.8	2.4	1.0	13.7	6.3	4.6	2.2
±SEM	2.5	2.0	2.4	1.9	1.2	0.6	6.9	3.2	2.3	1.1

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	12.1	5.4	7.2	6.9	7.2	6.3	9.4	7.7
GM	10.6	10.4	12.4	9.2	16.8	12.4	10.6	10.9
FS	-	14.3	-	6.0	9.1	11.1	4.6	6.7
CC	14.1	6.4	-	-	17.6	9.2	6.8	22.5
Mean	12.3	9.1	-	7.4	12.7	9.8	7.8	12.0
±SD	1.8	4.1		1.7	5.3	2.6	2.7	7.3
±SEM	1.0	2.0		1.0	2.6	1.3	1.3	3.6

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVB (cont'd). Phase II

Urine Magnesium (mEq/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	8.7	10.4	12.4	8.9	7.0	13.6	-	7.9	9.8 ± 2.4
GM	12.5	11.3	6.8	6.5	-	-	-	-	9.3 ± 3.0
FS	9.7	10.8	16.4	9.5	-	-	-	-	11.6 ± 3.2
CC	2.8	8.8	7.0	11.0	3.7	7.9	9.6	12.7	8.0 ± 3.4
Mean									9.7
±SD									1.5
±SEM									0.8

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	8.1	9.3	11.7	7.5	6.2	10.8	-	-	-	-
GM	4.7	1.6	9.3	14.0	4.1	14.9	7.8	9.5	7.5	1.7
FS	2.3	7.0	10.8	12.2	5.6	17.7	12.1	10.4	5.8	11.0
CC	0.1	4.5	7.0	5.5	0.2	0.2	-	-	-	-
Mean	3.8	5.6	9.7	9.8	4.0	10.9	-	-	-	-
±SD	3.4	3.3	2.1	4.0	2.7	7.7				
±SEM	1.7	1.7	1.0	2.0	1.3	3.8				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	10.7	10.8	8.4	17.4	10.4	7.3	7.1	7.9	9.4	5.1
GM	14.6	16.6	8.6	13.5	10.4	7.0	12.9	4.0	9.4	4.6
FS	6.2	10.1	8.6	9.1	6.0	5.9	7.7	7.6	8.7	6.4
CC	0.1	0.2	-	0.07	0.1	-	0.1	0.06	0.6	0.1
Mean	7.9	6.9	8.5	10.0	6.7	6.7	7.0	4.9	6.9	4.0
±SD	6.2	4.8	0.1	7.4	4.9	0.7	5.3	3.7	4.5	2.7
±SEM	3.1	2.4	0.06	3.7	2.4	0.4	2.6	1.8	2.2	1.4

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	6.9	7.8	7.8	5.6	1.6	6.7	0.8	1.6
GM	11.0	12.0	4.8	11.1	8.5	6.1	5.7	6.9
FS	-	0.03	-	0.05	1.6	2.0	0.07	0.04
CC	0.1	3.9	-	-	0.2	0.1	0.1	21.5
Mean	6.0	5.9	-	5.6	3.0	3.7	1.7	7.5
±SD	5.5	5.1		5.5	3.7	3.2	2.7	9.8
±SEM	3.2	2.6		3.2	1.9	1.6	1.4	4.9

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVB (cont'd). Phase II

Urine IPO_4 (mg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	1682	1189	992	1037	1058	1280	-	1079	1188 ± 238
GM	1037	990	780	1080	-	-	-	-	972 ± 134
FS	1260	1344	1114	1029	-	-	-	-	1187 ± 142
CC	775	1095	911	1392	1612	1136	1526	1380	1228 ± 297
Mean									1144
±SD									116
±SEM									58

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	1252	1238	1640	1586	1501	1323	-	-	-	-
GM	140	702	640	1938	627	2112	900	1968	1280	1050
FS	1140	1025	1415	1608	660	1463	1498	1069	733	1094
CC	620	750	1344	1610	1549	935	-	-	-	-
Mean	788	929	1260	1686	1084	1458	-	-	-	-
±SD	512	251	432	169	509	490			374	163
±SEM	256	125	216	84	255	245			187	81

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	1438	1333	1038	1216	1021	1102	1288	1292	1320	1295
GM	1600	1776	1064	1656	1450	1160	1456	846	1254	1680
FS	770	1188	912	992	903	1168	1160	1040	1428	1040
CC	701	1344	-	1126	1229	-	1259	820	1056	655
Mean	1127	1410	1005	1248	1151	1143	1291	1000	1264	1168
±SD	458	254	81	288	241	36	123	218	156	431
±SEM	229	127	47	144	120	21	62	109	78	216

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	444	1080	1386	936	480	990	408	621
GM	1534	1422	589	1404	578	444	585	1300
FS	-	636	-	1106	728	732	567	812
CC	1135	1206	-	-	1188	1096	540	770
Mean	1037	1086	-	1149	744	816	525	876
±SD	551	332		237	313	291	80	294
±SEM	318	166		137	157	146	40	147

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVB (cont'd). Phase II

Urine Uric Acid (mg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	1247	1353	800	768	1012	1185	-	909	1039 ± 228
GM	1088	918	696	1152	-	-	-	-	964 ± 204
FS	1560	1071	768	651	-	-	-	-	1012 ± 406
CC	530	966	874	1164	945	596	1293	1047	927 ± 260
Mean									986
±SD									50
±SEM									25

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	1576	837	1148	1074	1072	1361	-	-	-	-
GM	400	429	1120	1102	437	1056	560	864	780	630
FS	494	300	854	1080	528	961	1560	932	696	787
CC	471	690	812	837	1602	935	-	-	-	-
Mean	735	564	984	1023	910	1078	-	-	-	-
±SD	562	244	175	125	540	196				
±SEM	281	122	88	62	270	98				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	1066	817	1176	2400	1763	1555	805	731	520	595
GM	1280	1200	836	2352	1050	980	1664	701	950	868
FS	474	1188	710	880	710	743	720	632	1029	510
CC	935	2184	-	1233	1587	-	1421	780	1296	874
Mean	939	1347	907	1716	1278	1093	1152	711	949	712
±SD	341	586	241	776	485	418	462	62	322	187
±SEM	170	293	139	388	242	241	231	31	161	94

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	444	420	1056	468	600	690	592	854
GM	962	972	969	1656	765	624	780	832
FS	-	1056	-	1231	478	488	735	868
CC	1393	1151	-	-	704	1247	810	524
Mean	933	900	-	1118	637	762	729	770
±SD	475	328		602	126	334	97	164
±SEM	274	164		348	63	167	48	82

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE IVB (cont'd). Phase II

Urine Creatinine (mg/TV)

PRE-EXPOSURE CONTROL

Subject	7/31	8/4	8/5	8/6	8/7	8/8	8/9	8/10	Mean ± SD
MP	2117	2173	2144	2266	2185	2304	-	2215	2201 ± 66
GM	1870	1692	1200	1710	-	-	-	-	1618 ± 290
FS	1680	1722	1670	1470	-	-	-	-	1636 ± 112
CC	1129	2190	1655	2670	2391	1562	2114	2475	2023 ± 526
Mean									1869
±SD									289
±SEM									145

EXPOSURE

Subject	Day 1[a]	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
MP	2384	2330	2427	2001	2037	2268	-	-	-	-
GM	1140	1131	2528	2546	1140	2880	1440	2544	1680	1650
FS	1456	1450	1976	2160	924	2299	1810	1800	1354	1847
CC	1215	1710	2296	2351	2350	2255	-	-	-	-
Mean	1549	1655	2307	2264	1613	2426	-	-	-	-
±SD	573	508	240	236	688	304				
±SEM	286	254	120	118	344	152				

DECOMPRESSION

Subject	8/17[b]	8/18	8/19	8/20	8/21	8/22	8/23	8/24	8/25	8/26
MP	2058	2236	2076	2624	2227	2041	2001	1904	2120	1925
GM	2368	2256	2166	2232	2450	2000	2730	1836	2166	2744
FS	1184	1954	1286	1568	1935	1558	1900	1918	1826	1550
CC	1369	2240	-	2412	2509	-	2598	1760	2208	1404
Mean	1745	2172	1843	2209	2280	1866	2307	1854	2080	1906
±SD	560	145	484	456	260	268	418	72	173	600
±SEM	280	73	279	228	130	155	209	36	87	300

POST-EXPOSURE

Subject	8/27[c]	8/28	8/29	8/30	8/31	9/1	9/2	9/3
MP	1406	1200	1760	1656	1224	1980	1369	1746
GM	3952	2448	1900	2448	2210	1632	1482	2000
FS	-	1848	-	1847	1414	1366	1071	1330
CC	2477	1945	-	-	2244	2684	1740	1786
Mean	2612	1860	-	1984	1773	1916	1416	1716
±SD	1278	513		413	530	571	277	280
±SEM	738	256		239	265	285	139	140

[a]Exposure day 1 was 8/7 for subjects GM and FS; 8/11 for subjects MP and CC.

[b]Start of decompression for all subjects.

[c]Reach surface.

TABLE VA. Effects on Hematological Parameters of Exposures to Pressures Equivalent to 800 and 1200 fsw (Phase I)

Subject	Pre-Exposure Control				Post-Exposure			
	6/24	6/25	6/26	Mean ± SD	Day 1 7/15	Day 2 7/16	Day 3 7/17	Day 15 7/29
Mean Corpuscular Volume (cu micron)								
WS	87	82	85	84.6 ± 2.6	86	86	86	81
LJ	96	89	92	92.3 ± 3.5	92	95	92	87
FS	93	87	90	90.0 ± 2.9	93	94	93	87
CC	88	83	85	85.3 ± 2.6	88	87	87	81
Mean				88.1	89.8	90.5	89.5	84.0
±SD				3.7	3.3	4.7	3.5	3.5
±SEM				1.9	1.7	2.3	1.8	1.7
Mean Corpuscular Hemoglobin (gamma gamma)								
WS	28	29	29	28.7 ± 0.5	29	28	29	28
LJ	30	31	31	30.7 ± 0.5	30	30	31	31
FS	30	30	30	30.0 0	30	31	31	31
CC	28	28	28	28.0 0	28	28	28	28
Mean				29.4	29.2	29.2	29.8	29.5
±SD				1.2	1.0	1.5	1.5	1.7
±SEM				0.6	0.5	0.8	0.8	0.9
Mean Corpuscular Hemoglobin Content (%)								
WS	32	36	33	33.7 ± 2.1	34	34	32	35
LJ	31	35	33	33.0 ± 2.1	34	33	33	35
FS	31	35	32	32.7 ± 2.1	33	34	32	35
CC	31	34	32	32.3 ± 1.6	33	33	31	34
Mean				32.9	33.5	33.5	32.1	34.8
±SD				0.6	0.6	0.6	0.8	0.5
±SEM				0.3	0.3	0.3	0.4	0.3
Hematocrit (%)								
WS	44	42	43	43.0 ± 1.0	48	47	43	40
LJ	48	47	46	47.0 ± 1.0	45	45	44	45
FS	46	44	46	45.3 ± 1.2	49	49	46	47
CC	41	42	40	41.0 ± 1.0	44	42	43	39
Mean				44.1	46.5	45.8	44.0	42.8
±SD				2.6	2.4	3.0	1.4	3.9
±SEM				1.3	1.2	1.5	0.7	1.9
Hemoglobin (g/100 ml)								
WS	14.9	14.3	14.6	14.6 ± 0.3	16.1	15.8	14.2	13.8
LJ	15.8	15.6	15.2	15.5 ± 0.3	14.6	14.5	14.4	15.4
FS	15.3	14.5	15.1	15.0 ± 0.3	16.1	16.3	15.2	15.9
CC	13.6	13.7	13.2	13.5 ± 0.3	14.1	13.7	13.8	13.1
Mean				14.7	15.2	15.1	14.4	14.6
±SD				0.8	1.0	1.2	0.6	1.3
±SEM				0.4	0.5	0.6	0.3	0.7
Erythrocytes (x 10^6/cu mm)								
WS	5.2	4.9	5.0	5.0 ± 0.2	5.6	5.6	4.9	4.8
LJ	5.2	5.0	4.9	5.0 ± 0.2	4.8	4.8	4.6	4.8
FS	5.1	4.8	5.0	5.0 ± 0.2	5.3	5.3	4.9	5.1
CC	4.8	4.9	4.7	4.8 ± 0.2	5.1	5.0	4.9	4.6
Mean				5.0	5.2	5.2	4.8	4.8
±SD				0.1	0.3	0.4	0.2	0.2
±SEM				0.05	0.2	0.2	0.1	0.1
Reticulocytes (%)								
WS	1.3	2.0	1.3	1.5 ± 0.3	0.6	0.5	1.1	1.0
LJ	1.0	2 9	1.3	1.7 ± 1.0	0.8	0.7	0.2	2.9
FS	4.1	3.5	1.4	3.0 ± 1.4	1.8	0.2	1.0	4.2
CC	1.2	3.2	0.6	1.7 ± 1.4	1.4	0.4	0.2	1.0
Mean				2.0	1.2	0.4	0.6	2.3
±SD				0.7	0.6	0.2	0.5	1.6
±SEM				0.3	0 3	0.1	0 2	0.8
Platelets (x 10^3/cu mm)								
WS	185	174	199	186 ± 12	165	156	187	164
LJ	269	254	263	262 ± 7	185	169	213	189
FS	299	279	293	290 ± 10	276	266	323	249
CC	330	335	326	330 ± 5	212	194	200	221
Mean				267	210	196	231	206
±SD				61	48	49	62	37
±SEM				30	24	25	31	18
Leucocytes (x 10^3/cu mm)								
WS	5.0	6.1	6.0	5.7 ± 0.7	9.3	9.0	7.8	5.7
LS	6.5	6.2	6.8	6.5 ± 0.3	7.2	6.5	7.5	6.2
FS	5.8	6.1	6.9	6.3 ± 0:5	9.2	8.7	7.3	6.5
CC	4.5	4.9	4.3	4.6 ± 0.3	7.4	5.8	5.5	4.3
Mean				5.8	8.3	7.5	7.0	5.7
±SD				0.9	1.2	1.6	1.0	1.0
±SEM				0.4	0.6	0.8	0.5	0.5

TABLE VB. Effects on Hematological Parameters of Exposures to Pressures Equivalent to 800, 1200 and 1600 fsw (Phase II)

Subject	Pre-Exposure Control			Exposure	Post-Exposure		
	7/31	Day 1[a]	Mean	Day 3	Day 1 8/27	Day 3 8/29	Day 9 9/4
Mean Corpuscular Volume (cu micron)							
MP	93	-	93	88	84	88	-
GM	87	87	87	-	82	82	-
FS	92	94	93	-	89	89	-
CC	87	-	87	85	81	82	-
Mean			90.0	-	84.0	85.2	-
±SD			3.5		3.6	3.8	
±SEM			1.7		1.8	1.9	
Mean Corpuscular Hemoglobin (gamma gamma)							
MP	30	-	30	30	28	29	-
GM	28	27	28	-	27	28	-
FS	30	29	30	-	28	29	-
CC	28	-	28	28	26	27	-
Mean			29.0	-	27.2	28.2	-
±SD			1.2		1.0	1.0	
±SEM			0.6		0.5	0.5	
Mean Corpuscular Hemoglobin Content (%)							
MP	33	-	33	34	33	33	-
GM	33	31	32	-	32	33	-
FS	33	32	33	-	31	32	-
CC	32	-	32	33	32	32	-
Mean			32.5	-	32.0	32.5	-
±SD			0.6		0.8	0.6	
±SEM			0.3		0.4	0.3	
Hematocrit (%)							
MP	53	-	53	48	48	48	45
GM	47	46	47	-	42	43	42
FS	48	46	47	-	47	47	45
CC	42	-	42	43	42	41	38
Mean			47.2	-	44.8	44.8	42.5
±SD			4.5		3.2	3.3	3.3
±SEM			2.2		1.6	1.7	1.7
Hemoglobin (g/100 ml)							
MP	17.3	-	17.3	16.3	16.4	16.0	15.5
GM	15.1	14.3	14.7	-	13.9	14.8	14.3
FS	15.6	14.5	15.0	-	15.2	15.4	15.5
CC	13.3	-	13.3	14.2	13.5	13.5	13.0
Mean			15.1	-	14.8	14.9	14.6
±SD			1.7		1.3	1.1	1.2
±SEM			0.8		0.7	0.5	0.6
Erythrocytes (x 10^6/cu mm)							
MP	5.7	-	5.7	5.5	5.7	-	-
GM	5.4	5.3	5.4	-	5.1	5.0	-
FS	5.2	4.9	5.0	-	5.3	-	-
CC	4.8	-	4.8	5.1	-	-	4.7
Mean			5.2	-	5.4	-	-
±SD			0.4		0.3		
±SEM			0.2		0 2		
Reticulolytes (%)							
MP	4.1	5.0	4.6	4.0	3.7	3.0	3.0
GM	0.4	0.5	0.4	2.6	1.0	0.6	0.9
FS	1.2	1.4	1.3	2.4	1.8	2.0	2.9
CC	1.5	3.4	2.4	4.4	2.2	0.8	3.4
Mean			2.2	3.4	2.2	1.6	2.6
±SD			1.8	1.0	1.1	1.1	1.1
±SEM			0.9	0.5	0.6	0.6	0.6
Platelets (x 10^3/cu mm)							
MP	171	200	186	251	194	208	351
GM	224	214	219	303	288	288	355
FS	252	204	228	265	301	260	314
CC	114	191	152	159	232	244	277
Mean			196	244	254	250	324
±SD			35	61	50	33	36
±SEM			17	30	29	17	18
Leucolytes (x 10^3/cu mm)							
MP	8.7	-	8.7	7.4	10.5	8.0	11.4
GM	5.8	6.1	6.0	-	8.5	5.0	5.5
FS	8.4	5.7	7.0	-	10.6	5.7	6.0
CC	4.4	-	4.4	7.6	7.3	4.9	4.1
Mean			6.5	-	9.2	5.9	6.8
±SD			1.8		1.6	1.4	3.2
±SEM			0.9		0.8	0.7	1.6

[a]Blood sample drawn at sea level before start of compression on exposure day 1. 8/7 for subjects FS, GM; 8/11 for subjects CC, MP.

F. UNDERWATER WORK PERFORMANCE AT HIGH PRESSURE

F. PRACTICAL UNDERWATER WORK PERFORMANCE AT PRESSURES TO 1200 AND 1600 FSW

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As part of the integrated physiological investigations of the Predictive Studies IV program, the effects of progressive rapid compression to 400-800-1200 and 1600 feet of sea water were explored (9).

At the time of this study, in 1975, repeated exposures of man had been conducted at rest and with various forms of physical exercise in the compressed helium atmosphere, but with little extension to determining capability for practical underwater work. The exercise accomplished in the "dry" chambers at high helium pressures had included arm and leg activity (7,11,13,14) and moderate-to-severe work with an ergometer bicycle (8,15). Studies of arm ergometer exercise had been conducted in subjects sitting submerged in water at pressures equivalent to 500 and 600 fsw (6). However, performance of practical forms of work underwater, even within experiment chambers, tended to be avoided at high pressure due to concern over its feasibility. These concerns related to influences of respiratory stress, CO_2 retention and neurological effects of compression. One study using underwater ergometer leg exercise was carried out in tests of breathing apparatus by the U.S. Navy Experimental Diving Unit in a water-filled plastic box surrounded by a helium-oxygen atmosphere at a pressure of 1600 fsw (16,17). It is surprising that, while studies to such

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high pressures in dry chambers and some nonworking ocean penetrations to 1148 fsw (2) were proceeding, a 1967 open-sea saturation diving trial to 615 feet, with working excursions to 636 fsw (12), remained the deepest demonstration of practical work performance at high pressure underwater (whether in laboratory chambers or in the sea) through the eight years to the time of this Predictive Studies IV.

The intent of the present program was to integrate specific physiological and performance investigations with a normal second step toward applying such observations. This step entails performing in a laboratory ocean simulator the degree and technical form of work ultimately to be carried out beneath the sea. Therefore, when it was learned that the subjects exposed to rapid excursion and saturation pressurization maintained physiological competence, the trials of practical work performance in water were carried out in a manner designed to simulate actual underwater work in diving.

It was evident that, with the lack of prior trials of work underwater at the pressures contemplated for Predictive Studies IV, no major base of experience existed to aid such trials.

Since the primary physiological investigative plan of purposely rapid excursions was to induce prominent effects of compression for study of onset and adaptation, until such detailed information was obtained in the dry helium environment it could not be certain whether the desired trials of practical work underwater would be sensible. However, to make provision for such trials it was necessary to establish in advance the conditions of work, to adapt breathing and safety systems, to accomplish technical and procedural training of subjects and investigators, and to make measurements of the respiratory and metabolic demands imposed by the planned underwater work functions. The preparations were accomplished prior to the beginning of rapid compression for the prolonged saturation-excursion studies of Phase II. Further training for the ultimate underwater work on excursion to the highest pressure of 1600 fsw was carried out at the stable elevated chamber pressure of 1200 fsw. Additional trials were performed at a helium pressure of 1350 feet during saturation-decompression.

METHODS

SUBJECTS AND CIRCUMSTANCES OF UNDERWATER WORK

The practical work task was accomplished by each of the four trained diver-subjects of Phase II (CC, GM, MP, FS). Figure 1 illustrates the general arrangement of chamber systems, subjects and the underwater work station used both for training and for the subsequent underwater work performance trials at the several levels of increased pressure. Since the diver-subjects worked in the chamber at a water depth of 10 to 12 feet, the underwater work trials were performed at pressures of 1210, 1360 and 1610 fsw.

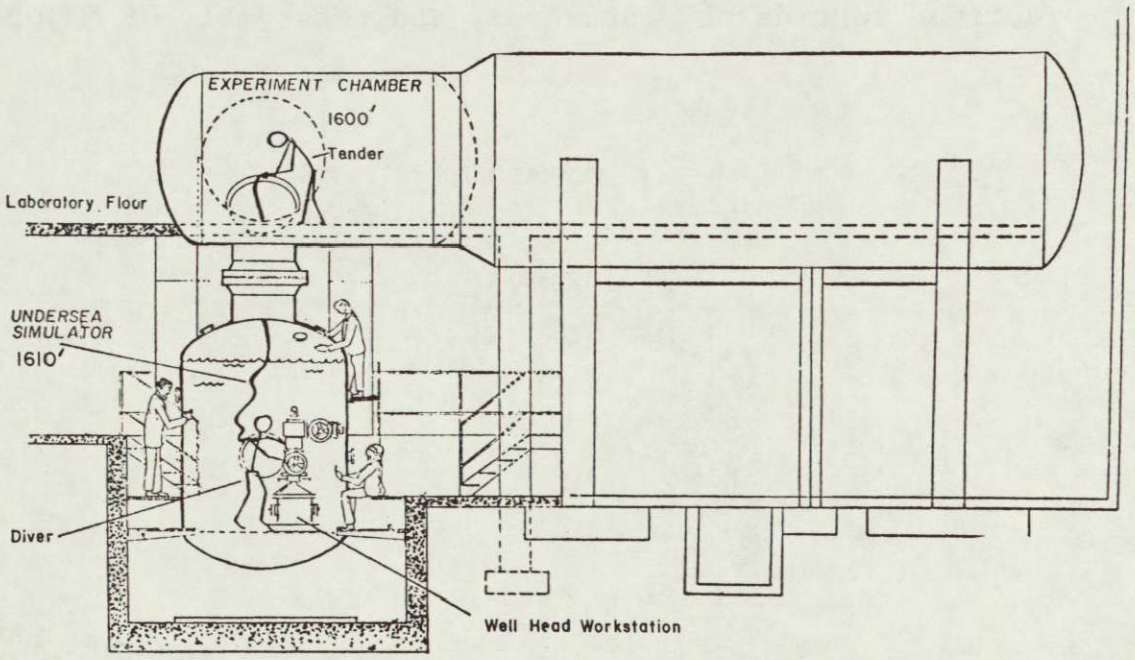

FIG. 1. Use of Water-filled Undersea Simulator Chamber System for Underwater Work Performance. Experiment Chamber pressurized with respirable helium-oxygen provides pressure on water surface of Undersea Simulator to simulate hydrostatic pressure in deep diving. Connecting Trunk is provided with stowable ladders.

The figure also indicates the positions of Tender and Diver-Subject relative to the Work Station during the underwater activity. The Diver-Subject and Tender compressed in a helium-oxygen atmosphere in the "Experiment Chamber" to the desired ambient pressure (e.g., equivalent to 1600 fsw). The Diver-Subject was secured by a harness, safety line,

gas hose and communication wire as he descended a stowable ladder into the water of the "Undersea Simulator," breathing helium with oxygen by mask. The timed sequence of tasks was carried out, following which the diver returned to the experiment chamber. Throughout the study the diver and tender were under continuous direct visual observation, as well as video and audio monitoring.

WORK STATION AND WORK SEQUENCE

An assembly of subsea oil wellhead components (Fig. 2) was designed (McEvoy Oilfield Equipment Company) and installed in the water-filled compartment of the undersea simulator system. The divers followed a prescribed and practiced routine of disassembly and reassembly of major

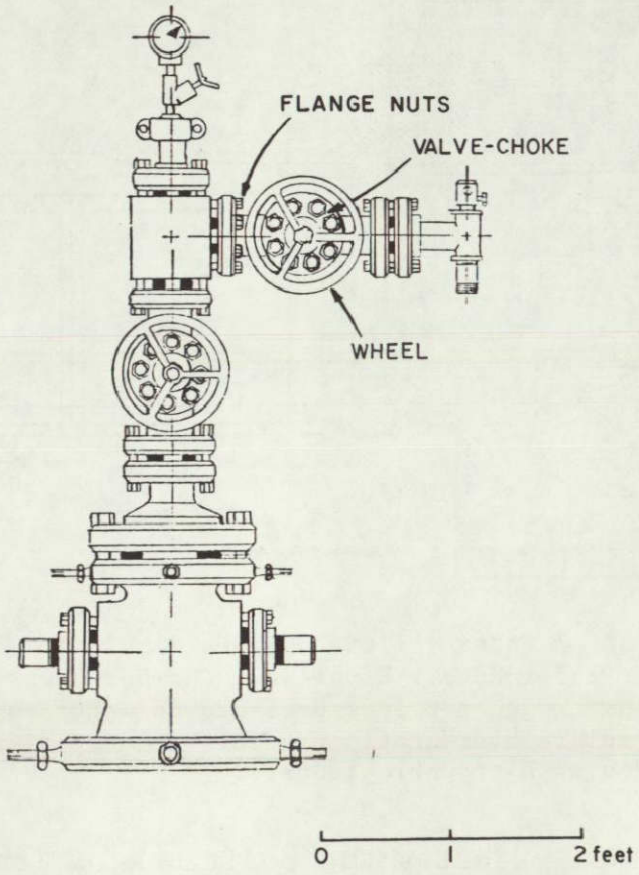

FIG. 2. Underwater Work Station. The assembly of high pressure fittings was devised by oil and diving industry engineers to provide appropriate task sequences for underwater work. Diver-subjects rigged, unbolted and removed valve-choke, then reassembled the system and removed the lifting rigging.

components, carrying out a timed sequence of work procedures lasting approximately ten minutes from start to finish. Table I shows the pattern of specific steps in disassembly and reassembly of the work station components. Tools employed included davit, winch, sling and wrench. Each diver-subject had accomplished at least five trials in

TABLE I. Pattern of Stages of Underwater Task Sequence. Percentages of Total Time Required to Perform Each Stage Are Based on Measurements Made at 10 fsw. (Mean Values From Four Subjects)

Task Stage	Components	% of Total Time
Rigging	Touch bottom Receive davit Assemble davit on well-head assembly Remove valve wheels Shackle hand winch ("come-along") to davit boom Rig wire cable winch sling to valve-choke body Apply tension with winch	18
Unfastening	Loosen valve-choke assembly nuts with wrench Remove all nuts to container	21
Disassembly-Reassembly	Remove valve-choke assembly Rig valve-choke assembly for surface lift Unrig for surface lift Replace valve-choke assembly Replace nuts, by hand	24
Torquing	Torque nuts with wrench	23
Completion	Release winch tension Remove sling Remove davit Rig davit for surface lift Replace valve wheels Finish	14
	Total	100%

water prior to excursion-saturation exposure and following training and practice in an air environment. The times required for completion of the task by each diver prior to pressurization are shown in Table II.

TABLE II. Times Required to Complete Underwater Work Task at 10 fsw

Subject			
CC	GM	MP	FS
10:20	9:59	10:07	8:37
11:00	11:06	11:37	9:36
11:10	12:08	11:48	10:14
15:08	12:55	11:53	11:37

Total times (min : sec) given for each subject are his four best attempts made during an initial trial sequence. These baseline values are compared with the subjects' performance at the high pressure circumstances in Table IV.

BREATHING APPARATUS

Helium with oxygen (1% O_2 at 1200 fsw, 2% O_2 at 1600 fsw) was the respired gas provided by open-circuit breathing systems which had been adapted to assure a sufficient free flow of gas at the highest ambient pressure utilized so that equipment-related resistance to inspiration would be avoided. Since the objective of the study was to determine human capability rather than equipment performance, it would have been undesirable to add apparatus resistance to the intrinsic pulmonary airway resistance associated with breathing helium at densities of 6.34, 7.24 and 8.92 g/l. It is well-recognized that superimposition of equipment resistance must limit ventilatory performance (16,17).

TEMPERATURE

Water temperature was maintained at 33°C (92°F) with no attempt to simulate the low water temperatures of the deep ocean since the intent was not to study temperature stress, but to determine whether hydrostatic influences would affect work ability. This design was further related to the awareness that means of heating divers in ocean water are now improving.

DEGREE OF WORK AND VENTILATORY RESPONSE

Respiratory minute volume was measured continuously during the practical work trials performed both in air and in the water-filled chamber at one ata pressure, using the breathing apparatus as part of the system for collecting the expired gas for volume measurement and analysis.

Repeated determinations of oxygen consumption were made in the four subjects during preliminary training in an air environment, the work chamber being empty of water. Figure 3 indicates the findings for the five task stages performed in air. Technical difficulties related to volumetric gas collection interfered with accuracy of initial measurements of ventilatory and metabolic cost of work underwater. For this reason one subject (FS) was studied repeatedly in air and in water following the Phase II saturation-excursion study, providing the necessary direct comparison of these conditions (Table III). From respiratory minute volume, inspired gas composition and continuous measurement of expired oxygen and carbon dioxide concentration, it was possible to derive a continuous indication of metabolic oxygen consumption and carbon dioxide production (Fig. 4). These measurements are used as indices of work pattern and degree of work for the standard task sequence employed at the increased ambient pressures.

TABLE III Ventilatory and Metabolic Costs of Work During Performance of Work Task Sequence in Air at 1 ata and in Chamber With Water Depth 10 fsw (Subject FS)

	Rest			0-2 min			2-4 min			4-6 min			6-8 min			8 - End		
	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)	$\dot{V}_E$ (l/min BTPS)	$\dot{V}_{O_2}$ (l/min STPD)	$\dot{V}_{CO_2}$ (l/min STPD)
SUBJECT IN AIR AT 1 ATA																		
Trial 1	9.86	0.39	0.33	46.33	1.63	1.44	58.10	1.99	1.76	55.87	1.99	1.65	76.33	-	-	-		
2	8.78	0.34	0.29	50.64	1.65	1.46	60.95	1.93	1.67	66.82	1.99	1.76	80.35	2.45	2.23	-		
3	21.92	0.69	0.56	55.88	1.72	1.46	66.30	1.98	1.78	59.72	1.79	1.60	67.06	2.11	1.86	-		
Mean	13.29	0.47	0.39	50.95	1.67	1.45	61.78	1.97	1.74	60.80	1.92	1.67	74.58	-	-			
SUBJECT UNDERWATER 10 fsw																		
Trial 1	15 74	0.56	0.56	44.85	1.70	1.38	62.90	1.75	1.72	60.38	1 76	1.61	71.90	2.41	2.04	88.66	2.63	2.65
2	14 01	0.47	0.46	48.10	1.67	1.38	55.42	1.79	1.54	60.68	1.88	1.74	63.36	2.26	1 84	78.31	2.43	2.24
3	9 16	0.36	0.33	46.70	1.56	1.37	57.66	1.85	1.67	57.12	1.85	1.59	52.95	1.71	1.46	62.60	2.36	1.90
4	10 25	0.38	0.35	50.79	1.66	1.39	58.92	1.74	1.47	56.60	1.73	1.43	76.97	2.68	2.12	-	-	-
Mean	12.29	0.44	0.43	47.61	1.65	1.38	58.73	1.78	1.60	58.70	1.80	1.59	66.30	2.27	1 87	76.52	2.47	2.26

RESULTS

WORK AND VENTILATION

The metabolic and the ventilatory cost of performing the underwater task sequence (Figs. 3 and 4) were intentionally allowed to be greater than would have been appropriate for effective, sustainable practical work functions in actual open sea diving. Both in degree and in duration they much exceeded the underwater ergometer leg exercise more recently performed at 1400 fsw (3).

Ventilation reached approximately 60 liters per minute at the mid-point of the work period and increased further in the more strenuously paced activity involved in final reassembly of the work station components. The oxygen consumption averaged approximately 1.8 1/min at mid-point in subject FS and increased to 2.2 1/min in reassembly. These indices reflect not the task difficulty but the prominent degree and rate of exertion self-selected by the subjects to compete with their previous performance and task completion times.

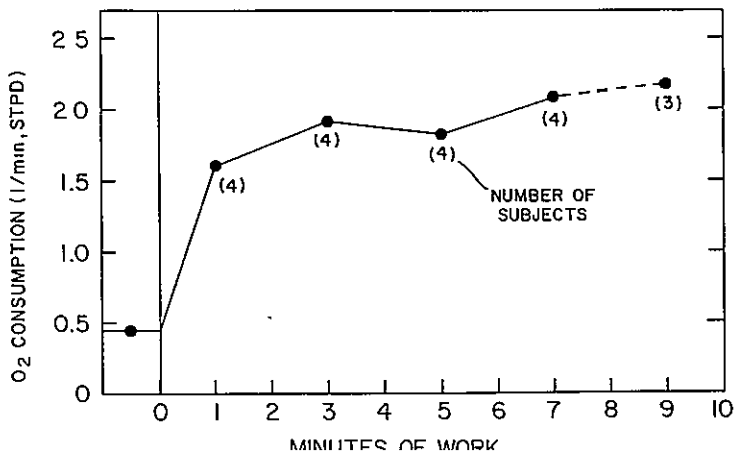

FIG. 3. Metabolic oxygen requirement of 2-minute periods in performance of underwater work sequence. Mean values of four subjects measured in air at 1 ata.

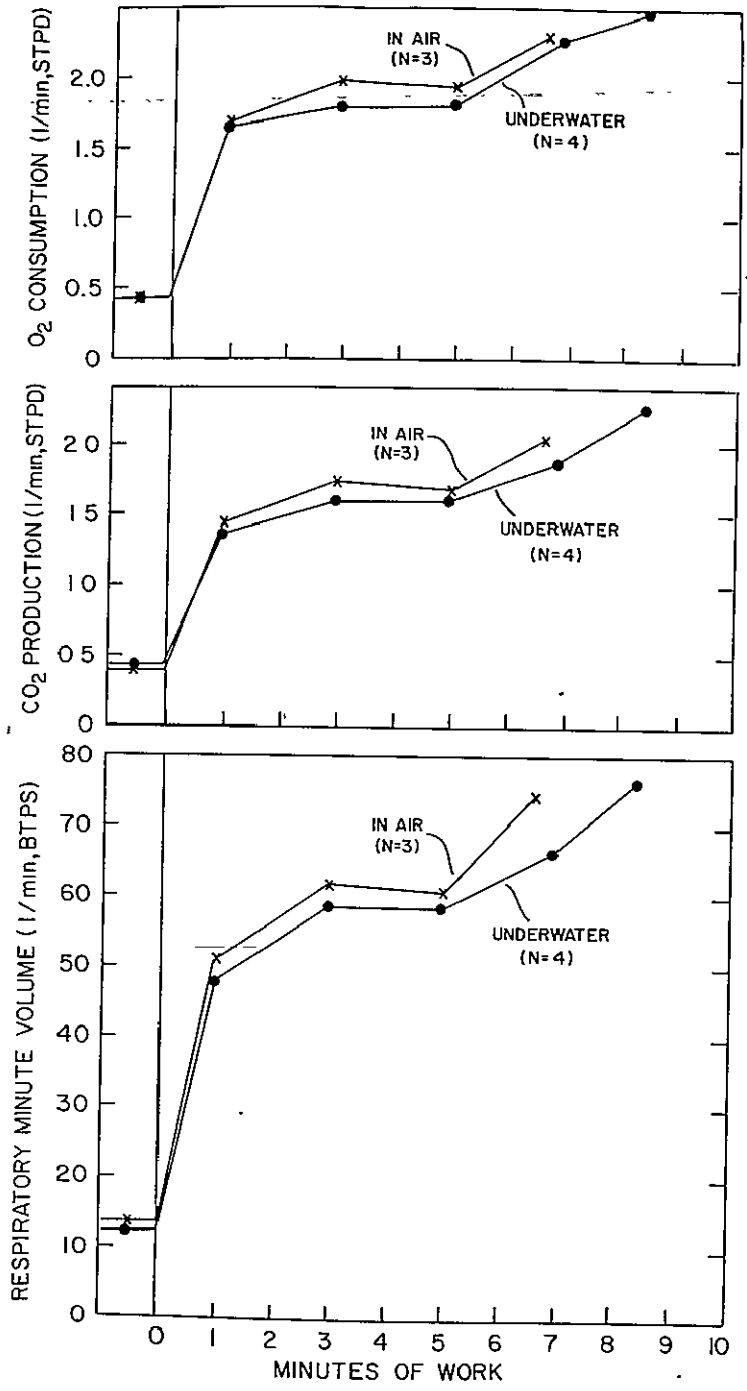

FIG. 4. Ventilatory and metabolic costs of work during performance of work task sequence. All measurements on subject FS: three trials in air at 1 ata and four trials underwater (10 fsw).

Metabolic rate was slightly diminished and time required for completion increased by work in water. Overall energy requirement was equivalent. Respiratory minute volume was slightly less in water than in air, matching the corresponding metabolic changes.

INFLUENCES OF WORK AND WATER

In Fig. 4 the effects of work in air and in water are shown to be not markedly different. The slightly lower oxygen consumption, carbon dioxide production and ventilation during work underwater evidently reflects the slightly lower rate of work imposed by adaptation to the water medium, and is consistent with the slightly longer time required to complete the total task sequence underwater.

PERFORMANCE AND TASK COMPLETION TIME

The specific work activities carried out in water by the Diver-Subjects included extensive movement and positioning of the body necessary to accomplish the tasks, fine finger activity and coordinated limb movement, skilled operation of the mechanical devices used to remove a heavy component, and the prominent exertion required for the self-paced overall task.

These specific performance functions, examined in shallow water by others (4), were photographically recorded here but not analyzed in detail. Table IV and Fig. 5 show the work duration required by each Subject-Diver to complete the total task sequence at each of the pressures used, from pre-compression to the excursion from 1200 fsw for underwater work at 1610 fsw. For each subject pair the first underwater work trial during the Phase II compression-pressure exposures was at a stable pressure of 1210 fsw, which represents essentially twice the prior "depth" for practical work underwater at increased pressures. In each case it followed the two successive daily excursion-compressions for detailed prior physiological measurements; this underwater work occurred on day 4 from the beginning of compression for each pair. It was therefore carried out in a period when considerable adaptation to the initial compression had been accomplished. No evident differences in symptomatic or objective responses to underwater work were observed between conditions of 1200 fsw and sea level.

The second underwater work sequence, performed on excursion from 1200 to 1610 fsw (Fig. 6), occurred for Subjects CC and GM on their compression day 5, and for Subjects GM and FS on their compression day 9. Except for controlled excitement at entering an entirely new situation, Subjects GM and FS

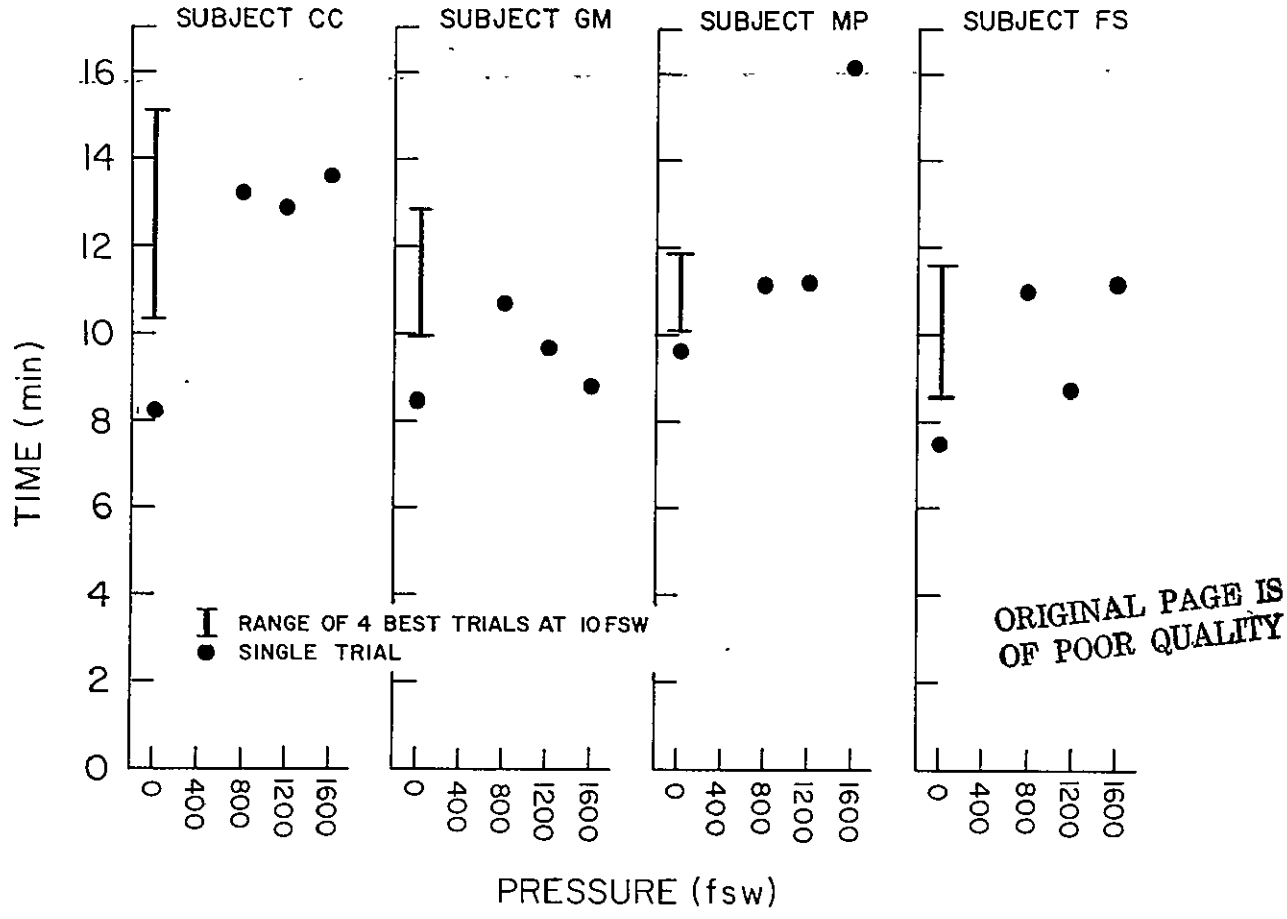

FIG. 5. Time for completion of underwater work task. Total times were generally not different at stable increased pressures compared with range of four trials at baseline condition of 10 fsw. Subject MP, however, had specific difficulty at 1610 fsw in aligning heavy valve flange with its stud fastenings.

experienced no unusual reactions and performed the practical underwater work and maneuvers in a manner equivalent to that demonstrated in water at 10 feet, under one atmosphere.

Immediately on compression from 1200 to 1600 fsw, subject CC felt an "anxiousness" and was allowed to conduct his underwater work on the same 55-minute excursion but following subject MP. Subject MP encountered a technical difficulty related to misalignment of the valve flange with the studs of the work station, which both extended his time (Table IV and Fig. 5) and led him to strive with greater than previous exertion before succeeding in reassembly and completing the task. Subject CC then proceeded with a smooth work performance.

TABLE IV Influence of Pressure and Conditions Upon Work Task Completion Time (time in min:sec). Underwater work at 10 fsw should be considered the baseline for comparison with the increased pressure states.

Condition	Subject			
	CC	GM	MP	FS
Air, 1 ata	8:16	8:30	9:40	7:31
Underwater, 10 fsw[a] (Pre-compression)	10:20-15:08	9:59-12:55	10:07-11:53	8:37-11:37
Underwater, 1210 fsw (at stable pressure)	13:14	10:45	11:10	11:02
Underwater, 1360 fsw (during saturation-decompression)	12:56	9:42	11:12	8:43
Underwater, 1610 fsw (during 1200-1600 fsw excursion)	13:40	8:48	16:09[b]	11:10

[a] Range of four best trials for each subject.

[b] Subject-diver MP encountered great difficulty when an unequal positioning of the winch sling interfered with the alignment of the flange holes of the heavy valve-choke assembly with the studs, grossly increasing work and duration.

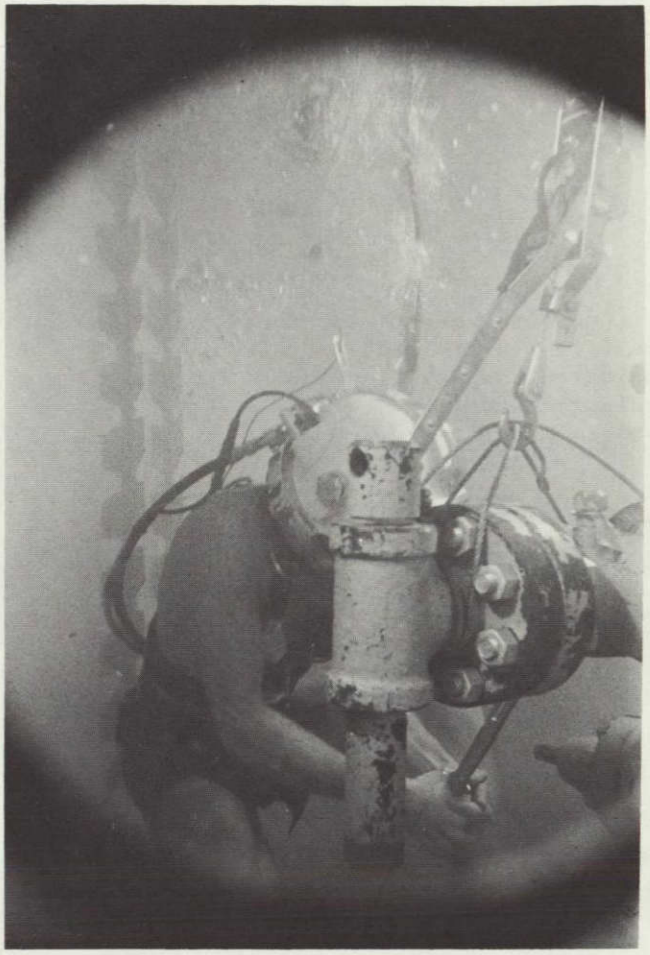

FIG. 6. Subject-Diver performing task sequence at 1610 fsw pressure equivalent, on excursion from 1200 fsw.

The third underwater work sequence was performed at the 1360-fsw pressure during a stable phase of saturation-decompression, without excursion, to allow increased underwater working experience. No evident symptomatic or objective abnormalities occurred, and the work performance was generally equivalent to that at sea level.

BREATHHOLDING

As part of a general appraisal of respiratory control functions at increased ambient pressure, simple breathholding time was determined. Measurements were made without prior

tremor occurred in two subjects on rapid compression from 1200- to 1600-fsw pressure even after several days at 1200 fsw (Section E-1) and may have slightly affected the quality of work performance. Underwater trials on an earlier day might have generated a definite degree of interference with practical work. This is uncertain, but likely.

Clearly, in addition to duration of adaptation time, both the magnitude and rate of compression can be expected to determine the time course and completeness of the adaptations. These factors therefore should influence the delay required at a high pressure before effective and safe performance in water can be expected. This study has indicated that such delay falls within practical limits.

Following the practical demonstrations in this study and the physiological observations which led to them, it should be considered entirely feasible to carry out detailed investigation of underwater exercise and practical work. Studies of underwater ergometer exercise have recently been extended to stable pressures of 1000 fsw for arm activity (5), and 1400 fsw for leg work(3), and should be followed by investigation of other forms of work and other compression environments.

For the present, while continuing specific studies, it is important to recognize that skilled work has in fact been performed at 1610 fsw, and at sustained rates of physical exertion and ventilation greater than should ordinarily be necessary in the undersea circumstance simulated. In the performance of this underwater work the prominent dyspnea, reported in other investigations (3,17) in which subjects were continuously maintained at a stable 1400- or 1600-fsw pressure, was not encountered.

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G. DECOMPRESSION PROCEDURES AND THERAPY OF DECOMPRESSION SICKNESS

G-1. Decompression from Excursion Exposures

G-2. Decompression from Saturation Exposures

G-3. Ultrasonic Detection of Venous Bubbles in Decompression from Deep Excursion and Saturation Exposures

G-4. Therapy of Decompression Sickness

G-1. DECOMPRESSION FROM EXCURSION EXPOSURES

R.E. Peterson[1], K.M. Greene[1] and C.J. Lambertsen[1]

The experiment program of Predictive Studies IV required essentially daily, repetitive excursions from 800 to 1200 fsw (Phase I) and from 1200 to 1600 fsw (Phase II). Compression rates averaging from 5 to 40 feet/minute, with minimum times of 30 minutes for experiments at the maximum pressures, were anticipated for these 400-fsw compressions. Since suitable decompression schedules for such excursions did not exist, it was necessary to develop safe, rapid procedures in order to make the planned research program possible. This development required practical results with a minimal amount of testing and no effort to obtain information concerning decompression sickness thresholds.

Since decompression obligations for excursion diving from saturation states have been shown to decrease significantly as the saturation depth increases (1,6,13), it was anticipated that a safe schedule capable of meeting the program's needs could be devised. It was also expected that the decompression times for the planned excursions at high pressures would be considerably shorter than for similar excursions from sea level.

Procedures were devised which provided for the exceptionally rapid decompression required. In accordance with the purposes and plans of Predictive Studies IV, Phase I was employed for the controlled testing of the decompression method.

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BACKGROUND

In previously designed methods of saturation-excursion diving, compression times and "bottom times" were too short, saturation and excursion depths too shallow and/or decompression times too long to be of use in the experimental program of this Predictive Study. Approaches considered for the development of new decompression schedules included: 1) to employ without modification an existing, widely used decompression computation method, extrapolating to satisfy the needs of the study; 2) to use a standard method, modified by use of calculation parameters derived from diving experience relevant to study needs; or 3) to develop a new approach or modify an existing one, using new information available.

Widely used methods considered for direct application included those of the U.S. Navy (9, 15-18), of Buhlmann (5) and of Hamilton et al. (7). Gross extrapolation of computation procedures would have been required to devise deep excursion schedules from any of these, and specific features of each limited its applicability to the special purposes of the study.

An existing decompression computation method could incorporate recent information obtained from the "no-decompression"-excursion experiments conducted by the U.S. and British Navies (1,6). Based on these experiments, the maximum calculated tissue supersaturation[2] (ΔP) for

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[2] The amount of excess inert gas contained in a tissue is a function of, among other factors, the partial pressure of the inert gas in the tissue and the ambient pressure. ΔP, in a strict sense, is the difference between the tissue pressure of a gas and the ambient pressure; if all the gas remains in solution in a state of supersaturation, ΔP and excess inert gas are directly related. While the computation of values of ΔP was made with the above definition, the conceptual analysis presented here and in the section on decompression from saturation exposures (G-2) relating ΔP and time is believed to be independent of the actual state of any excess inert gas in a tissue. Whether the excess gas is in a state of true supersaturation, or there is phase separation, or a combination of the two states exists, it is considered that the longer the conditions of excess inert gas exist, the greater is the potential for bubble formation and growth. The term and values for ΔP, therefore, are used here in a broad sense as an index of the degree of any excess inert gas in a tissue.

oxygen-helium diving which did not produce decompression sickness should follow the relationship $5.4\sqrt{D_{abs}}$ for depths (D) from sea level to 600 fsw. This relationship, derived by a least squares regression analysis of the available data from Royal Naval Physiological Laboratory and U.S. Navy Experimental Diving Unit (EDU) no-decompression excursion results, is shown in Fig. 1. Based on EDU findings, a helium ΔP of 136 fsw could be tolerated at 600 fsw without development of decompression sickness. This value corresponds to the maximum ΔP sustained as the result of a 24-hour exposure to a helium-oxygen atmosphere at 750 fsw with a direct ascent to 600 fsw at the rate of 60 feet/minute.

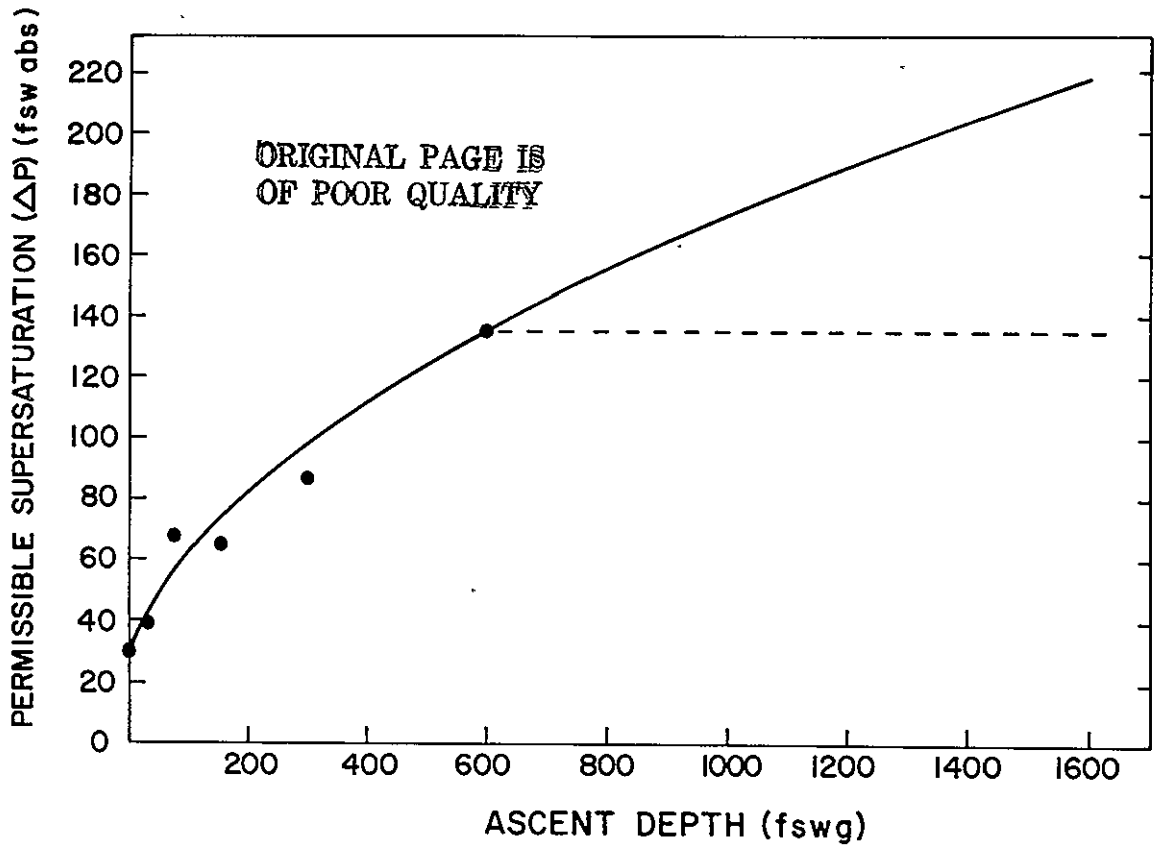

FIG. 1. Maximum permissible supersaturation (ΔP) as a function of ascent depth for oxygen-helium diving used for decompression computations in this study. Individual points represent data derived from single-step decompressions following 24-hour exposures (1,6). The power function ($\Delta P = 5.4\sqrt{D_{abs}}$) is the best least squares fit to these data (solid line). The ΔP at 600 fsw was used at all greater depths (dashed line) since no information for greater depths was available at the time of the study.

While the above empirical relationship is valid for no-decompression dives, its use to determine M-values (20) for computation of stepwise (stage) decompressions was considered an unwarranted extrapolation of the data. If used to compute stage decompressions, extended exposure times to large ΔPs could result (Fig. 2), whereas in the no-decompression ascents the maximum ΔP is maintained only for brief periods, with exponential decrease beginning immediately. It has been hypothesized that this increased duration of exposure to the maximum allowed ΔPs in stage decompression increases the probability for bubble formation and growth (10). It was therefore decided to use a new approach initially taking excess pressure and time into account to compute decompressions for the deep excursions of this simulated dive program.

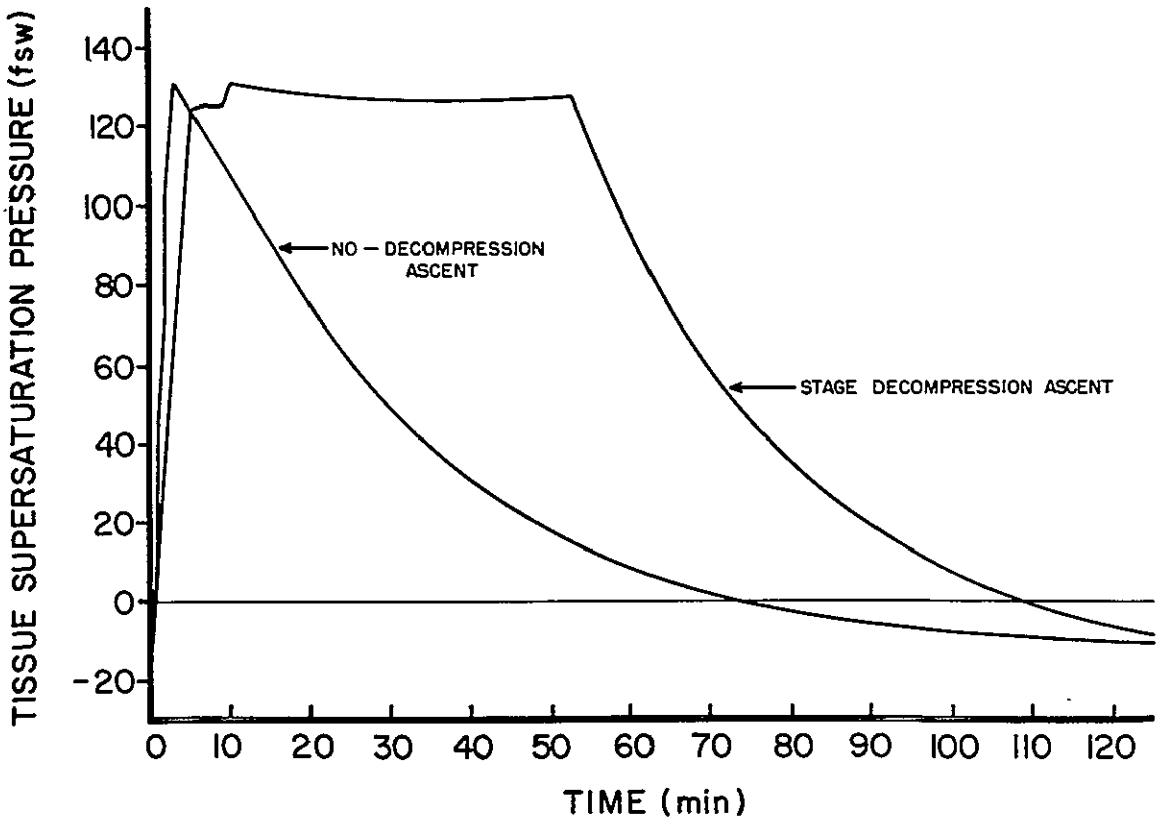

FIG. 2. Theoretical tissue inert gas supersaturation in "no-decompression" and stage decompression ascents (compartment half-time = 20 minutes). Due to the presence of oxygen, compartment inert gas pressure eventually falls below ambient pressure.

BASIC APPROACH

The computation system for a decompression schedule generally consists of two components: a gas exchange model and a pattern of ascent constraints. The approach used in these studies involved a standard multi-compartment exponential model (4,11,20) based on perfusion-limited inert gas exchange (8,11) with tissue gas exchange half-times extending from a 1:1 to 2.8:1 nitrogen:helium ratio (Table I).

TABLE I. Inert Gas Exchange Half-Times Used in Computing Decompression Schedules

Compartment Number	1	2	3	4	5	6	7	8	9	10	11
Nitrogen half-times (min)	5	10	30	60	100	150	200	260	320	480	670
Helium half-times (min)	5	10	20	40	60	80	100	130	160	200	240

The ascent constraint for decompression was based on considerations of the ΔP-time relationships for each compartment during the course of the decompression (10), rather than a standard maximum tolerable inert gas pressure (M-value) (20) or ratio of tissue inert gas pressure to ambient pressure (4). As applied here, this constraint requires that $\int \frac{\Delta P(t)}{\sqrt{D(t)_{abs}}}\, dt$ and the maximum ΔP for each compartment should not exceed the same values computed for the EDU experimental dives with direct ascents from 750 to 600 fsw and from 450 to 300 fsw (6).

Values of ΔP were weighted according to the square root of the absolute depth based on the British and U.S. Navy no-decompression data discussed previously. This weighting scheme (which reduced the significance of any ΔP as the depth at which it occurred increased) was limited, however, to a depth range from sea level to 600 fsw. The limit was imposed since, at the time of Predictive Studies IV, testing of these basic concepts at greater depths had not been undertaken and it was considered unwise to extrapolate beyond this depth. Thus no depth-weighting factor greater

than $\sqrt{633}$ was used in these computations, which were made with the University of Pennsylvania's decompression calculation and analysis program (PADUA) (2). This program was modified to compute the cumulative ΔP-time function with the ΔP a linear function of time and weighted by the square root of absolute depth. Decompressions were computed on the basis of a maximum allowable ΔP for each compartment; if $\int \frac{\Delta P}{\sqrt{D_{abs}}}$ dt exceeded its limit for the decompression schedule, the M-values used in the calculation starting with the one preceding the first violating compartment were lowered by 5 fsw and the schedule was recalculated (10). This iterative procedure was repeated until all limits were satisfied.

Recent findings have justified the conservative approach of limiting the maximum ΔP to 5.4 $\sqrt{633}$. Although animal and human studies have shown that decompression obligation continues to decrease with increasing depth of saturation beyond 600 fsw (3,13), the variability of the data increases greatly. In a later exposure in the series first reported by Flynn and Spaur (6), upward excursions to 820 fsw from 1000 fsw were found to be safe (ΔP = 167 fsw), but ascent to 800 fsw (ΔP = 187 fsw) produced decompression sickness (12). For the depths reached on ascent both ΔP values from these excursions are in excess of the planned limit ($5.4\sqrt{D_{abs}}$). Vorosmarti et al. (19) reported decompression sickness after repetitive one-hour excursions from 820 to 984 fsw. The maximum ΔP of 151 fsw produced by each of these excursions was slightly less than the computed 158-fsw ΔP limit.

PHASE I

DECOMPRESSION SCHEDULE COMPUTATION (for 800-1200-800 fsw Excursions)

The approach outlined in the preceding section was used to calculate the excursion-decompression schedules for trial in Phase I. The excursion involved a 40-minute pseudo-exponential compression from 800 to 1200 fsw with a 65-minute "bottom time" (Section D). Other conditions

included: 1) use of normoxia for periods of compression and exposures to maximum pressure; 2) hyperoxia during decompression to allow faster ascent and to provide an additional margin of safety against decompression sickness; and 3) use of helium-oxygen throughout to avoid alteration of physiological or performance states by other inert gases.

The gas mixture for decompression was helium with an oxygen pressure of 1.0 ata at 1200 fsw, decreasing to 0.68 ata at 800 fsw. The computed schedule with an 89-minute decompression time is given in Table II.

The test program was designed to minimize the occurrence of early or severe decompression sickness. The 40-minute compression profile and the decompression schedule devised for a 65-minute bottom time were used on the first two trials with only a 15-minute bottom time. The plan was to follow these shortest exposures with 30-minute, 45-minute, 55-minute and five 65-minute exposures. The 15-minute bottom times were even more conservative than they appear since they followed the initial compressions from 0 to 800 fsw and the subjects were not yet saturated at 800 fsw. It was not planned to extend bottom times beyond 65 minutes to determine the decompression sickness threshold in the several subjects.

RESULTS--Phase I

Table III shows the actual excursions completed in Phase I and the results of the decompressions. No problems were encountered until the decompression from the 55-minute bottom time at 1200 fsw, when a clear case of decompression sickness (with onset at 830 fsw) occurred. This led to a review and revision of the procedures.

Using the successful decompression for the 800- to 1200-fsw excursion with the 45-minute bottom time as the basis for establishing new ΔP and integral limits, a schedule (Table IV) was computed for a 55-minute working time at the 1200-fsw pressure. This schedule was computed for an exposure identical to the 45-minute profile used to derive its limiting parameters, but with a longer actual bottom time. The new depth-time integral limits for the slowest half-time compartments (130,160,200 and 240 min) were much

TABLE II. Excursion-Decompression Schedule for a 105-minute Excursion from 800 to 1200 fsw

Pressure (fsw)	Stop Time (min)	Ascent Rate (ft/min)	Elapsed Time[a] (min)
1200-1025	-	30	6
1025- 935	-	5	24
935	2	-	26
930	2	-	28
925	2	-	30
920	2	-	32
915	2	-	34
910	2	-	36
905	2	-	38
900	2	-	40
895	2	-	42
890	2	-	44
885	2	-	46
880	2	-	48
875	2	-	50
870	2	-	52
865	2	-	54
860	2	-	56
855	3	-	59
850	3	-	62
845	3	-	65
840	3	-	68
835	3	-	71
830	3	-	74
825	3	-	77
820	3	-	80
815	3	-	83
810	3	-	86
805	3	-	89

[a] Time at end of stop.

smaller than the limits derived from the EDU exposures. They would have therefore required an enormous increase in decompression time, unless higher oxygen pressures were used in the decompression. For this reason the limits for these compartments, believed not to be controlling the decompression from these exposures, were left at their original values. This adjusted schedule was used with the three subjects who had not experienced decompression sickness (CC, LJ, FS) (Table III). In spite of the deeper initial stop and the doubled total decompression time, vestibular decompression sickness occurred in one subject at 850 fsw. After successful treatment, the experiments of Phase I were completed, and the subjects decompressed to atmospheric pressure (Section G-2).

TABLE III. Summary of Decompression Schedule Testing for Excursions from 800 to 1200 fsw[a]

Date	Bottom Time at 1200 fsw (min)	Decompression Schedule Used	Time from Previous Excursion (day/hr:min)	Subject	Activity	Decompression Sickness[b] 0-No Symptoms +-Symptoms
7/1/75	15	Original (see Table II)	-	FS WS	Rest Exercise	0 0
7/2/75	15	Original	-	CC LJ	Exercise Rest	0 0
7/2/75	30	Original	27:30	FS WS	Rest Exercise	0 0
7/3/75	45	Original	20:33	CC LJ	Rest Exercise	0 0
7/3/75	55	Original	20:23[c]	FS WS	Rest Rest	0[d] +
7/6/75	55	Recomputed (see Table IV)	2/20:14 2/20:14 2/13:24	CC LJ FS	Exercise Rest Rest	0[d] + 0[d]

[a]Conditions during excursions.
Breathing gases:
Compression Normal O_2 (0.21 atm) in He
Bottom time Normal O_2 (0.21 atm) in He
Decompression 2.7% O_2 in He
Compression profile: (Total time = 40 min)
800-1000 fsw at 20 ft/min
1000-1100 fsw at 10 ft/min
1100-1200 fsw at 5 ft/min

[b]See Section G-4 for description of circumstances, treatment and outcome of all cases of decompression sickness.

[c]Includes hyperoxic pre-breathing of 7% O_2 in He for 30 minutes ending 6 hours and 49 minutes prior to start of excursion.

[d]Symptom-free subject recompressed for therapy of partner and must be omitted from this evaluation.

TABLE IV. Excursion-Decompression Schedule Computed for a 95-minute Excursion from 800 to 1200 fsw. (Revision Based on Experience Using Schedule Given in Table II)

Pressure (fsw)	Stop Time (min)	Ascent Rate (ft/min)	Elapsed Time[a] (min)
1200-1025	-	30	6
1025- 945	-	5	22
945	2	-	24
940	2	-	26
935	3	-	29
930	4	-	33
925	4	-	37
920	4	-	41
915	4	-	45
910	4	-	49
905	4	-	53
900	4	-	57
895	4	-	61
890	4	-	65
885	5	-	70
880	6	-	76
875	5	-	81
870	6	-	87
865	6	-	93
860	6	-	99
855	6	-	105
850	6	-	111
845	6	-	117
840	6	-	123
835	6	-	129
830	7	-	136
825	8	-	144
820	7	-	151
815	8	-	159
810	9	-	168
805	10	-	178

[a] Time at end of stop.

DISCUSSION--Phase I

The original decompression schedule (Table II) was successful for an exposure (compression + bottom time) about 80% as long as that for which it was computed. This schedule was considerably shorter than those computed by any of the other decompression computation methods mentioned earlier. Since the 45-minute bottom time was sufficient for the study purposes, and doubling the decompression time failed to eliminate decompression sickness, it was decided to proceed with final development of schedules for the Phase II excursions (1200 to 1600 fsw) based on the initial concepts and procedures, but making no further attempt to extend the exposure time beyond the 40-minute compression, 45-minute bottom time combination.

PHASE II

DECOMPRESSION PROCEDURE DEVELOPMENT (1200-1600-1200 fsw)

Three steps were involved in the development of decompression procedures for Phase II. First, the decision was made to transpose the 89-minute schedule successfully tested in Phase I for 1200-800 fsw decompressions for the 1600-1200 fsw decompressions (Table V). This approach was considered conservative since it involved the same depth change as in Phase I but at a greater absolute depth. Second, since compression rate could be an experimental variable, and the attainable excursion depths could not be predicted in advance (Section D), bottom time limits had to be computed for different rates and depths. The longest safe excursion exposure made in Phase I was used as the basis for these computations. Tissue compartment inert gas tensions were calculated for the successful 40-minute compression, 45-minute bottom time exposure. Maximum bottom times (Table VI) were then computed for exposures with various compression rates and excursion depths such that tissue gas tensions did not exceed those in the longest safe test dive.

Third, oxygen partial pressure at the beginning of actual decompression was elevated to 1.6 ata to increase the safety margin. As additional safety measures, time

TABLE V. Excursion-Decompression Schedule Computed for Excursions from 1200 to 1600 fsw

Pressure (fsw)	Stop Time (min)	Ascent Rate (ft/min)	Elapsed Time[a] (min)
1600-1425	-	30	6
1425-1335	-	5	24
1335	2	-	26
1330	2	-	28
1325	2	-	30
1320	2	-	32
1315	2	-	34
1310	2	-	36
1305	2	-	38
1300	2	-	40
1295	2	-	42
1290	2	-	44
1285	2	-	46
1280	2	-	48
1275	2	-	50
1270	2	-	52
1265	2	-	54
1260	2	-	56
1255	3	-	59
1250	3	-	62
1245	3	-	65
1240	3	-	68
1235	3	-	71
1230	3	-	74
1225	3	-	77
1220	3	-	80
1215	3	-	83
1210	3	-	86
1205	3	-	89

[a] Time at end of stop.

intervals between excursions were arranged to be no less than 20 hours, and excursions were limited to three days in succession.

TABLE VI. Maximum Bottom Times[a] Allowed at Various Depths for Excursions from 1200 fsw for Use With Different Pseudo-Exponential Compression Profiles

Compression Profile[b]	Excursion Depth (fsw)				
	1400	1450	1500	1550	1600
10- 5- 2½	150	100	75	40	15
16- 8- 4	150	110	80	55	35
20-10- 5	155	110	85	60	45
32-16- 8	155	115	90	70	50
40-20-10	155	115	90	70	55
80-40-20	155	120	95	75	60

[a] Times, to next lowest 5-minute interval, that keep inert gas tensions in all compartments below values calculated from the longest, safe Phase I exposure.

[b] Pseudo-exponential compression profile for 400-fsw excursion. The first number in the sequence indicates ft/min for first 200 feet, the second number for next 100 feet and the last number is ft/min for last 100 feet.

RESULTS--Phase II

The results of the Phase II excursions and decompressions are summarized in Table VII, showing six exposures of two men each. No cases of decompression sickness occurred in the first 10 man-dives. The last excursion resulted in an episode compatible with a diagnosis of vestibular decompression sickness, occurring 58 minutes after the completion of decompression.

Actual bottom times varied from 42 to 55 minutes (Table VII) depending on experiment and operational needs; between-dive intervals were 20 hours or more. Excursions with underwater work involved exposure conditions which differed from those encountered in excursions within the helium-oxygen gas environment. The subjects performed hard work in comfortably warm water for 10-15 minutes,while breathing helium with 1 ata oxygen at an average simulated depth of 1610 fsw (Section F).

TABLE VII. Summary of Excursion-Decompressions from 1200 to 1600 fsw[a]

Date	Compression Time (min)	Bottom Time at 1600 fsw (min)	Time from Previous Excursion (day/hr:min)	Subject	Activity	Decompression Sickness 0-No Symptoms +-Symptoms
8/8	40	42	-	GM	Exercise	0
				FS	Rest	0
8/9	20	55	20:34	GM	Exercise	0
				FS	Rest	0
8/12	20	55	-	CC	Exercise	0
				MP	Rest	0
8/13	20	44	20:30	CC	Exercise	0
				MP	Rest	0
8/15	20	49	5/18:01	GM	Work	0
				FS	Work	0
8/15	20	55	1/23:33	CC	Work	0[b]
				MP	Work	+

[a] Breathing gases:
Compression — Normal O_2 (0.21 atm) in He
Bottom time — Normal O_2 (0.21 atm) in He for excursions with rest and exercise activity
1 ata O_2 in He for excursions with work activity
Decompression — 3.2% O_2 in He

[b] Symptom-free subject was recompressed for therapy of partner and must be omitted from this evaluation.

DISCUSSION--Phase II

The decompression procedures devised for the deep saturation-excursion pressurizations were based on the demonstrations by the U.S. and British Naval investigators that, for no-decompression ascents from a saturated state, the permissible ascent step size becomes greater as the helium saturation depth increases. The new excursion-decompression procedures were computed with consideration of degree and duration of excess tissue gas pressure (ΔP and time relationships) for each tissue gas exchange compartment. They called for less than one-tenth of the decompression time which would be required to return to atmospheric pressure at sea level from a 60-minute dive to 400 fsw made from the surface (Fig. 3).

During Phases I and II, 25 man-excursions from 800 to 1200 fsw and from 1200 to 1600 fsw were made with three episodes of decompression sickness. Of the remaining 22

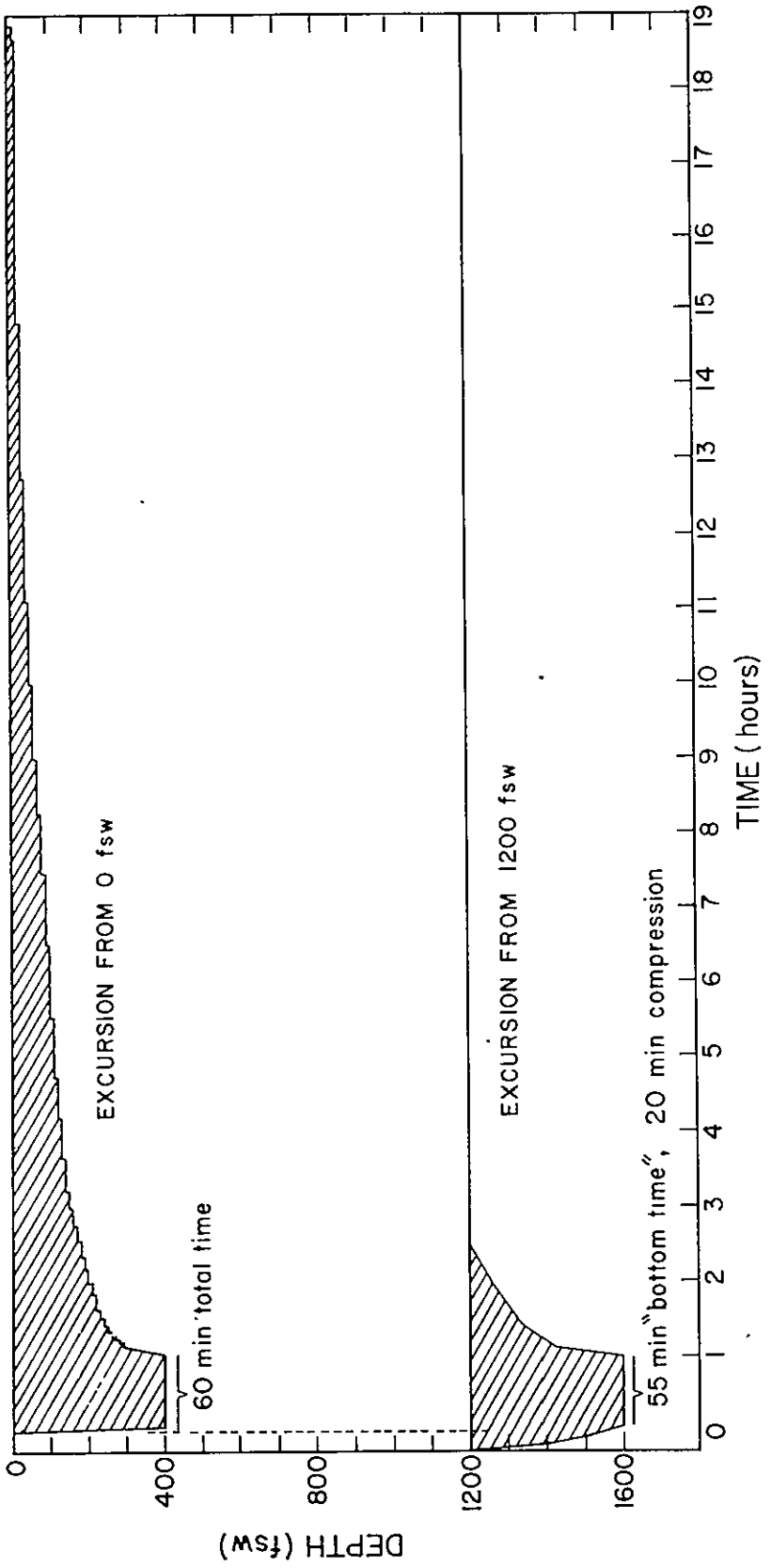

FIG. 3. Comparison of decompressions for 400-fsw excursions from saturation depths of 0 fsw (top) and 1200 fsw (bottom).

man-exposures without symptoms of DCS, 4 could not be fully evaluated with respect to decompression since the divers were recompressed with those suffering decompression sickness before the decompression was completed or adequate time to assess their state had been allowed. The incidence of decompression sickness, therefore, must be based on 21 exposures, of which 3 resulted in positive or possible decompression sickness. In all three cases, the divers responded immediately to treatment by recompression and hyperoxygenation and experienced no residual deficits (Section G-4).

Of primary importance is the fact that these procedures, by providing rapid decompression for excursion exposures, enabled all the experimental objectives of the Predictive Studies IV program to be met, even though the procedures were significant departures from current decompression practices with respect to decompression time. While decompression sickness did occur, this experience demonstrates that, as in the case of no-decompression excursions from deep saturation, stage decompression requirements are reduced at increased saturation depth, and that data from no-decompression exposures can be used in computing stage decompressions. The "time-weighted ascent contraint" method employed here to extrapolate from no-decompression ascents to stage decompression can be used to estimate decompression obligation even to depths and times for which there is little or no prior experience.

The method provides a rationale for two phenomena connected with decompression schedule computation and use:

1. Previously, the necessity to reduce maximum allowable inert gas pressures (M-values) in decompression computations as exposure pressure or time has increased has not been explained. This may now be explained as follows: The increased tissue inert gas load associated with deeper and longer exposures requires more time for elimination during decompression. An M-value (ΔP_{max} + 33) found entirely safe for decompressing from a shallower or shorter exposure may not be safe for decompressing from the deeper and extended exposure because the cumulative ΔP-time function established by this M-value becomes too large, with increased probability

for bubble formation and growth. One way to reduce $\int \Delta P(t)\,dt$ is to use arbitrarily smaller M-values in the calculation of the decompression schedule.

2. With a given system of M-values and a decompression schedule shortened by the use of hyperoxia in its calculation, the incidence of decompression sickness is less than expected. This may be due to decrease in the $\int \Delta P(t)\,dt$ which is a direct result of the shorter decompression.

Recent experimentation (14) and common sense suggest that tolerable excess gas pressure (ΔP) should probably be a nonlinear rather than a linear function of time. This consideration must receive attention as one way of improving the accuracy and thus the safety margin of the concept and computation method used for this study. That there was little such margin even in the Phase II procedures was demonstrated by the episode of presumed decompression sickness of subject MP following a hard-working underwater exposure at 1600 fsw with the maximum allowable bottom time.

It was not a practical purpose of the decompression trials in this Predictive Study to provide a fully validated system of optimal decompression in deep saturation-excursion diving. The success attained indicates that there is abundant room for improvement in such operational methods. There is, however, need both for reducing incidence of decompression sickness and for exploring the basic mechanisms that permitted a tenfold shortening of the decompression required. These have not been identified, in spite of the emphasis here on the importance of considering duration as well as degree of exposure to excess tissue gas pressure.

RECOMMENDATIONS

1. At this stage in the evolution of operational saturation-excursion diving it is desirable to stay within the no-decompression limits for saturation-excursion exposures established by investigations of the U.S. Navy Experimental Diving Unit (13). This interim approach eliminates

both a need for various schedules for different diving profiles and also concern for the effects of repetitive exposures.

2. If excursions greater in degree than those allowed by the U.S. Navy Unlimited Excursion Table (13) must be made, then the decompression system developed for these studies can be the basis for developing decompression procedures for many different exposures. Without further practical trial this approach is not without risk, and two precautions are recommended:

a. The exposure used as the basis of schedule calculation should be a conservative one, by reducing either the exposure time or depth used in this study.

b. Although therapeutic recompressions in both Phases I and II did not exceed the saturation depth by more than 250 fsw, availability of surface-based saturation capability to the maximum depth of excursion is recommended for the treatment of decompression sickness (see also Section G-4).

3. The time-weighted calculation technique devised for these simulated saturation-excursion dives should be applicable to solution of decompression requirements for many other exposure situations, including less extreme circumstances. Specific investigation is necessary, however, to describe accurately the relationships of duration and excess gas pressures in precipitating decompression sickness in order to exploit this technique over the full range of its applicability.

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G-2. DECOMPRESSION FROM SATURATION EXPOSURES

K.M. Greene[1], R.E. Peterson[1] and C.J. Lambertsen[1]

The prolonged exposure of men to helium at pressures of 800 and 1200 fsw required use of a "saturation-decompression" method for return to atmospheric pressure. An entirely satisfactory procedure for this had not been previously established by the several laboratories, navies and industrial groups employing helium in deep, long exposures. Design of the profiles for decompression from saturation in Phases I and II of this Predictive Study attempted to prevent the high incidence of "pain only" decompression sickness (DCS) which has been a common feature of deep saturation-decompressions.

Knee pain, with onset usually in the final 100 feet of ascent, has been observed with a wide range of decompression rates, with and without periodic holding at constant pressure. While occurring during decompression from both shallow and deep exposures, the incidence of decompression sickness has been considerably greater following deep dives. Because of these previous experiences a review of past and possible new approaches was undertaken with the objective of providing efficient and safe saturation-decompression profiles for the pressure exposures required by these studies.

BACKGROUND

It has been reported that in U.S. Navy helium saturation diving, 95% of all symptoms of decompression sickness occur at depths shallower than 150 fsw, and that the

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incidence following saturations deeper than 300 fsw is 11% as opposed to 1.7% for shallower exposures (17). Following a U.S. Navy 1600-fsw helium exposure, four of the six divers developed knee bends, with the deepest onset at 106 fsw (16). Table I indicates other examples of decompression sickness occurring after deep dives. A conspicuous exception to the general trend of shallow-onset knee bends is the inner ear decompression sickness which was observed in the 1970 pressure exposures of the Royal Naval Physiological Laboratory (10); onset was at 1260 fsw after an initial decompression rate of 40 fsw/hour from 1500 fsw. Final decompression was accomplished with a slower schedule and with knee bends in one of the two divers at a depth of 26 fsw. Another case of inner ear decompression sickness was seen early in the ascent from a 2001-fsw helium pressure exposure conducted by COMEX (5).

The 14 man-decompressions from 1600 fsw or deeper referred to in Table I employed extensions of methods already in use for decompression from shallower saturation depths. The shapes of the decompression profiles and the times to reach the surface varied widely in the five exposures. Both "slow" (U.S. Navy, 1973) and "fast" (Sagittaire IV, 1974; Physalie V, 1970) decompressions produced decompression sickness.

BASIC APPROACH

The major considerations involved in choosing the approach to decompression from saturation exposures in this program included selection of ascent rates, use of nitrogen-oxygen (air) breathing to speed helium elimination, and use of oxygen on approaching atmospheric pressure to speed all inert gas elimination.

Since decompression sickness has occurred principally at shallow depths, following both relatively slow and rapid deep saturation-decompressions, it was decided to pause for 24 hours at 200 fsw, a pressure slightly greater than the zone within which saturation-decompression DCS is common. It was expected that this maneuver would allow resolution of asymptomatic bubbles and elimination of excess dissolved gas. Decompression could then be resumed with

TABLE I. Summary of Experience with Decompression from Deep Saturation Exposures

Exposure	Reference	Depth (fsw)	Decompression Time (days)	Number of Subjects	Number of DCS Cases	Depth of Onset (fsw)
U.S. Navy-Duke University, 1968	19	1000	12	5	1	150
R.N.P.L., 1970	3	1000	3-2/3	3	1	63
University of Pennsylvania Predictive Studies III, 1971	7	1200	9	4	1	20
University of Pennsylvania	-	1050	7-2/3	4	1	500
Predictive Studies IV, 1975[a]	-	1400	10	4	2	917,10
R.N.P.L., 1970	10	1500	11	2	2	1260[b],26[c]
U.S. Navy, 1973	16	1600	19	6	4	≤ 106
COMEX Sagittaire II, 1972	14	1640	8	2	0	-
COMEX Physalie V, 1970	12	1706	9	2	1	66
COMEX Physalie VI, 1972	13	2001	10	2	0	-
COMEX Sagittaire IV, 1974	5,6	2001	10	2	1[d]	-

[a] Present study.

[b] Vestibular decompression sickness (see text) recompressed to 1535 fsw.

[c] Recompression from 26 fsw for subject with knee bend, said to have been present for three days.

[d] This is the case of vestibular decompression sickness referred to by Fructus and Wide (5).

what was expected to be a risk of decompression sickness closer to that associated with shallower saturation-decompressions.

The slow, essentially linear decompression pattern of ascent rates followed by the U.S. Navy schedule was not selected, since this method was no more effective than faster procedures in preventing bends in its single trial deeper than 1000 fsw. Most other schedules employed to date begin decompression with a relatively rapid rate which is decreased significantly during ascent. This approach, which was derived empirically in U.S. Navy trials, relates to the general concept that the diver's tolerance to excess inert gas[2] (ΔP) increases with exposure depth (1,4,18). This concept was used for the design of the excursion-decompression schedule for this Predictive Study (see Section G-1) and was also employed as the basis for computation of these saturation-decompressions.

In the case of a single-step decrease in depth, the tolerable $\Delta P = 5.4\sqrt{D_{abs}}$, where D_{abs} = absolute depth or gauge depth plus the 33 fsw representing the atmospheric pressure at sea level. However, it was presumed here that a ΔP of this magnitude could not be sustained for prolonged periods without producing symptoms (Section G-1). For the extended exposures to positive ΔPs in saturation-decompression, it was assumed that while a depth-related function of the form ($k\sqrt{D_{abs}}$) could be used, it should

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[2]The amount of excess inert gas contained in a tissue is a function of, among other factors, the partial pressure of the inert gas in the tissue and the ambient pressure. ΔP, in a strict sense, is the difference between the tissue pressure of a gas and the ambient pressure; if all the gas remains in solution in a state of supersaturation, ΔP and excess inert gas are directly related. While the computations of values of ΔP were made with the above definition, the conceptual analysis presented here and in the section on decompression from excursion exposures (G-1) relating ΔP and time is believed to be independent of the actual state of any excess inert gas in a tissue. Whether the excess gas is in a state of true supersaturation, or there is phase separation, or a combination of the two states exists, it is considered that the longer the conditions of excess inert gas exist, the greater is the potential for bubble formation and growth. The term and values for ΔP, therefore, are used here in a broad sense as an index of the degree of any excess inert gas in a tissue.

be modified to provide a smaller constant (k) than 5.4. To derive the value for k it was necessary to solve $\Delta P = k\sqrt{D_{abs}}$ for a ΔP known to be safe at some depth. Decompression rates approximating 10 fsw/hour have been used several times in the depth range of 1000 fsw without incidents of decompression sickness (3,10,12,13), including the first 300 fsw of Predictive Studies III (7). A prolonged decompression at this rate sets up a ΔP which can be calculated if the respired P_{O_2} and the controlling tissue gas exchange half-time are known. This calculation, based on the usual exponential decompression model as described by Workman and Bornmann (22), is shown in the appendix to this section. A conservative value of 240 minutes was chosen as the limiting half-time for helium saturation (3,15,22). With an inspired P_{O_2} of 0.5 atm, a value of 41 fsw was obtained for the stabilized ΔP corresponding to the decompression rate of 10 fsw/hour, and k then became 1.28.

DECOMPRESSION SCHEDULE DESIGN

With this formula ($\Delta P = 1.28\sqrt{D_{abs}}$), related decompression rates can be calculated for any depth range. Table II shows integer values of decompression rates (6 to 12 minutes/fsw) together with associated ΔPs and the range of depths to which they apply. The 11 minutes/fsw value was omitted to reduce the number of rate changes required to implement the decompression. The resulting time-depth profile is faster than the U.S. Navy schedule but slower than or parallel to others such as the Royal Naval Physiological Laboratory 1000- and 1500-fsw exposures, Physalie V and VI, and Sagittaire II.

The depth range in Table II terminates at 100 fsw since transfer from a helium-oxygen mixture to air was scheduled at that depth to accelerate the final phase of ascent. This depth was chosen as a compromise between maximizing the decompression advantage and minimizing narcosis and pulmonary oxygen toxicity. The parameters used for this final ascent included M values (maximum permissible inert gas pressure at sea level) and ΔM values (additional permissible inert gas pressure for an increment in depth) (21) as follows: for the nitrogen, 48 fsw

and 1 fsw/fsw depth; for helium, 52 fsw and 1 fsw/fsw depth (8). Two half-time values of tissue gas exchange were used for nitrogen (480 and 670 minutes) and one for helium (240 minutes). These values for half-times span the range generally attributed to the slowest-exchanging nitrogen compartment producing a conservative decompression calculation (11). The computations were done by digital computer using this Institute's decompression calculation and analysis program (PADUA) (2).

TABLE II. Decompression Rates for Given Depth Ranges

Depth Range (fsw)	ΔP[a] (fsw)	Decompression Rate (min/ft)
>1000	41	6
1000-650	33	7
650-425	27	8
425-275	22	9
275-200	20	10
200-100	12	12

[a] $\Delta P = 1.28\sqrt{D_{abs}}$

The shift from breathing a helium-oxygen mixture to breathing air has the potential for producing a "superficial form" of the isobaric inert gas counterdiffusion syndrome (7,9). The condition, due to the breathing of nitrogen while surrounded by helium (9), was avoided by changing the entire ambient atmosphere and transferring the subjects to a connecting, air-filled compartment. This avoided mask breathing of air in a helium-oxygen ambient environment (9). A brief, 30-minute period of hyperoxic

helium breathing (P_{O_2} = 2 atm) was introduced immediately before transfer to the air-filled compartment to increase tissue helium elimination and thus add a safety margin for the rapid acceleration of decompression rate associated with the air switch. This hyperoxic breathing period was not taken into account in the schedule computation.

The oxygen partial pressure in the helium-filled chamber was maintained at 0.5 atm, below the threshold of measurable pulmonary oxygen toxicity. Following the period of hyperoxygenation at 100 fsw, air was breathed until 20 fsw was reached. At this time, the divers commenced breathing pure oxygen on a schedule of 20 minutes oxygen, 5 or more minutes air. The exact oxygen-air schedule delivered an oxygen "dose" of 546 UPTD (Unit Pulmonary Toxic Dose) units (23) while providing a reserve margin of oxygen tolerance for therapy of decompression sickness. As a safety factor in decompression it was assumed that oxygen breathing commenced at 10 fsw for purposes of the decompression schedule calculation.

A decompression pressure change step-size of one fsw was used during the helium-oxygen phase but after the transfer to air five-foot steps were employed. A "terminal recompression" was made from 10 to 30 fsw on oxygen as a final effort to reduce gas phase separation before surfacing (8). The final schedule for Phase I including the 24-hour stop at 200 fsw is shown in Table III. Figure 1 compares this profile with others. All profiles shown have 1000 fsw as their starting point (time = 0) to make comparison easier; in cases where the decompression started at a depth deeper than 1000 fsw, that portion of the profile has been omitted.

RESULTS

PHASE I DECOMPRESSION

The saturation-decompression was begun on the schedule shown in Table III and Fig. 1, starting at 1050 fsw after a period of 38 hours, 58 minutes at that depth for the treatment of decompression sickness in subject LJ. Decompression was interrupted at 414 fsw for treatment of pain-only DCS

TABLE III. Decompression Schedule for Helium-Oxygen Saturation Exposure (Phase I)

Depth (fsw)	Ascent Rate (min/ft)	Ascent Time (hr)	Elapsed Time from 1000 fsw (hr)	Breathing Gas
>1000	6	-	-	0.5 atm O_2, bal He
1000-650	7	41	41	0.5 atm O_2, bal He
650-425	8	30	71	0.5 atm O_2, bal He
425-275	9	22.5	93.5	0.5 atm O_2, bal He
275-200	10	12.5	106.0	0.5 atm O_2, bal He
200	-	24	130.0	0.5 atm O_2, bal He
200-103	12	19.4	149.4	0.5 atm O_2, bal He
103-100	12	0.6	150.0	50% O_2, 50% He

Depth (fsw)	Stop Time (min)	Elapsed Time from 100 ft (min)	Elapsed Time from 100 ft (hr:min)	Breathing Gas
100	19	19	0:19	Air
95	19	38	0:38	Air
90	20	58	0:58	Air
85	21	79	1:19	Air
80	23	102	1:42	Air
75	24	126	2:06	Air
70	25	151	2:31	Air
65	27	178	2:58	Air
60	29	207	3:27	Air
55	31	238	3:58	Air
50	34	272	4:32	Air
45	37	309	5:09	Air
40	40	349	5:49	Air
35	44	393	6:33	Air
30	49	442	7:22	Air
25	54	496	8:16	Air
20	25	521	8:41	100% O_2
	5	526	8:46	Air
	25	551	9:11	100% O_2
	7	558	9:18	Air
15	25	583	9:43	100% O_2
	5	588	9:48	Air
	25	613	10:13	100% O_2
	14	627	10:27	Air
10	30	657	10:57	100% O_2
	8	665	11:05	Air
30	30	695	11:35	100% O_2
30-0	-	725	12:05	100% O_2

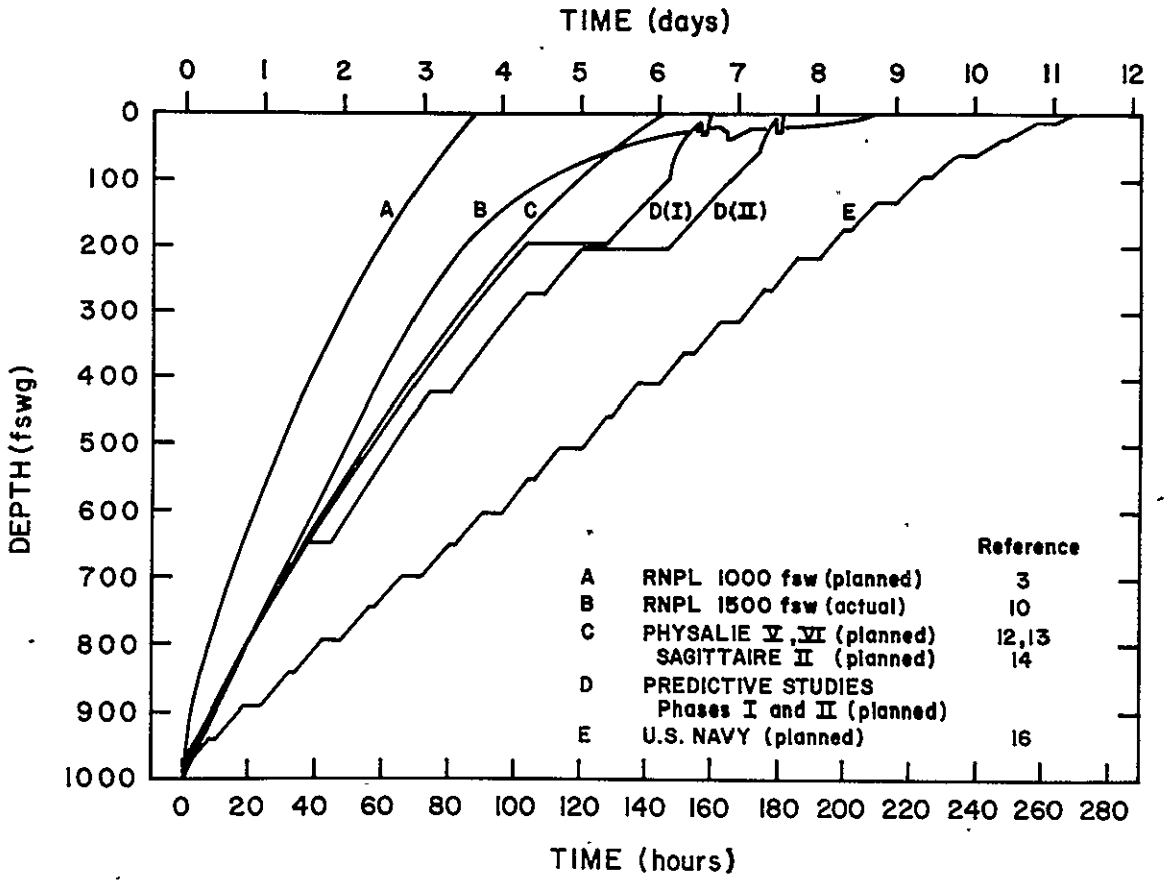

FIG. 1. Comparison of saturation-decompression schedules from pressure exposures equal to or greater than 1000 fsw. The portions of the profiles deeper than 1000 fsw have been omitted and zero time started at 1000 fsw for comparison.

in the same subject (see Section G-4). After a successful eight-hour treatment at 465 fsw, decompression was resumed from that depth on the original schedule. To decrease the likelihood of recurrence of DCS, the chamber P_{O_2} was increased intermittently to cover a range from 0.7 to 0.5 atm. The oxygen percentage was increased to give a P_{O_2} of 0.7 atm and maintained until decompression reduced the P_{O_2} to 0.5 atm. This procedure was repeated as shown below until the shift to air at 100 fsw. The original schedule was then followed to the surface.

Depth Range (fsw)	Percent O_2	P_{O_2} (atm)
465-423	3.6	0.55 → 0.5
423-293	5.1	0.7 → 0.5
293-200	7.1	0.7 → 0.5
200-134	9.9	0.7 → 0.5
134-100	13.8	0.7 → 0.55

Subject LJ emerged with residual soreness in the knee and a severe headache; both were resolved without recompression. The other three subjects emerged without symptoms or signs of decompression sickness. Total decompression time from 1050 fsw (including treatment time) was 7 days, 14 hours, 28 minutes.

RECOMPUTATION OF DECOMPRESSION PLAN FOR PHASE II

Although three of the four subjects surfaced from the Phase I saturation-decompression without decompression sickness, the schedule was modified in an attempt to further improve the margin of safety for the Phase II saturation at 1200 fsw. Periodic "holds" or "stops" have been used by the U.S. Navy and others as a means of reducing the risk of decompression sickness during saturation-decompressions. This approach was applied here in a

systematic way by inserting stops at each rate change in the schedule. The length of each hold was adjusted to allow each calculated ΔP to decrease to the value controlling the next segment of the schedule (Table II), and the 24-hour stop at 200 fsw was retained. The revised schedule, shown in Table IV, has a total calculated oxygen toxicity dose of 399 UPTD units, with P_{O_2} maintained at 0.5 atm during the helium-oxygen phase. The pressure for transfer to air was changed from 100 to 60 fsw because of concern about possible complications in the event of need for recompression therapy. It was considered that recompression on air as treatment for decompression sickness occurring shortly after the transfer might cause problems with narcosis and oxygen toxicity, while changing back to oxyhelium might set up deep tissue isobaric counterdiffusion supersaturation (9) (see also Section G-4). The decompression rate from 100 to 60 fsw was 12 minutes/fsw, which is still within the curve of $\Delta P = 1.28 \sqrt{D_{abs.}}$ The decompression subsequent to the transfer to air was recomputed using the same techniques as before. The hyperoxygenation immediately prior to the transfer to air was repeated, with the oxygen percentage adjusted appropriately.

PHASE II DECOMPRESSION

The final decompression from Phase II began at 1400 fsw following 42 hours, 49 minutes at that depth for the treatment of inner ear decompression sickness in subject MP. Decompression was halted at 1200 fsw for the 9 hours, 28 minutes required for completion of programmed experimental studies and then resumed according to plan. Subject MP experienced a prolonged episode of diffuse lower extremity pain which began about 917 fsw and, in retrospect, appears to have been an atypical case of DCS as described in Section G-4. The decompression was initially not altered since symptoms stabilized on aspirin and rest and he was eventually pain-free until at about 115 fsw where the pain recurred in one knee and eventually required recompression.

During the air phase of decompression, three subjects (CC,GM,FS) had transient tooth pain and subject CC had brief sensations of discomfort ("niggles") in the knees with each five-foot decompression step. Beginning at 30

TABLE IV. Decompression Schedule for Helium-Oxygen Saturation Exposure (Phase II)

Depth (fsw)	Ascent Rate (min/ft)	Ascent Time (hr)	Elapsed Time from 1000 ft (hr)	Breathing Gas
>1000	6			0.5 atm O_2, bal He
1000	-	8		0.5 atm O_2, bal He
1000-650	7	40.8	40.8	0.5 atm O_2, bal He
650	-	7	47.8	0.5 atm O_2, bal He
650-425	8	30	77.8	0.5 atm O_2, bal He
425	-	6	83.8	0.5 atm O_2, bal He
425-275	9	22.5	106.3	0.5 atm O_2, bal He
275	-	5	111.3	0.5 atm O_2, bal He
275-200	10	12.5	123.8	0.5 atm O_2, bal He
200	-	24	147.8	0.5 atm O_2, bal He
200-63	12	27.4	175.2	0.5 atm O_2, bal He
63-60	12	0.6	175.8	68.75% O_2, bal He

Depth (fsw)	Stop Time (min)	Elapsed Time from 60 fsw (min)	Elapsed Time from 60 fsw (hr:min)	Breathing Gas
		0	0:00	
60	25	25	0:25	Air
55	26	51	0:51	Air
50	28	79	1:19	Air
45	30	109	1:49	Air
40	33	142	2:22	Air
35	35	177	2:57	Air
30	39	216	3:36	Air
25	42	258	4:18	Air
20	20	278	4:38	100% O_2
	5	283	4:43	Air
	20	303	5:03	100% O_2
	2	305	5:05	Air
15	3	308	5:08	Air
	20	328	5:28	100% O_2
	5	333	5:33	Air
	20	353	5:53	100% O_2
	5	358	5:58	Air
10	20	378	6:18	100% O_2
	5	383	6:23	Air
30	30	413	6:53	100% O_2
30-0	-	443	7:23	100% O_2

fsw the step size was reduced to 1 fsw without changing the overall rate, in an attempt to eliminate the "niggles." Beginning at about 10 fsw, subject CC developed clearcut knee decompression sickness which was successfully treated at 60 fsw by use of an oxygen treatment procedure (Section G-4). Two subjects (GM and FS) completed the scheduled decompression independent of the therapeutic recompressions and emerged without symptoms. Total decompression time from 1400 fsw for the untreated subjects, including the constant pressure period at 1200 fsw, was 9 days, 23 hours, 44 minutes.

DISCUSSION

The attempt to devise a decompression procedure which would prevent decompression sickness at the lower range of pressures was not entirely successful. The procedures which were employed, however, were more efficient than the U.S. Navy schedule which had a higher DCS incidence on a slower ascent profile. They were comparable in efficiency to the decompression used in the Sagittaire II and IV, and Physalie V and VI dives.

The residual effect, if any, of the excursions from 800 to 1200 fsw and from 1200 to 1600 fsw prior to the start of the saturation-decompressions is not known. It has been shown that saturation-decompression on the U.S. Navy schedule may safely begin immediately following lesser, no-stop excursions(18). Final decompression was to be started only after a 36-hour hold following the last excursion, since the excursion exposure histories differed between the subjects. Furthermore, it was considered possible that the occurrences of decompression sickness in the excursion-decompressions might have an adverse effect in the subsequent saturation-decompressions. Actually, the two subjects (LJ and MP) who developed bends during the Phase I and II saturation-decompressions while still at high pressure had both suffered inner ear DCS in their final excursions, approximately 40 hours prior to beginning saturation-decompression. On the other hand, subject WS, who had DCS following an excursion five days prior to leaving the 1050-fsw saturation pressure in Phase I, had no problem in the saturation-decompression. The unusual occurrences of DCS at high pressure during the

saturation-decompressions may indicate that the initial saturation-decompression rates were too early or too rapid for divers recently recovered from decompression sickness.

The transfer from a helium to an air environment shortened the decompression time significantly. The only case of decompression sickness with onset after the transfer was easily treated. The transfer from helium to air at depths too great for the use of pure oxygen in DCS therapy is safe only if transfer back to helium-oxygen and rapid recompression to relieve symptoms are existing capabilities. The recompression must effectively reduce bubble size even in the face of an unfavorable inert gas exchange rate balance which results from the air-to-helium transfer. Since the requirements were met in the laboratory conditions of the Predictive Studies IV exposures, the transfer from helium-oxygen to air at 100 fsw in the Phase I saturation-decompression was entirely reasonable.

The potential utility of the "terminal oxygen recompression" feature (8) cannot be assessed from the results of this Predictive Study, since there are no control data for comparison.

The use of one fsw as a pressure increment provides the closest practical approximation to a continuous linear reduction in pressure. While this theoretically maximizes inert gas elimination, larger step sizes are operationally simpler and have the potential advantage of clarifying the onset of decompression sickness. A step size of 5 meters of sea water (msw) at a rate of 1 msw/minute is used in a new Royal Navy schedule (20). This schedule was bends-free in 36 man-dives to 250 msw (820 fsw); however transient "niggles" were frequently reported at the time pressure was dropped. While decompression schedules with relatively large steps have not been used in decompressions incorporating transfer to air before surfacing, there is no reason to expect different results.

No allowance was made in the Predictive Studies IV decompression schedule for the effects of sleep. While some investigators hypothesize that inert gas elimination is decreased by the lowered circulation and tissue perfusion in sleep, it cannot be determined whether those particular slowly perfused tissues which control saturation-decompression

would be affected. However, the diver's awareness of pain is reduced in sleep and two of the three decompression sickness cases which occurred in this study had onset of pain during sleep. If stops are to be inserted to slow the decompression, it would be prudent to synchronize them with the sleep schedule.

In spite of the occurrence of decompression sickness in these studies during decompression from helium saturation, the concept that the tolerable ΔP (excess tissue inert gas) is a function of depth is considered to be a rational basis for designing saturation-decompression profiles. The limited experiences of the Phases I and II saturation-decompressions by themselves do not establish either its validity, or the value of the prolonged stop at 200 fsw in reducing the influence of the deep phase of the dive.

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APPENDIX G-2

Relationship of Linear Saturation Decompression Rate to Allowable Inert Gas Supersaturation (ΔP)

At the maximum tolerable decompression rate (dP/dt), the rate of change of tissue inert gas tension ($d\pi/dt$) will equal that rate:

$$d\pi/dt = dP/dt = -K(\Delta P + P_{O_2})$$

where π, P, ΔP and P_{O_2} are in fsw. Rearranging,

$$\Delta P = \frac{(dP/dt)}{-K} - P_{O_2}$$

If: dP/dt = -10 fsw/hour = -0.1667 fsw/minute

P_{O_2} = 0.5 atm = 16.5 fsw

K = $2.888\ (10^{-3})$ = rate constant for 240 minute-compartment

then $\Delta P = \frac{-0.1667}{-2.888(10^{-3})} - 16.5 = 41$

At D = 1000 fsw and ΔP = 41,

$$\Delta P = k\sqrt{D_{Abs}} = 41 = k\sqrt{1033}$$

$$k = 1.28$$

G-3. ULTRASONIC DETECTION OF VENOUS BUBBLES IN DECOMPRESSION FROM DEEP EXCURSION AND SATURATION EXPOSURES

K.H. Smith[1], R. Gelfand[2], and C.J. Lambertsen[2]

Investigation of decompression in Predictive Studies IV involved decompression from helium saturation exposures at 800 and 1200 fsw, and decompression from 400 fsw "deeper" excursions from these pressures (Sections G-1 and G-2). Demonstrations that decompression sickness is less likely to occur at high ambient pressures than near one atmosphere (1,2) led the Institute for Environmental Medicine to derive excursion-decompression procedures for 800 to 1200 to 800 fsw and for 1200 to 1600 to 1200 fsw, requiring one-tenth of the total decompression time necessary during a 400-foot excursion of equivalent duration from sea level (3,4) (Section G-1).

A specific component of the Predictive Studies IV program was to apply the ultrasonic Doppler flow/bubble detector method, already extensively investigated at relatively shallow depths (7-9,11), to examine occurrence of circulating bubbles in rapid decompression from transient deep excursions and slow decompression from saturation exposure to high ambient pressures. This Predictive Study planned to utilize both the experience of investigators familiar with the method and equipment representative of the best currently available. These goals were met by invitation to the Virginia Mason Research Center to join

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with the Institute for Environmental Medicine in this aspect of Predictive Studies IV. This specific collaboration was practical only in Phase II (400-800-1200-1600 fsw) of the overall program. Therefore no bubble detection investigations are presented for Phase I (400-800-1200 fsw) exposures.

METHODS

The Doppler ultrasonic bubble detector system employed was that described by Smith and Spencer (7), Spencer et al. (9) and Spencer and Clarke (10), and elaborated by Spencer (8).

The principle upon which the bubble detection investigation was based is that existing ultrasonic evidence of embolic bubbles occurring in man during and after decompression from air-breathing exposures indicates that they are detectable in large peripheral veins and the right heart (8). The occurrence in peripheral arteries, observed in sheep during more drastic decompression than employed for humans, undoubtedly represents passage of gas emboli through the pulmonary circulation and usually results in collapse, convulsions and death (11).

Monitoring in the present study included the regions over the pulmonary artery to detect changes in mixed venous blood. The right or left subclavian vein, the right or left femoral vein and the inferior vena cava were also monitored. No measurements were made over peripheral arteries.

The equipment employed was, for the precordial (pulmonary) artery and the peripheral vein monitoring, the special 5-MHz CW Doppler ultrasonic flowmeter with deep focusing and shallow focusing detectors, respectively (8). This system provided for transcutaneous detection of bubbles moving in the stream of blood.

The subject, trained in placement of the transducer over the desired blood vessels, held the transducer in position while the investigator carried out both auditory monitoring to assure correct transducer placement, and magnetic tape recording of the detected signals for subsequent analysis. Placement for the pulmonary artery was over the third or fourth intercostal space at the left lateral border of the sternum.

Signals obtained from over the pulmonary artery were analyzed by a counter responding to the impulses recorded on the magnetic tape; these are tabulated as counts or "bubbles" per minute (bpm). Signals obtained from the other locations examined were graded only qualitatively in terms of intensity of the summation of Doppler effects.

Measurements were made on the subjects of Phase II during excursion-decompressions and during decompression from the saturation exposure to helium at the 1200-fsw pressure equivalent.

RESULTS

The Doppler bubble detection observations for each of the four subjects studied (CC, GM, MP, FS) are presented in Table I for excursion exposures and in Table II for saturation-decompression.

The excursion-decompressions produced bubble counts in all individuals, with differences among individuals and in the monitored locations. During the excursion-decompressions, higher bubble counts were detected with the pulmonary artery probe than during the saturation-decompressions, when they were routinely found only with muscle contraction. Signals from the subclavian vein during the saturation-decompression were relatively free of bubble indications (Doppler effects) while those from the femoral vein measurements showed evidence of bubbles more frequently. Monitoring over the inferior vena cava was done in only one subject (GM) during saturation-decompression, with Doppler indications of bubbles throughout much of the period examined.

DISCUSSION

The ultrasonic Doppler detector monitoring indicated that bubbles were being carried in venous blood in most decompression circumstances of this study, both during decompression from excursions and in decompression from prolonged saturation with helium. Since the system is not truly quantitative, and exposure to very high ambient pressures is not fully comparable with exposure to lower pressures, the findings in these decompressions from 1600- and

TABLE I. Detection of Intravascular Bubbles During Decompression from 400-fsw Excursion Exposures[a,b] (Phase II, 1200 to 1600 to 1200 fsw)

Stage	SUBJECT CC						SUBJECT GM						SUBJECT MP						SUBJECT FS					
	Elapsed Time[c] (hr·min)	Depth (fsw)	PA (bpm)	SV	FV	IVC	Elapsed Time[c] (hr·min)	Depth (fsw)	PA (bpm)	SV	FV	IVC	Elapsed Time[c] (hr:min)	Depth (fsw)	PA (bpm)	SV	FV	IVC	Elapsed Time[c] (hr:min)	Depth (fsw)	PA (bpm)	SV	FV	IVC
EXPOSURE DAY 2 - first excursion to 1600 fsw																								
Pre-excursion														1200	-	0	0	-		1200	0	-	-	-
During decompression from 1600 fsw	0:41	1300	-	+	-	-							0·41	1300	9	-	-	-	0:23	1335	14	-	-	-
	1:09	1245	314	-	-	-							1:11	1235	7	-	-	-						
	1:15	1225	-	++	-	-							1·19	1220	-	+	-	-						
	1:26	1210	139	-	-	-							1:23	1215	-	-	0	-						
	1:28	1205	-	-	+	-																		
Post-excursion	2:23	1200	147	++	++	-	1:25	1200	56	-	-	-	2·33	1200	7	0	-	+	2:08	1200	34	-	-	-
	4:47	1200	-	++	++	-							4:35	1200	54	+	-	-						
EXPOSURE DAY 3 - second excursion to 1600 fsw																								
Pre-excursion		1200	?	-	?	-								1200	10	+	-	-						
During decompression from 1600 fsw	0:30	1325	208	-	-	-							0·27	1330	2	?	-	-	1 06	1245	13	-	-	-
	0:33	1320	-	+	-	-							1:06	1245	13	-	-	-	1:14	1230	42	-	-	-
	0:36	1310	-	-	+	-							1:23	1215	23	-	-	+	1:24	1215	65	-	-	-
	1:11	1235	137	-	-	-																		
	1:13	1230	-	++	-	-																		
	1:16	1225	-	+	?	-																		
Post-excursion	2:38	1200	97	++	?	-	1·28	1200	87	-	-	-	2;19	1200	22	-	-	-	2;12	1200	51	-	-	-
							2:15	1200	126	-	-	-	7:28	1200	60	-	-	-	2 32	1200	95	-	-	-
							2:37	1200	51	-	-	++							3:34	1200	60	-	-	-
							3:36	1200	121	-	-	?												
AUGUST 15 - excursion to 1600 fsw for underwater work																								
Pre-excursion																								
During decompression from 1600 fsw	0:10	1400	4	-	-	-							0:13	1385	4	-	-	-	0:06	1420	2	-	-	-
	0:19	1360	21	-	-	-							0:22	1340	8	-	-	-	0:10	1400	3	-	-	-
	0:32	1311	17	-	-	-							0:38	1300	8	-	-	-	0·13	1385	4	-	-	-
	0·44	1286	46	-	-	-							0:49	1274	10	-	-	-	0:17	1365	7	-	-	-
	0·55	1267	77	-	-	-							1:00	1251	4	-	-	-	0:26	1330	19	-	-	-
	1:08	1239	37	-	-	-							1:13	1226	13	-	-	-	0:31	1320	12	-	-	-
	1:28	1204	104																0·37	1300	7	-	-	-
																			0·45	1285	37	-	-	-
																			0:52	1270	29	-	-	-
																			1·01	1255	68	-	-	-
																			1·06	1240	30	-	-	-
																			1:13	1230	45	-	-	-
																			1:21	1215	287	-	-	-
																			1:27	1205	171	-	-	-
Post-excursion	2·09	1200	350	++	++	-							1·33	1200[d]	11	-	-	-	1·36	1200	276	-	-	-

[a] Locations are indicated by: PA, pulmonary artery (mixed right heart blood), SV, subclavian vein; FV, femoral vein, IVC, inferior vena cava.

[b] Results are indicated by the following symbols. ++, continuing flow of bubbles; +, sporadic bubbles or appearance only with movement, ?, questionable occurrence of bubbles, 0, no bubble detection signals; -, no measurements made.

[c] Elapsed time is time from start of decompression from 1600 fsw

[d] Subject MP experienced vertigo 58 minutes after reaching 1200 fsw (see Section G-4)

TABLE II. Detection of Intravascular Bubbles During Decompression from Saturation Exposure[a,b,c]

Decompression Day	Date	SUBJECT CC: Clock Time (hr·min)	Depth (fsw)	PA (bpm)	SV	FV	IVC	SUBJECT GM: Clock Time (hr:min)	Depth (fsw)	PA (bpm)	SV	FV	IVC	SUBJECT MP: Clock Time (hr:min)	Depth (fsw)	PA (bpm)	SV	FV	IVC	SUBJECT FS: Clock Time (hr:min)	Depth (fsw)	PA (bpm)	SV	FV	IVC
1	8/17	10·40	1400	8	-	-	-	10·35	1400	-	-	-	?	10·33	1400	?	-	-	-						
		19:38	1338	1	-	-	-	18.53	1336	-	-	-	0	19:47	1337	4	-	-	-	19:57	1335	4	-	-	-
2	8/18	18 03	1200	4	0	-	-	18 00	1200	-	-	-	?	18 00	1200	11	-	-	-	16·20	1200	2	-	-	-
3	8/19							9·15	1057	-	-	-	?	9.05	1059	11	-	-	-	9:09	1058	9	-	-	-
		14:09	1008	-	0	-	-	14:20	1006	-	-	-	?	14:17	1007	-	0	-	-	·14:12	1008	4	-	-	-
4	8/20													9 08	917[d]										
		10 06	903	-	?	-	-							10 13	903	7	0	-	-	10·01	904	1	-	-	-
								16:14	851	-	-	-	+	15·39	856	-	?	+	-						
		17 22	841	4	+	+	-	17:13	843	-	-	-	+	18:30	831	4	+	+	-	18:19	834	3	+	-	-
5	8/21	11 20	687	2	+	+	-	10:24	695	-	-	-	?	10·16	696	2	0	-	-	11:04	690	1	+	-	-
		15 06	655	3	+	+	-	15:28	652	-	+	-	+	14:56	656	2	?	+	-	15:15	654	4	+	+	-
		20·42	650	2	+	+	-							20:53	650	-	+	+	-						
6	8/22	10 29	560	5	+	+	-	10.10	563	-	-	-	+	10·40	559	8	0	+	-	10:20	561	4	+	+	-
								15 45	521	-	+	-	+	16:30	516	4	?	+	-	15:08	526	7	+	+	-
		19 30	493	34	+	+	-	19:36	492	-	?	-	-	20 12	488	1	0	+	-	20·02	489	9	+	+	-
7	8/23	10.54	422	14	+	+	-	10.34	424	-	0	-	+	10:24	425	4	?	+	-	10·42	423	19	+	+	-
		15.39	390	6	0	+	-	15:31	391	-	0	-	+												
														18:18	372	8	0	+	-	18:27	371	22	0	+	-
8	8/24	12·00	275	30	?	+	-	10:34	275	-	-	-	+	11:51	275	5	?	+	-	10:43	275	44	-	+	-
		16:07	261	10	+	+	-	14:46	269	-	+	-	+	16 15	260	10	0	+	-	14:57	268	31	?	+	-
		19.32	246	31	?	+	-	18 36	246	-	0	-	+	19·38	246	9	+	+	-	19:43	245	14	0	+	-
9	8/25	10:36	200	7	?	+	-	10:43	200	-	0	-	+	10 28	200	12	0	+	-	10:49	200	10	0	+	-
		16 24	200	4	?	+	-	15:56	200	-	0	-	?	16·03	200	15	0	+	-	16:10	200	2	0	+	-
		19:31	200	1	0	?	-							19:08	200	15	-	0	-	19·15	200	14	0	+	-
10	8/26	11:00	155	-	0	0	-	10·43	157	-	0	-	+	10 53	156	8	0	+	-	10·39	157	-	0	+	-
		15:37	132	?	0	0	-	15:46	132	-	0	-	+	15:30	133	+	?	+	-	15:23	133	+	0	+	-
		19:24	113	+	0	+	-	19:36	112	-	0	-	?	19:12	114[e]	+	0	+	-	19 30	113	9	0	+	-
11	8/27	7:35	45	16	0	?	-	8:02	40	-	0	-	+	7.49	43	14	?	+	0	7·28	45	29	?	+	-
			40[f]					8:38	35	-	-	-	?												
		9:48	24	8	0	+	-	9:41	25	-	?	-	-	10 03	22	7	?	+	-	10.12	21	26	0	+	-
														12 00	11	-	-	+	-						
														12 00	60	-	-	0	-						
Post-exp.	8/28		0[g]	-	0	0	-		0	-	0	-	0		0	?	-	-	-		0	?	0	?	-

[a]Saturation-decompression in Phase II began at the equivalent pressure of 1400 fsw All subjects had been at 1400 fsw for 42 hours for therapy of decompression sickness in subject MP.

[b]Locations are indicated by: PA, pulmonary artery (mixed right heart blood), SV, subclavian vein, FV, femoral vein; IVC, inferior vena cava

[c]Results of ultrasonic bubble detection are indicated by the following symbols· ++, continuing flow of bubbles; +, sporadic bubbles or appearance only with movement; ?, questionable occurrence of bubbles; 0, no bubble detection signals, -, no measurement made.

[d]Subject MP reported bilateral knee pain.

[e]Subject MP reported right knee pain

[f]Subject CC reported "niggles" both knees at 40-35 fsw (see Section G-4).

[g]Subject CC reported bilateral knee pain.

1200-foot pressure equivalents cannot be strictly compared with the numerous instances of relatively shallow excursion in which evidence of bubble detection has already been obtained (8).

Qualitative comparison is entirely practical, however. Decompression sickness involving vertigo did occur in one subject (MP) on whom bubble detection monitoring was performed following a 400-foot pressurization excursion (Section G-4). The total counts or "bubbles" per minute registered over the pulmonary artery in that subject during the decompression which resulted in his symptoms were smaller than in the other subjects for the corresponding excursion (Table I). Subject MP also had fewer detected "bubbles" than the other subjects for the 400-foot excursions in which symptoms did not occur (Table I). No prominent differences in counts of "bubbles" per minute were obtained over the pulmonary artery in the three subjects so examined during saturation-decompression (Table II). The significance of these findings to pulmonary function, and to development of subjective symptoms of decompression sickness, is not now established.

Even though specific correlations between counts of venous bubbles and decompression sickness incidence could not be derived, the results obtained met the several purposes for which the ultrasonic monitoring was used in this Predictive Study. An especially important result of the monitoring was the demonstration of venous bubble emboli in situations not previously studied. Bubbles were detected both in the deep excursion decompression and saturation-decompression, indirectly indicating that a gas phase exists in both situations. While the decompression used for the 400-foot excursions was about 10 times shorter than is now customary for equivalent 400-foot depth, one-hour excursion exposure from sea level (Section G-1), the monitoring did not indicate the occurrence of fulminating and progressively increasing venous bubbles. However, Doppler indications of gas bubbles were found and where such evidence of bubbles exists, rates of decompression cannot rationally be further increased.

LIMITATIONS OF INTERPRETATION

The usefulness of ultrasonic detection of bubbles by Doppler flow transducers in the study of decompression and decompression sickness will become more clear only with continued applications of the method over a wide variety of different forms of compression-decompression exposures, and with more precise monitoring of specific effects comprising decompression sickness. Under the particular conditions of the Predictive Studies IV program, certain known limitations of the bubble detection method apply. These limitations include the following:

1. It is necessary that bubbles, to be detected, be in motion. Therefore, only those bubbles moving freely through large veins are detected. Bubbles in tissue extravascular spaces or in the low velocity flow stream of peripheral tissue or pulmonary capillaries will not induce recognizable signals.

2. Flow of blood within large veins derives from an enormous number of tissue sites. The detection of bubbles in venous blood indicates the existence of excess gas saturation somewhere but cannot indicate the anatomical location or perfusion/composition characteristics of the tissue from which the actual gas phase (bubbles) derived.

3. In spite of important qualitative usefulness of Doppler bubble detection, it is not now practical with in vivo use of the method to correlate the output of the detector transducer with individual bubble size or with volume flow of gas phase passing the detector (e.g. when monitoring over the pulmonary artery). It is practical to obtain such correlation in vitro (5), where calibration of the transducer system is possible, and the method as used in vitro certainly provides a qualitative index of the severity of the embolic process.

4. The Doppler flow transducer (5 MHz, 10 milliwatt) used in Predictive Studies IV had a lower limit to the bubble size which could be detected. This limit is estimated as 1 μm (one micron) in diameter, close to theoretical estimates of bubble size in decompression (6), and close to "supercritical size" for bubbles in blood (7). While this detection capability should cover

bubbles which have expanded beyond supercritical size, the precipitating circumstances--both for decompression and for isobaric inert gas counterdiffusion--concern initial growth of bubbles from nuclei as well as the subsequent local growth and the intravascular transport of larger bubbles.

In spite of such recognized limitations, which still allow usefulness of the ultrasonic Doppler method in studies of decompression, the results obtained in Predictive Studies IV indicate that bubbles were transported in the venous blood of all subjects during decompression from the 400-foot excursion exposures. It cannot yet be known whether the individual size and the collective volume of such bubbles were more serious in implication than in the case of the bubbles detected in "no-decompression" air diving with standard Navy tables (8). It is entirely possible that many bubbles were formed but remained small and undetected, with little growth by expansion during the decompression from 49.3 ata to 37 ata. This would be in marked contrast to the over 12-fold increase of individual bubble size and collective bubble volume to be expected by expansion in rapid decompression from 400 fsw (13.1 ata) to one atmosphere.

While the Doppler detection method cannot itself distinguish among factors affecting formation of bubbles, bubble growth and bubble expansion, the method should be useful for such purposes when employed in animals in conjunction with other methods.

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G-4. THERAPY OF DECOMPRESSION SICKNESS

K.M. Greene[1] and C.J. Lambertsen[1]

Protocols for the management of decompression sickness (DCS) devised during the planning for Predictive Studies IV involved separate consideration of the novel decompressions from deep excursions (Section G-1) and the decompressions from saturation (Section G-2). A survey of the literature indicated that cases which might occur during decompressions from deep excursions differed in type, severity and responsiveness to therapy from those which might occur during ascents from saturation. Consequently, plans for different treatment methods were prepared.

The actual experience in Phases I and II of the Study involved treatment of three cases of DCS originating in decompressions from excursion and three in decompressions from saturation. The excursion cases involved two with inner ear symptoms and one with spinal cord manifestations. All were promptly and completely relieved by treatment. The saturation cases involved the knees. Two were resolved without difficulty while the third required a protracted course of therapy.

DECOMPRESSION SICKNESS IN SATURATION-DECOMPRESSION

BACKGROUND

Recent experience in decompressions from helium-oxygen saturation dives can be generalized as follows: There is a low incidence of DCS in decompression from relatively shallow

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depths, principally involving knee pain,with onset frequently occurring near the surface or even after surfacing (19, 29,34). Decompressions from deeper saturation dives have produced inner ear as well as knee symptoms (15,19,25); neurological symptoms have occurred in a few cases (19). Even highly conservative decompression profiles have resulted in DCS. Repetitive excursions during the dive do not appear to alter the outcome of the subsequent saturation-decompressions provided the excursions are symptom-free (6,14,15) although one report (28) may not substantiate this (see also Section G-2).

Reports on the therapy of saturation DCS indicate that complete relief does not result in all cases, and some indication exists that excessive recompression (greater than 30 fsw) may aggravate symptoms (29). Another study found that there was no correlation between depth of onset and amount of recompression required for relief (19). In most cases some relief is obtained with recompression of as little as one atmosphere. Hyperoxic levels of 0.4 ata (19) and 1.5 to 2.5 ata (29) have been employed during therapy at increased ambient pressure.

PROTOCOL FOR THERAPY DURING SATURATION-DECOMPRESSION

The protocol for therapy of saturation DCS in Predictive Studies IV was based on the decompression schedule itself (Section G-2). This schedule was computed to keep the degree of excess inert gas pressure (ΔP) in the controlling tissue between 41 and 12 fsw, depending on depth (Table II, Section G-2). For a bubble in equilibrium with gas pressures in tissue fluid or venous blood at some excess pressure (ΔP) above ambient, the partial pressure gradient for the inert gas would be reversed with a recompression in the amount [ΔP + venous ($P_{O_2} + P_{CO_2} + P_{H_2O}$)] (ignoring bubble surface tension). This amounts to a recompression of about 25 fsw for depths less than 200 fsw, the region where most cases of DCS associated with saturation-decompression have been reported. If pathological consequences of DCS retard inert gas exchange, then the actual local ΔP at the site of bubbles could be greater than this value.

On this basis the plan developed for treatment of DCS occurring deeper than 100 fsw involved a 50 fsw

recompression and use of inspired oxygen partial pressures between 1.5 to 2.5 ata. Between 100 fsw and atmospheric pressure (following the scheduled transition to air breathing, see Section G-2), therapy employed recompression with various nitrogen-oxygen or helium-oxygen breathing mixtures, or with pure oxygen, depending on depth of onset and degree of recompression needed for relief. Provisions were made for precise monitoring of oxygen pressure-time exposures to stay within safe oxygen exposure limits (7,8). It was planned to recompress rapidly to the depth of relief should serious symptoms develop.

CASE REPORTS OF DECOMPRESSION SICKNESS DURING SATURATION-DECOMPRESSION

Three cases of DCS (one in Phase I, two in Phase II) involving pain in one or both knees occurred during the saturation-decompression periods of these exposures. Two of these occurrences of joint pain were atypical in that the onset was deep. Both subjects had been treated earlier for DCS resulting from excursions. The third was typical in that the depth of onset was shallow and there was no DCS in the preceding excursions.

Information concerning the instances of DCS and their therapy will be provided as case descriptions. Cases are identified by a Roman numeral identifying the study phase (I or II), initials identifying the subject, and saturation or excursion signifying whether it occurred during a saturation or an excursion decompression.

Case ILJ-Saturation

During ascent from 1050 fsw to the surface, subject LJ had insidious, intermittent left knee pain with uncertain onset at approximately 500 fsw. Symptoms did not appear definite until about 12 hours after discomfort was first reported. With recompression from 414 fsw to 465 fsw at about 30 fsw/minute, substantial relief was obtained within 20 minutes, but residual soreness persisted even after intermittent breathing of oxygen at increased partial pressure (20 minutes of 2.1 ata P_{O_2}, 5 minutes at natural P_{O_2}, for 10 cycles). The total dose of oxygen during this treatment amounted to 520 UPTD (unit pulmonary toxic dose)

units (35). At the end of the therapy the subject had mild paresthesias of his fingers and toes. Five days earlier he had been treated successfully for inner ear DCS (see case ILJ-Excursion) and had received 460 UPTD units at a P_{O_2} of 2.3 ata without symptoms of oxygen toxicity.

Decompression was resumed after six hours at 465 fsw. The finger tip paresthesias recurred during the final decompression from 30 fsw (546 additional UPTD units) but no neurological deficit was found on examination. This subject had mild residual soreness of the knee upon reaching atmospheric pressure four days after the therapy at 465 fsw.

Case IIMP-Saturation

During the final ascent from 1400 fsw in Phase II, subject MP had a protracted problem with bilateral knee pain. The onset was insidious and indefinite, and was mentioned at 917 fsw. The discomfort was greater on the left than the right but it increased and decreased, did not prevent sleep, and could be relieved by taking weight off the leg or by rest. Because of uncertainties related to the depth of onset and a history of minor trauma and compression arthralgia, it was not immediately treated by recompression.

Over a four-day period the pain gradually resolved with aspirin (975 mg every four hours), rest, elevation, and local heat ad lib. MP was asymptomatic before reaching 200 fsw. Although the left knee gave no further trouble, the right one began to hurt again at about 115 fsw. The medical watch members were not made aware of the severity and progression of the pain until much later.

Recompression was then undertaken on oxygen from 11 fsw on a profile equivalent to USN Treatment Table 6. Substantial relief was obtained at 60 fsw, but a residual pain persisted and required another recompression from 38 fsw to 60 fsw for one more oxygen period. After a further seven hours of decompression and administration of 500 ml of dextran-40, 500 ml of 5% dextrose in water, local heat, and additional oxygen exposure, subject MP surfaced with moderate residual knee pain. This resolved over the next several days. However, during this final day of decompression he had breathed a total oxygen dose of 1222 UPTD

units. Following the second recompression to 60 fsw, he experienced substernal pain, dyspnea, paresthesias and numbness of mouth, hands and feet, and anxiety. These symptoms of oxygen toxicity were improved with a one-hour air break . After surfacing he had residual distal paresthesias and dyspnea on exertion, both of which gradually resolved during the two days following exit.

Case IICC-Saturation

Upon surfacing from the final decompression of Phase II, subject CC disclosed severe bilateral knee pains which had been increasing since they became evident at 10 fsw. Transient discomfort in both knees had previously been reported by this subject when pressure was reduced from 40 fsw to 35 fsw. He was easily relieved of DCS by recompression on standard USN Treatment Table 5. He too had some symptoms of oxygen toxicity at the end: namely, a mild tightness of the chest and finger tip numbness, following a total oxygen dose on that day of 735 UPTD units.

DISCUSSION--INCIDENTS IN SATURATION-DECOMPRESSION

The occurrence of pain-only DCS at depths of 500 fsw and 917 fsw in these exposures is not typical of the generally shallow depth of DCS onset following saturation dives, even those involving great depths. Since both subjects had previously been treated for DCS originating in excursion-decompressions, it must be considered that this might have left them more vulnerable to later DCS. Only six other cases of DCS in decompression from deep helium-oxygen excursions were known. One of these was associated with a subsequent Type I DCS during the saturation-decompression (14). Another was followed by a symptom-free saturation-decompression (33) while the results of the saturation-decompressions in the other four cases were not reported. The cautious approach to saturation-decompression after an episode of DCS should allow time for recuperation at the treatment depth before final ascent to the surface begins. However, there is no theoretical basis for determining the length of such a holding period and it must be arbitrary. Subject LJ had been at treatment depth for 39 hours while subject MP was at his treatment depth for 42 hours before decompressions from saturation began.

The recompression of subject LJ by only 51 fsw produced substantial relief, despite the insignificant volumetric change which would be predicted by Boyle's Law at that treatment depth (465 fsw). This result is consistent with the experience of others (19,29) and lends support to the authors' hypothesis that successful recompression therapy requires primarily a reversal of the inert gas gradient between the causative gas phase and the surrounding tissue.

The extent of recompression which might be needed in a saturation dive should be related to the decompression rate or to the ΔP designed into the schedule. A recompression of 30 to 60 fsw should be sufficient to cover most schedules in present use. This is the amount recommended in guidelines formulated by the European Undersea Biomedical Society (11). Excessive recompression is not usually productive (29) and complete relief of "pain-only" DCS is unusual in saturation diving; substantial relief should be sought even if total relief is not. However, in the rare event of serious symptoms (e.g., neurological or vestibular) during saturation-decompression, the initial therapeutic recompression should be greater in degree and more rapid, with actual relief being the endpoint sought.

The prolonged episode of bilateral knee pain in subject MP was, in retrospect, most likely DCS from the start, but indications of resolution of symptoms in spite of continued decompression initially weighed against that diagnosis. When treatment is greatly delayed and tissue reaction occurs, a dramatic response to recompression cannot be expected, and MP's response illustrates this well. Therefore, in dives not involving validation of decompression procedures, early recompression should be used to clarify the diagnosis in doubtful cases. Following therapy, decompression should be resumed from the treatment depth after an appropriate delay. A hold of two to six hours has been recommended (11). Longer holds of 12 to 24 hours may be considered when the residual symptoms are substantial or when the response to treatment is slower than usual.

The planned use of increased oxygen pressures during decompression should be purposely limited to allow a substantial reserve for oxygen tolerance for any DCS therapy that may be needed. The pressure-time history of hyperoxygenation should be carefully recorded in terms of UPTD

units (35) with the total dose (including reserve for an oxygen therapy) in a single decompression held below 1200 units--which is about twice the dose administered in a USN Treatment Table 6. This dose is equivalent to eight hours at a P_{O_2} of 2 ata, an exposure which produces prominent symptoms in normal man and a reversible 8% decrement in vital capacity in 50% of subjects exposed (7,8). Subject MP received a dosage of approximately 1200 units and had significant discomfort from pulmonary toxicity, despite cyclic interruptions in the hyperoxic breathing. The EUBS guidelines (11) call for limiting hyperoxygenation incorporated in the decompression plan to 600 UPTD units.

In the event that a large oxygen dose is used for therapy, prior to completion of a decompression with planned additional hyperoxia, modification of the subsequent decompression procedure may be required to minimize additional hyperoxic exposure. A slow, helium-oxygen schedule may be used with normoxic mixture or, if transfer to air has been done, an air-saturation schedule without added oxygen can be used (18). If treatment is necessary during the deeper phase of a dive, with further hyperoxic-mixture breathing not scheduled until more than 24 hours later, then the original schedule can be followed, with vigilance concerning oxygen effects.

When DCS occurs after a transfer to air, the recompression gas must be chosen according to the depth of onset, nature of symptoms and response to treatment. When the onset is shallow enough to permit recompression on pure oxygen, treatment on a profile equivalent to USN Treatment Table 6 should be tried. If relief is obtained, decompression may be resumed on the original schedule or on Table 6, whichever is the longer. When substantial relief is not obtained at 60 fsw, further recompression is needed, on helium-oxygen to avoid a nitrogen saturation exposure at excessive depth. This recompression can be achieved by adding pure helium to the 60 feet of air, or by transferring the diver to a helium-oxygen filled compartment with a P_{O_2} less than 0.6 ata. This should be tolerable for many days unless pulmonary oxygen toxicity has already developed.

If the onset of symptoms has been on air but at deeper than 50 fsw, only the latter methods of helium recompression should be used to assure that the chamber P_{O_2} is safe for prolonged exposure.

The depth of recompression with helium after the air shift should be governed by the diver's response to the therapy. A transient supersaturation has been predicted (23) and actually found to result (9,20) from the difference in exchange rates for nitrogen and helium in this circumstance, but additional depth should compensate for this phenomenon (16). An initial 60 fsw recompression with helium can be followed by additional increases of 100 fsw or more if symptoms worsen.

If serious symptoms such as inner ear DCS occur following the transfer to air, the diver should be immediately returned to helium-oxygen and rapidly recompressed by at least 100 fsw, a procedure which has been successful (36). It must be emphasized that once a return is made from air back to helium the subsequent decompression must take this into account and should be more conservative than the routine schedule.

DECOMPRESSION SICKNESS IN EXCURSION-DECOMPRESSION

BACKGROUND

In planning the procedures for therapy of deep excursion DCS which might occur in Predictive Studies IV, the limited recorded prior experience of DCS in deep diving in general was reviewed. Surprisingly few decompression accidents have occurred in the development and testing of excursion-decompression procedures. The procedures used initially were clearly conservative in light of more recent developments. Consequently, there is little empirical information concerning what forms DCS may take in excursion-decompressions and what types of therapy would be effective.

SATURATION-EXCURSION

The earliest reported systematic excursion trials were from saturation on air at 35 fsw, with 17 man-excursions to depths of 100 to 165 fsw (24); all excursions were free of DCS. The US Navy continued this developmental work through the Sealab series, using the Workman model of allowable supersaturation (4). A total of 1123

man-excursions were made from saturation depths ranging from 150 to 850 fsw, all on helium-oxygen, without DCS (14) No cases of DCS were reported after 4000 hours of excursion diving at sea and in the laboratory, using schedules similar to the US Navy procedures (22). Recent experiments at the Navy Experimental Diving Unit have greatly extended the excursion profiles permissible in helium-oxygen diving (14). During these experiments two episodes of DCS occurred. The first followed a five-hour excursion to 450 fsw from saturation at 300 fsw; it consisted of pruritus and chest discomfort and was 95% relieved by recompression to 350 fsw and hyperoxic breathing mixture. The second (31) occurred after this study was completed; it consisted of a spinal cord lesion following an ascending excursion from saturation at 1000 fsw up to 800 fsw. Relief was obtained by returning to 1000 fsw.

Four other cases of DCS are known to have resulted from helium-oxygen excursions. Three of these occurred during a "Ludion" procedure of repetitive excursions from 120 meters of seawater (msw) to 180 msw (15). All three were vestibular disorders and were relieved by recompression and drugs; the treatment depth was not reported. Another incident of vertigo and nausea occurred after six excursions in two days between 250 and 300 msw, during the British Royal Navy saturation diving trials (33); this was relieved by immediate recompression to 300 msw.

During an earlier phase of the Royal Navy trials, saturation-decompressions were made in a stepwise fashion, resembling no-stop ascents from 24-hour excursions (1). During these dives ("Phase I" of reference 19), seventeen cases of DCS occurred, of which two involved inner ear symptoms. The remainder consisted of joint pain, three of which also had local neurological symptoms. No correlation was noted between the depth of onset of symptoms and the differential pressure (ΔP) needed for relief. One case with inner ear symptoms was successfully managed by recompression from 47 msw to 69 msw, while the other improved slowly after recompression from 47 msw to 75 msw. In none of these 17 cases was more than a 30-msw recompression applied.

In both ascending and descending excursions from saturation with nitrogen-oxygen, the only manifestations

of DCS reported have been joint pain and pruritus (3,18). These are not directly applicable to the Predictive Studies IV program because of the differences in depth range as well as in inert gas used.

OTHER DEEP DIVING

Since experience concerning the nature and treatment of DCS in deep excursion diving was limited, correlations with other kinds of pressure exposures were sought as aids to planning. For two reasons it seemed likely that extreme excursions at great depths might produce DCS resembling that which occurs during decompression from very deep nonsaturation diving from the surface. First, in both cases large inert gas gradients are produced. Second, the volume response of the gas phase to depth changes at greater depths would be similar to that during excursion-decompressions.

A review of USN experience (32) showed that in helium-oxygen subsaturation dives, Type II decompression sickness comprised 29.8% of incidents and the onset depth for symptoms was approximately proportional to the dive depth. The probability of successful treatment could be related to the ratio of absolute depth of treatment to depth of onset. The probability of relief of Type II decompression sickness was better than 96% if this ratio were 1.9:1 or greater.

In 23 cases observed during decompression from Royal Navy deep experimental helium-oxygen dives, 6 had symptoms of neurological or inner ear disease (2). The four inner ear episodes had onset deeper than 200 fsw but required recompression ratios of 1.99 to 1 or less. The mean ΔP for the four was 178 fsw and the maximum was 270 fsw; not all treatments were completely successful.

Other investigators have noted a tendency toward inner ear symptoms (vertigo and/or hearing loss) as a major manifestation of DCS in deep subsaturation diving (21,30). Immediate recompression to the depth of the dive is reported to have been successful in a series of experiments at 650 fsw (30), when onset of DCS was at 470, 450 or

390 fsw. Prompt treatment by recompressing 100 fsw below the depth of onset has been reported to relieve vertigo or deafness without residual effect (13). A report on eleven cases of inner ear DCS in helium-oxygen diving to depths from 16 to 31 ata (5) showed six to be alleviated by recompressions averaging 80 fsw for a ratio of 1.24 to 1 (one of these divers was relieved of vertigo but was left with a high frequency hearing decrement; no pre-dive audiometric control was available). The other five individuals in this group had significant residual inner ear defects. Their recompression ratios also averaged 1.24:1 but since the onsets were generally deeper, the average ΔP was greater (117 fsw) than that of the individuals who were completely cured. None of these cases, however, was returned to the full dive depth for treatment.

The pressure ratios which resulted in successful treatment in the cases cited above are clearly not as large as those required for therapy of surface-onset DCS. Even those cases recompressed to the full depth of the dive did not have recompression ratios approaching the 6:1 ratio of the once widely used 165 fsw air treatment table. The degree of recompression used in compression to full depth of dive, however, rarely exceeded 200 fsw, which is close to the 165 fsw of the air treatment table.

These observations lend support to the hypothesis which guided planning of treatment contingencies for Predictive Studies IV: namely, that successful treatment of DCS of deep onset may require only the reversal of the inert gas gradient between tissue or blood and bubbles (17). In the worst case, this reversal can be accomplished by returning to the maximum depth of excursion.

In planning for Predictive Studies IV, the possible handicap to therapy presented by neurological effects of deep recompression was considered. An illustrative example is one instance of inner ear DCS which occurred at great depth and consisted of incapacitating vertigo after a rapid decompression from saturation at 1500 fsw (25,27). Recompression from 1160 to 1535 fsw ameliorated but did not eliminate symptoms, but both the depth and rate of recompression were limited by symptoms of the "high pressure neurological syndrome" (HPNS) in the other subject. The resulting slowness of the recompression was probably a major factor in the failure of treatment. Other authors have noted the clear correlation

between prompt treatment and successful outcome (2,12,32). It was because of this problem that the initial excursion trials in Phase I of Predictive Studies IV were made from saturation at 800 fsw. Therapeutic compression from this depth, even to the full excursion depth (1200 fsw), was not expected to be limited by HPNS. Information gleaned from Phase I about compression rate tolerance guided planning for Phase II, in which HPNS might limit compression rate from 1200 fsw to 1600 fsw.

In summary, previous experience showed that DCS resulting from excursion diving was likely to consist of serious symptoms which could be treated adequately if treatment were prompt. Treatment plans were therefore laid out in advance for use in these Predictive Studies excursions. They called for recompression at a rate up to 30 fsw/minute to the depth of relief, not to exceed the depth of the excursion unless demanded by the subject's condition. The therapeutic depth selected was to be held at least 12 hours before decompression. Electronystagmography and bone conduction audiometry were available in the chamber to assess the results of treatment of inner ear symptoms. Hyperoxic mixtures would be given in up to ten 20-minute cycles. Supplemental drug therapy was available, and the subjects were trained to administer intravenous infusions.

CASE REPORTS OF DECOMPRESSION SICKNESS IN EXCURSION FROM SATURATION

One incident of spinal cord DCS and one involving vertigo occurred in Phase I. One case involving vertigo occurred in Phase II. All three subjects responded to prompt recompression and there were no residual deficits.

Case IWS-Excursion

On his exposure day 3 of Phase I, subject WS (with partner FS) was being decompressed back to 800 fsw from his third excursion to 1200 fsw. The excursion profile (Fig. 1) was a 40-minute compression and 55-minute additional actual bottom time. Normoxic helium-oxygen was breathed during the compression and bottom phases. The scheduled decompression duration was 89 minutes with 2.7% oxygen by mask throughout. At 830 fsw (71 minutes elapsed decompression

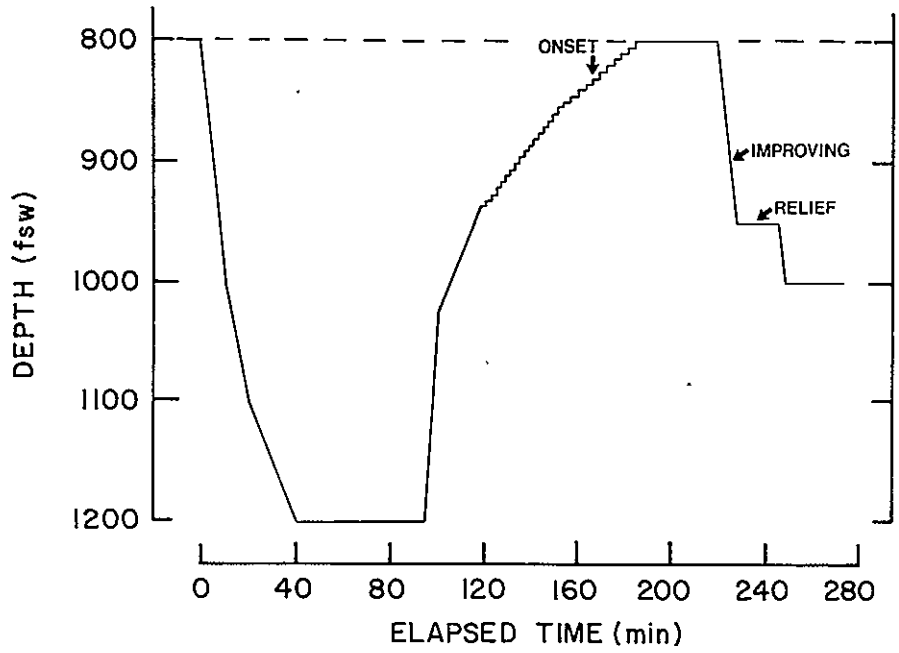

FIG. 1. Exposure profile showing occurrence and treatment of decompression sickness. Subject WS, Phase I, exposure day 3 (Case IWS-Excursion).

time) subject WS noted the onset of paresthesias and numbness in his right leg and thigh. Although he was the "exercise" subject and was seated on the bicycle ergometer, no exercise was performed during the decompression. It was first considered possible that he had a simple sciatic neurapraxia from the pressure of his thigh against the seat. Decompression continued while he walked about the chamber and also lightly pedalled the ergometer to aid diagnosis. After the return to 800 fsw, the paresthesias spread downward to his toes, and upward, eventually reaching the T-6 dermatome on the right. He also had slight weakness and tremor of the right leg and aching in the right flank. As part of the diagnostic procedure one of the other subjects elicited the following signs under supervision: hyperesthesia to pin prick on the right trunk, leg and thigh; decreased temperature sense on the right; and normal patellar tendon reflexes.

Recompression of all four subjects (for operational-safety considerations) was begun 52 minutes after the first

mention of symptoms. The rate of descent averaged 16 fsw/minute. Treatment gas was administered by mask in cycles of 20 minutes interrupted by 5 minutes of chamber atmosphere ($\bar{P}O_2$ = 0.2 ata) with the first two cycles consisting of 7% oxygen, balance helium followed by 8% oxygen for six more cycles. The flank pain was relieved while passing 900 fsw and recompression was stopped at 950 fsw because the remaining symptoms began to regress. All signs and symptoms had resolved within 19 minutes of leaving 800 fsw. Although the DCS was apparently overcome at 950 fsw, the subjects were recompressed to 1000 fsw as an added safety measure, reaching that depth 30 minutes after leaving 800 fsw. Decompression back to 800 fsw began 12 hours and 34 minutes after the start of therapy at the rate of 7 minutes/fsw and was eventful. As a precautionary measure subject WS was given two cycles of 7% oxygen breathing at the beginning of decompression. He made no further excursions and had no DCS during the final ascent to the surface. Detailed neurological examination after surfacing revealed no residual deficit.

ILJ-Excursion

On his exposure day 5 of Phase I, subject LJ incurred inner ear DCS after his third excursion to 1200 fsw, which began 25 hours and 50 minutes following his return to 800 fsw after the treatment of WS. Compression was 40 minutes in duration and the actual bottom time was 55 minutes (Fig. 2). The decompression schedule for this excursion was the recomputed version (see Section G-1) which would have required nearly twice the time of the original.

During the stay at the excursion depth of 1200 fsw this subject felt nauseated and dizzy, but nystagmus was not present. He did not report difficulty in equalizing his ears during either descent or ascent. The symptoms resolved at about 945 fsw during decompression, but at 850 fsw he reported he was "spinning," and a pronounced right-beating nystagmus was seen simultaneously on the electronystagmogram (ENG); this reached a maximum of 40°/second for the slow component (Section E-6). He soon developed nausea but did not vomit.

Recompression was begun from 845 fsw within five minutes of onset and was halted at 1050 fsw nine minutes later, since the vertigo and nausea were improving. Therapeutic

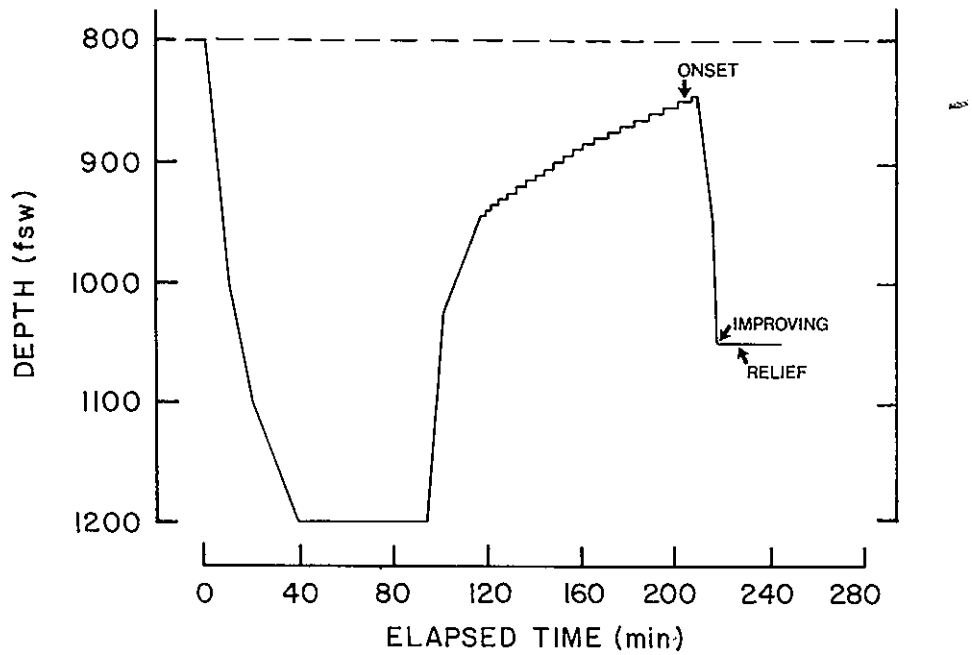

FIG. 2. Exposure profile showing occurrence and treatment of decompression sickness. Subject LJ, Phase I, exposure day 5 (Case ILJ-Excursion).

gas (7% oxygen, balance helium) was given in a total of eight 20-minute cycles, interrupted by five minutes of chamber atmosphere, beginning while passing 875 fsw. The ENG leads were disconnected temporarily to allow subject LJ to lie on a cot but the tracing was restored seven minutes after reaching 1050 fsw. It then showed greatly reduced nystagmus which had changed to left-beating. Two minutes later there was no nystagmus and no vertigo. Despite the change in direction of the ENG, LJ did not feel any change in the direction of the vertigo (environment spinning counterclockwise). There was no subjective hearing loss, but transient right-sided tinnitus was noticed at 1050 fsw. Bone conduction audiometry at this depth showed an equivocal 15 dB decrease at 4 kHz on the right (Section E-7). Repeated, more extensive measurement about 3.5 hours after onset showed no loss of auditory acuity, no spontaneous nystagmus, minimal positional nystagmus, and slight decrease in the threshold for electrical vestibular stimulation.

Although he had no further inner ear problems, this subject did have an episode of knee pain DCS during the final ascent to the surface (ILJ-Saturation). Examination at the surface revealed normal vestibular responses to warm and cold caloric tests. Audiometry on the day after surfacing showed a uniform decrease averaging about 15 dB bilaterally in bone and air conduction compared with predive control. The next day the decrease averaged about 25 dB, but five weeks later the audiogram was the same as predive control. ENT physical examination was normal upon surfacing, as was the remainder of the neurological examination.

IIMP-Excursion

On his exposure day 5 of Phase II subject MP developed inner ear DCS following decompression from his third excursion to 1600 fsw on his fifth day at pressure. The excursion profile was a 20-minute compression from 1200 fsw to 1600 fsw, 55-minute actual bottom time with hard work underwater on a scaled-down oil wellhead (Section F), and an 89-minute decompression (Fig. 3). While underwater he

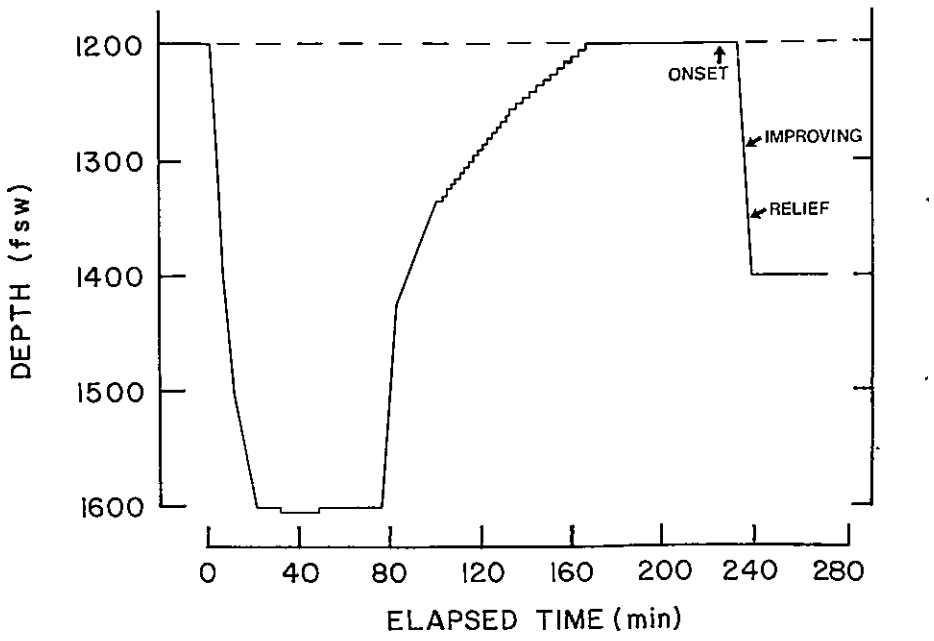

FIG. 3. Exposure profile showing occurrence and treatment of decompression sickness. Subject MP, Phase II, exposure day 5 (Case IIMP-Excursion).

was immersed in fresh water (33.6°C) at an average pressure equivalent to 1605 fsw. The breathing apparatus was a modified Kirby-Morgan Bandmask, supplied with 2% oxygen in helium. MP breathed this 2% oxygen mixture for 19 minutes, the final 16 minutes in the water. He breathed chamber gas for the remaining 28 minutes at 1600 fsw while acting as tender for subject CC. The chamber oxygen concentration was initially normoxic, but it rose to 0.5 ata by the end of the bottom time due to admixture with the hyperoxic breathing apparatus exhaust. A mixture of 3.2% oxygen in helium was breathed by mask during the decompression. Although MP did not dry himself or remove his wet clothes while serving as tender for CC or during the decompression, he covered himself with a blanket and did not complain of being cold.

Fifty-nine minutes after reaching 1200 fsw, immediately after eating dinner, MP experienced the sudden onset of vertigo. Although he had no nausea, the vertigo was prominent, and he was assisted to a cot. Immediately prior to onset of vertigo, he had performed the Valsalva maneuver repeatedly, with the comment that his right ear felt "stuffy" and he had been trying to clear it. He had no tinnitus and no noticeable hearing loss.

Recompression was begun seven minutes after the initial complaint of vertigo, at about 30 fsw/minute. The vertigo stopped while passing 1350 fsw, five minutes after leaving 1200 fsw. Recompression was halted at 1400 fsw, two minutes later. The treatment gas mixture (5% oxygen, balance helium) was started at 1290 fsw and was continued for six cycles of 20 minutes each. Electronystagmography was performed five minutes after reaching 1400 fsw, and no spontaneous nystagmus was seen. Positional stimulation did not produce nystagmus, and no significant change was seen in bone conduction audiometry. Although this subject later suffered knee pain DCS, he had no further inner ear symptoms. On the day after surfacing he had normal caloric responses bilaterally and no significant change in audiometry from his predive control.

DISCUSSION AND RECOMMENDATIONS--EXCURSION FROM SATURATION

Subject WS had a definite episode of spinal cord DCS, presumably affecting at least the left spinothalamic tracts and possibly the right pyramidal tract. In the small

number of previous cases, reviewed above, only one other incident like this had been observed in excursion diving. Despite a delay of 52 minutes before recompression, this diver was completely relieved of symptoms after 19 minutes of treatment.

In subject LJ the onset of vestibular DCS was dramatically confirmed by ENG, and so was relief of the condition. Bone conduction audiometry at 1050 fsw showed no residual loss, and there were no inner ear symptoms during the subsequent ascent to the surface. A temporary, bilateral decrease in auditory acuity was later observed at the surface but is unexplained. The transient dizziness and nausea reported at 1200 fsw before the decompression began were not accompanied by nystagmus and could not, therefore, be attributed to inner ear barotrauma. These symptoms may have been associated with the rapid compression from 800 fsw to 1200 fsw.

For subject MP the interval of 59 minutes between his return to the saturation depth and the onset of symptoms was unexpectedly long. His attempts at ear clearing after decompression required a consideration of alternative etiologies for his vertigo, such as forceful autoinflation and middle ear barotrauma of ascent (alternobaric vertigo) (10). Nystagmus was not observed since he had complete relief before the ENG electrodes were re-applied. However, the severity, time course and response to recompression all tend to support the diagnosis of DCS. Indeed this subject may have felt the need to clear his ear because of a transient hearing loss induced by the onset of DCS.

All three subjects were completely relieved of their symptoms by recompression to less than the depth of the excursion. Subject WS was symptom-free within 19 minutes at 950 fsw (ΔP = 150 fsw). Subject LJ had relief in 18 minutes at 1050 fsw (ΔP = 205 fsw). Subject MP was relieved in five minutes by ΔP of 200 fsw at 1400 fsw. These treatment results support the hypothesis that reversal of the inert gas gradient is the primary mechanism of therapy since the ΔP represented by these recompressions is of about the same magnitude as the hypothetical supersaturation at the time of the onset of DCS (Section G-1). The ratios of treatment depth to depth of onset were all less than 1.24, which is too small to affect bubble size substantially.

The helium-oxygen excursion experience reviewed previously comprised four inner ear cases, one spinal cord lesion, and one case of chest discomfort with pruritus. To these can now be added two more instances of inner ear symptoms and one incident of spinal cord involvement. Presumably, any syndrome is possible, but it can be predicted that inner ear lesions will be seen frequently in excursion DCS.

Clearly, complete relief can be obtained in deep excursion DCS, and this should be the endpoint sought in treatment. If rapid and progressive improvement is noted during recompression, then further recompression may be briefly halted to ascertain whether complete relief will follow. For inner ear disease, a recompression of not less than 100 fsw has been recommended (11, 13). While this may be a good general rule for cases of DCS after helium-oxygen excursions, if there is any doubt about residual damage, the patient should be recompressed to the depth of the excursion. This is especially important when inner ear lesions are present and hearing cannot be tested with precision, as in operational diving where accurate diagnosis and evaluation are not possible. Field personnel, therefore, should routinely recompress to the full depth of the excursion. If the incident occurs in a submersible compression chamber (bell), recompression should be initiated in the bell before transfer to the deck decompression chamber, since any delay in starting treatment reduces the chance of a complete cure.

Although the Predictive Studies IV excursion schedule did not allow more than one excursion per day, other techniques or circumstances do permit repetitive excursions in one day. If DCS follows one of these, the appropriate treatment depth should perhaps be the depth of the deepest excursion of the preceding 24-hour period.

With adequate environmental control, recompression at rates of 30 fsw/minute can probably be used for 400 fsw increments down to 1600 fsw without severe symptoms of HPNS. However, if chamber overheating occurs during compression, compression effects may be exaggerated. Since severe HPNS has not produced pathological sequelae in humans and DCS

has, the occurrence of HPNS during treatment should not force the acceptance of treatment failure. Of course, disregarding the effects of HPNS is only acceptable if the attention and cooperation of the divers inside the chamber were not required for safe completion of the treatment.

Hyperoxic treatment gas was used in the three cases cited here, but others have not found this necessary, either in the deep diving cases (2,5) or in at least one excursion instance (33). The administration of treatment gas should not be allowed to delay the primary therapy, recompression, and appropriate attention must be paid to oxygen toxicity when hyperoxia is used.

Recompression to excursion depth would be expected to achieve relief unless there were a great delay before initiating treatment. Recompression beyond the excursion depth should not be necessary, because in all cases the inert gas gradient will have been reversed at the excursion depth. In cases where relief is not achieved at excursion depth, the use of hyperoxic breathing mixtures and ancillary drugs is appropriate. Intravenous fluids and dextran would be indicated for CNS lesions not promptly resolved, for shock or for severe pain. Steroids might also be used in CNS cases. Diazepam is indicated for relief of residual nausea and vomiting from inner ear DCS but drugs which tend to inhibit coagulation, such as heparin or dextran, are not recommended in inner ear lesions because the lesion may be hemorrhagic (13,26).

The optimal subsequent decompression procedure for decompressing to the surface divers who have been treated for excursion DCS is not established. Two of the three cases described here had pain-only DCS during final ascents which began 39 hours (LJ, Phase I) and 43 hours (MP, Phase II) after treatment was initiated for excursion DCS. Saturation-decompression was free of DCS when it began 104.5 hours after initiation of therapy for excursion DCS. However, no firm guidance can now be given on the optimal rate of decompression, nor on the appropriate delay before commencing it.

SUMMARY

Decompression sickness occurring as a consequence of excursions from helium-oxygen saturation dives should be treated by prompt recompression at about 30 fsw/minute, to the depth of relief or to the depth of the deepest excursion of the preceding 24-hour period. In any case, the recompression should not be less than 100 fsw.

If maximal recompression is unsuccessful, hyperoxia and drugs should be used as appropriate.

Careful search for unresolved deficits should be made at treatment depth to determine whether further compression is needed.

Symptoms of HPNS during recompression should not be allowed to interfere with prompt recompression to treatment depth.

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H. SUMMARY AND CONCLUSIONS

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The findings of the composite investigations of Predictive Studies IV are cited in summary below, with intent to provide interpretation of observations relating to either competence or failure of the subjects in their activities. In addition to the Summary of observations, it is necessary in an integrated study such as this to offer judgments and conclusions which bridge the diverse lines of experimental search. Together with impressions derived from important related investigations on man and animals in other laboratories in several countries, these conclusions or interpretations can be used to influence further investigation, as well as to influence practical use of the findings described.

SUMMARY

This summary of both positive and negative findings pertains primarily to the higher pressure investigations and more stressful circumstances of Phase II.

SYMPTOMATIC AND OVERT EFFECTS

On slow, multi-day compression to stable helium pressures of 37.5 ata, as in Predictive Studies III, no limiting physiological or performance derangements are detectable.

First-day rapid excursion to 1200 fsw in this study produced the prominent symptoms that had been sought for investigation. These were temporarily severe on arrival at 1200 fsw, with partial recovery over a period of one to two hours during continued exposure to 1200 fsw on the first day. Adaptation was progressive, and effects still present in some subjects on the second morning at 1200 fsw were of minor significance and did not limit technical function.

Second-day excursion to 1600 fsw produced minor symptoms and signs of a pressure syndrome, but far less prominent than on the initial day of compression to 1200 fsw. Adaptation was sufficient overnight to allow effective technical and physical performance on arrival at the 1600-fsw pressure. This represented useful function in compression to the 1600-fsw pressure in approximately five days less than in previously reported exposures.

Successive daily excursions from 1200 to 1600 fsw showed progressively less deleterious effect, indicating continued adaptation to at least some effects of compression, with capability for vigorous and precise mental, manual and physical activity.

Compression from 1200 to 1600 fsw for working dives after five days of dry chamber studies between 1200 and 1600 fsw did elicit a just visible, nonlimiting tremor and some tenseness, indicating that some nonlimiting aspects of adaptation might still be incomplete at that time. Effects generated by excursions to 1600 fsw disappeared on return to 1200 fsw.

For the intricate, precisely timed and skillful technical work as subjects, no evident obstacles to working performance, even at 1600 fsw, existed following the initial day of compression to 1200 fsw.

VESTIBULAR FUNCTION AND BALANCE

Rapid compression from 1 ata to the pressures equivalent to 1200 fsw elicited symptoms typical of vestibular dysfunction, such as dizziness, nausea, vomiting and altered states of alertness. These symptoms were usually accompanied by increased body sway, and by subjective evidence of vestibular hypersensitivity during electrical stimulation of the vestibule, but not by nystagmus. Eye muscle coordination was not affected and there was no ocular dysmetria. The symptoms, subjective feelings of vestibular hypersensitivity, and alterations in postural control improved within one to two hours at 1200 fsw,

were barely detectable in any subject by the morning of the second exposure day, and were absent after day 3.

AUDITORY FUNCTION

No otological signs, hearing impairment or changes in audiometric spectrum were found in any of the six subjects during or after compression.

VISUAL FUNCTION

Relatively small, transient and functionally insignificant changes in visual accommodation and acuity were observed. Most, but not all, of these changes coincided with the severity of systemic discomfort reported by the subjects.

No other visual or ocular subjective or objective disturbances were observed in compression or prolonged residence at stable high pressure. Color vision and peripheral visual fields were normal at the stable pressures of 800 and 1200 fsw. Complex tasks involving vision and eye-hand coordination were performed throughout without interference due to visual difficulties.

SPEECH GENERATION AND DISTORTION

Rapid compressions and acute exposures to pressure equivalents of 800, 1200 and 1600 fsw in a helium-oxygen breathing mixture did not interfere with neuromuscular or other functions in speech formulation or articulation in the four subjects studied. The intelligibility of unprocessed speech recorded in helium-oxygen atmospheres from 400 to 1600 fsw by wide-bandwidth condenser microphones was not substantially affected by pressure (gas density).

PERCEPTUAL, MEMORY, COGNITIVE AND PERFORMANCE FUNCTIONS

Individual subjects varied markedly in susceptibility to compression and pressure. A feeling of tenseness or "nervousness," transient and not limiting, occurred in all

subjects during early compressions and in some during subsequent compressions. Some mental slowness, slowness of response, prolonged reaction time, increase in error incidence, and occasional brief episodes of failure to follow specific test procedures occurred during the initial compressions to 1200 fsw. These were largely associated with the period of prominent subject discomfort accompanied by nausea and vomiting. During all periods at stable pressure following the first day the subjects appeared close to full mental capacity, alertness and manual dexterity in their technical functions, but some showed detectable mental or performance test effects with subsequent compressions.

SLEEP

No influences on sleep or electroencephalographic activity in sleep were evident under stable saturation-compression at 1200 fsw.

ELECTROENCEPHALOGRAPHIC CHANGES

Changes were observed in the electroencephalogram, generally progressive with depth as pressure was increased to levels beyond 640-800 fsw. These changes, not consistently evident from day to day, included some disorganization of background activity and apparent lowering of frequency of components of background activity; occurrence of infrequent, intermittent, irregular forms low in frequency; and infrequent lower-frequency activity which was more overtly paroxysmal. A single burst of spike wave activity was recorded in one diver over the several weeks of exposure.

There was no apparent direct correlation of most of the electroencephalographic observations with either clinical phenomena or performance.

The conduction latency of the secondary N2 component of the somatosensory evoked cortical response was the only measured index of peripheral and central nerve conduction time that changed significantly. This change was found

only during the first few hours after the initial compression to 1200 fsw and could have been caused by nonspecific effects.

Visual evoked cortical responses showed small but consistent decreases in amplitude and increases in latency with increasing absolute pressure. The changes could not be systematically correlated with compression rate.

TREMOR

Integrated amplitudes of intentional and postural tremor doubled during rapid compression to 800 and 1200 fsw, then partially or completely reversed within the first two to four hours of continued exposure to a constant increased pressure. However, this compression-induced twofold increment in tremor amplitude was essentially invisible and caused no detectable functional impairment. Despite early apparent adaptation, amplitudes of both forms of tremor were again approximately doubled during 400-fsw excursions on exposure days 2 and 3. There were no specifically related deficits in testing sequences or in the performance of coordinated fine and coarse motor activity of complex work tasks.

The neuromuscular, circulatory and metabolic responses to light exercise had no consistent influence on the amplitudes of intentional and postural tremor.

CARDIAC ELECTRICAL AND MECHANICAL FUNCTION

Rapid compressions and sustained exposures to increased pressure in helium-oxygen did not alter the electrocardiogram or affect cardiac conduction or ejection times. Heart rates were quantitatively appropriate for physical activity levels while subjects were seated and engaged in light activities, and during light exercise on a bicycle ergometer. Cardiac outputs estimated indirectly by an impedance method were also appropriate to activity. The expected heart rate response to postural change was present, with a few exceptions during the more severe symptoms of initial compression exposures.

There was no evidence for a pressure-related bradycardia in the subjects of these studies.

It is considered that neither compression nor hydrostatic effects in combination with high helium concentrations affected cardiovascular functions.

ACUTE HYDROSTATIC EFFECTS ON PULMONARY MECHANICAL FUNCTION

Rapid compressions to 1200 fsw caused transient decrements in pulmonary ventilatory performance, in addition to those expected to be related to gas density alone. They were most marked when symptoms and manifestations of compression and high pressure were prominent, and they receded with time as the symptoms diminished. These transient decrements were apparently partly effort-dependent.

Sensations of respiratory distress were reported by only one subject, during exercise when his work level increased above that planned for the study.

Ventilatory levels in normoxic helium-oxygen were adequate to support the selected exercise of 450 kpm/min immediately following rapid compressions in helium to the equivalent of 800-1200-1600 fsw. Pulmonary function measurements after several days at 1200 fsw and during decompression were equivalent to those measured under stable conditions at the same pressure in the earlier Predictive Studies III.

VENTILATION AT REST DURING COMPRESSION AND AT STABLE HIGH PRESSURES

Neither alveolar hypercapnia nor hypocapnia nor changes in resting metabolism resulted from the exposures. There were probably no prominent CO_2-related alterations in the acid-base state to interact with compression-pressure effects on excitable tissues.

VENTILATORY AND METABOLIC RESPONSES TO EXERCISE

Ventilatory and metabolic responses to the light exercise during rapid compression generally resembled responses

to comparable workloads at normal ambient pressure. Exercise subjects showed reductions, presumably density-related, in respiratory minute volume and frequency, with an increment in tidal volume, compared with responses at 1 ata for the same level of exercise.

Two subjects had no apparent symptoms of respiratory distress during exercise at increased pressures. A third subject, who did have sensations of dyspnea and "increased resistance" during ergometer exercise at depth, had ventilatory responses to workload that differed little from those at sea level.

The exercise appeared to induce no consistent changes in tremor, brain electrical activity or mental performance.

BREATHHOLDING CAPACITY

Breathholding duration was found to be as long in stable exposures at high ambient pressures to the equivalent of 1200 fsw as at one atmosphere.

THERMAL HOMEOSTASIS

Elevation of ambient temperatures in the high-pressure He-O_2 environment reduced body heat loss to levels consistent with subjective comfort and protection of deep body temperature sensors. However, detectably elevated resting heart rate and cardiac output observed in most of the subjects indicated the presence of a mild cardiovascular stimulus. Even small, transient deviations of ambient gas temperature of two degrees from a comfort zone caused either shivering or complaints of excessive heat at the 1600-fsw pressure equivalent.

Body weight decreased slightly and progressively during the exposures to high pressure He-O_2 despite reasonable caloric intake and undetectable change in whole body oxygen consumption.

DECOMPRESSION FROM EXCURSION EXPOSURES

The decompression procedures devised for the deep saturation-excursion pressurizations were based on U.S. Navy and British Royal Naval investigations showing that, for "no-decompression" ascents from a saturated state, the magnitude of safe initial ascent becomes greater with the helium saturation depth from which ascent occurs. The excursion-decompression procedures devised considered both degree and duration of excess tissue gas pressure. They required one-tenth of the decompression time needed to return to atmospheric pressure at sea level after a 60-minute dive from 1 ata to 400 fsw. While decompression sickness did occur, the results demonstrate that, as in the case of no-decompression excursions from deep saturation, decompression requirements for longer excursions are reduced at increased saturation pressures, and that data from no-decompression exposures can be used in deriving such longer decompressions.

UNDERWATER WORK PERFORMANCE

Skilled and vigorous work was carried out underwater at the 1210- 1360- and 1610-fsw depth equivalents using low-resistance open-circuit breathing equipment.

Training for the underwater work was carried out under 10 to 12 feet of water, pre-compression, where measurements of associated oxygen consumption and ventilation were performed. Work oxygen consumption averaged 2.0 liters per minute, and pulmonary ventilation, approximately 60 liters per minute. The timed and programmed sequence involved dismantling major valve flange and oil wellhead components in the water-filled chamber compartment, with timed monitoring of performance.

Underwater work before compression and at high pressures was performed at self-competitive rates, with timing and performance skill found to be equivalent to those at the surface.

Since man had not previously performed practical work in water at depths greater than 636 feet, this underwater

work represented an approximately 1000-foot extension beyond any earlier practical working experience in water either in laboratory chambers or at sea.

CONCLUSIONS AND INTERPRETATIONS

On relating findings in this study to observations by others, interpretations can be made concerning compression effects. These conclusions, some of which have been derived previously in other laboratories, include:

The severity of disruptions elicited by initial compression is related both to the degree (or absolute pressure) of exposure and to the rate of compression.

Effects of extreme rate or degree of compression probably are generated not only in the central nervous system or other neural tissue, but in chemical and biophysical systems of other structures as well.

In even extremely rapid compression to moderate pressures of less than about 10 to 20 atmospheres, compression effects on neural or other functions are usually not evident. If alterations occur they are small in degree and tend not to persist as subjectively detectable effects. It can nevertheless be presumed, but is not known, that some cellular or other influences of compression may begin at pressures and compression rates which do not induce recognizable symptomatic effects.

There is no definite indication that helium gas contributes to the development of compression effects at pressures thus far experienced by man.

It is probable that the specific disruptive consequences of compression continue to exist during masking of effects by narcotic drugs or gases.

Both the degree of adaptation and rate of adaptation to induced compression effects are probably influenced by the rate of compression and the absolute pressure of initial exposure.

The complex physiological and symptomatic expressions of compression effect can be expected to vary with different individuals and at different times.

The effects of compression are real: they may be exaggerated in some individuals by influences of excitement, urgency, nervousness, lack of training or lack of personal motivation.

Prominent general adaptation occurs to symptomatically tolerable compression effects.

Transient excursion to higher pressures can be expected to bring out latent compression effects not evident at a stable saturation pressure.

Transient excursion to a higher pressure from a saturation state also probably increases the rate of adaptation to the stable elevated pressure.

Rates of adaptation of various functions can be expected to differ, with some effects being mitigated almost immediately and others requiring many days. It is conceivable that at extremely high pressures some of the functional disruptions produced will not fully adapt regardless of length of exposure.

From current experience, compression to 800 fsw in an hour allows further transient excursion compression to 1200 fsw on the same day, with relief of any symptoms of the excursion exposure on return to 800 fsw.

Programmed compression to 1200 fsw in one day allows sufficient adaptation for compression to 1600 fsw on the following day, with effective technical and physical performance.

Compression of man to helium pressures equivalent to 1600 and 2000 fsw has induced no evident residual effects following decompression from these pressurizations. While controlled exposure of animals to still higher helium pressures equivalent to 4000 fsw has induced more severe compression effects, exposures

less than required to induce convulsions have not led to persistent effects on decompression.

Man, rapidly enough compressed, to high enough pressure, must develop incapacitating physiological and symptomatic effects. It must also be presumed that, as pressure of exposure increases beyond that thus far investigated, compression effects will lead to the convulsions found in compression of smaller mammals.

It can be assumed that if convulsive reaction is generated in man by rapid or slow compression to extreme pressure, the pressure circumstances generating the condition will not be readily reversible by decompression, and the induced derangements will therefore not be relievable. These should be therefore presumed inevitably destructive and lethal for man.

APPENDIX

Documentation Storage

Separate documentation of the Predictive Study, maintained in Institute Data Storage for further analysis and correlation, is as follows:

Decompression-Gas and Environmental Parameters Log

Electroencephalographic and Cortical Evoked Potential Tapes and Recordings

Performance Measurement Recording Tapes

Pulmonary Function Recording Tapes

Speech Recording Tapes

Audio Log

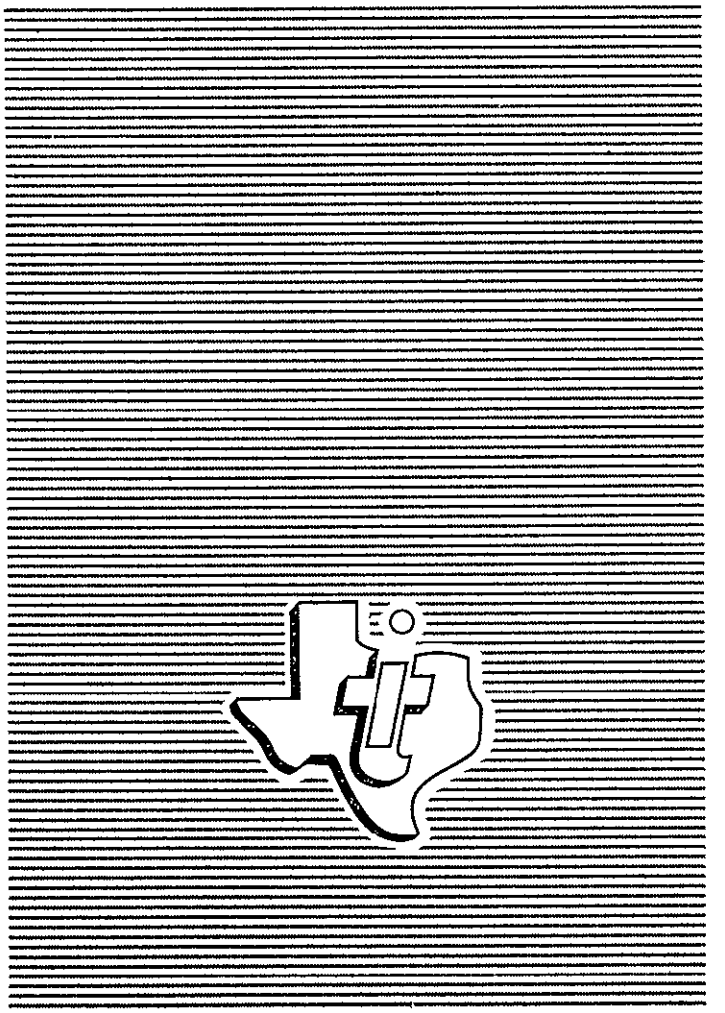
Symptoms Log

Motion Picture Film and Video Tape Log

FINAL REPORT ADDENDUM FOR THE DESIGN AND DEMONSTRATION OF AN ADVANCED DATA COLLECTION/POSITION LOCATING SYSTEM

Prepared for

NASA-GODDARD
SPACE FLIGHT CENTER



Prepared by

Equipment Group
01-879210-FA
Contract NAS 5-23599

TEXAS INSTRUMENTS
INCORPORATED

May 1978

FOREWORD

The work described in this report was performed by the Digital and Space Systems Department, Equipment Group, Texas Instruments Incorporated, Dallas, Texas, under NASA Contract NAS 5-23599 for Goddard Space Flight Center. The reporting period is from October 1977 to April 1978. The technical director of this investigative program was J. Leland Langston. He was assisted by John F. DuBose Jr., James L. Coates, and Dennis Young of the Digital and Space Systems Department. Acknowledgment and appreciation are also due Earle Painter of GSFC for his constructive criticism and suggestions throughout the course of the program.

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May 1978

FINAL REPORT ADDENDUM FOR THE DESIGN AND DEMONSTRATION OF AN ADVANCED DATA COLLECTION/POSITION LOCATING SYSTEM

01-879210-FA

SECTION 1

INTRODUCTION AND SUMMARY

1.1 INTRODUCTION

This is an addendum to the Final Report entitled "Design and Demonstration of an Advanced Data Collection/Position Locating System," No. 01-879210-FA. This addendum covers the design, construction, and tests of a coherent demodulator for minimum shift keying (MSK) signals generated by the Advanced Data Collection/Position Locating System breadboard. Section II is the theoretical background for the coherent demodulator. Section III is a detailed description of the design of the MSK coherent demodulator. Section IV discusses various tests and test results obtained with the breadboard system. In particular, the tests included evaluation of bit-error rate (BER) performance, acquisition time, clock recovery, recycle time, frequency measurement accuracy, and mutual interference.

1.2 BREADBOARD DESCRIPTION

The coherent demodulator was constructed on a single card measuring approximately 5 by 10 inches. It was designed to be interchangeable with the noncoherent demodulator constructed earlier. A photograph of the MSK coherent demodulator is shown in Figure 1-1. The right half of the board contains the mark/space phaselock loops. The VCXOs are constructed on separate boards and attached to the main board.

A photograph of the equipment built for test and evaluation of the Advanced Data Collection/Position Locating System is shown in Figure 1-2.

Figure 1-3 is a photograph of the receiver chassis. The first board at the extreme left on the chassis is the MSK coherent demodulator. The next board contains the receiver IF circuitry. The next two boards contain the clock generation circuitry. The three boards on the right side of the chassis contain the chirp-z transform search unit; the two large CCD chips are seen near the lower edge of the right-most card.

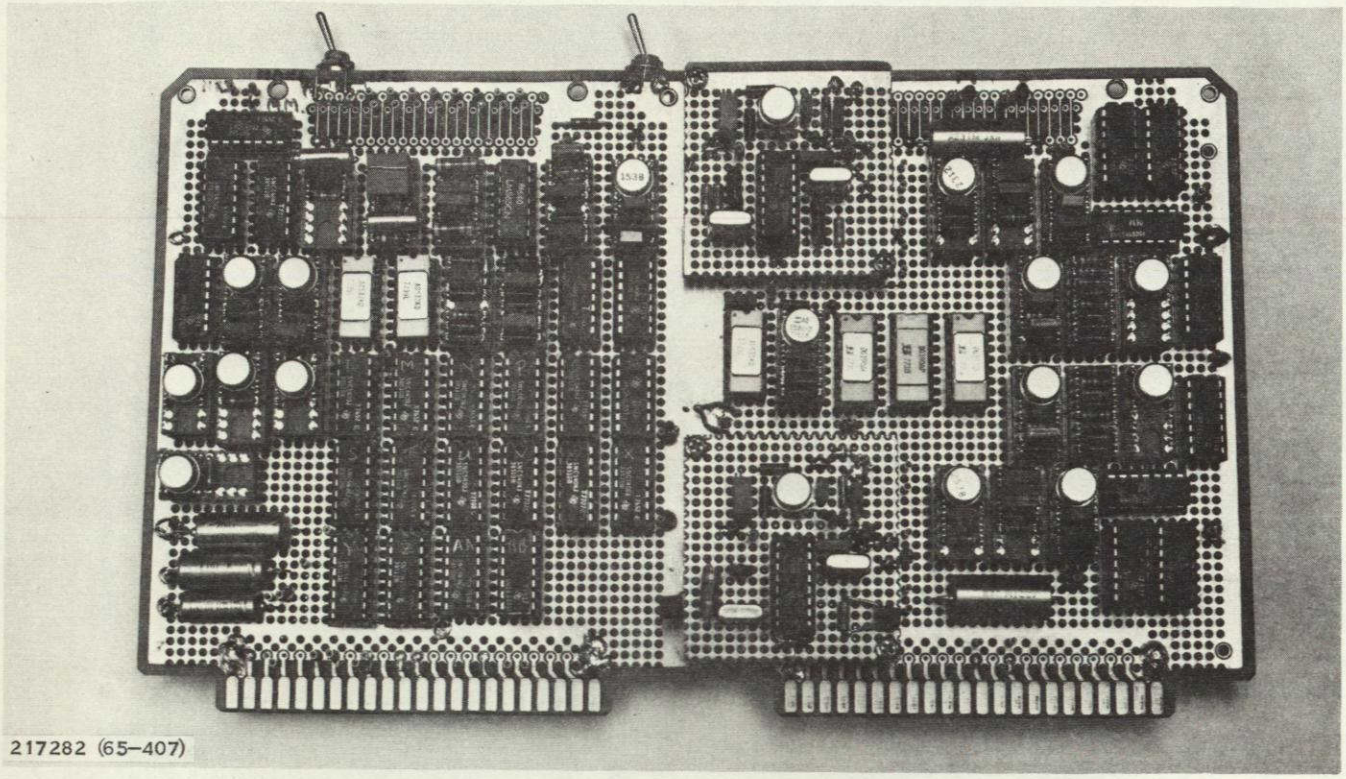

Figure 1-1. Coherent Demodulator for MSK

The system configuration remained the same as before except for the addition of an AGC circuit and a crystal filter to the receiver card. A few changes were made to other sections so that Miller data and differentially encoded Manchester data can be generated. In addition, provisions were made to use the noncoherent modulator as a source of interference for mutual interference tests. Otherwise, the equipment is as described in Subsection 1.B of the main report.

1.3 SUMMARY OF TEST RESULTS

The ability of MSK to increase system dynamic range (and thus allow increased platform density) by reducing crosstalk and adjacent-channel interference has been well established earlier. This phase of the study attempted to address the feasibility of coherently demodulating the MSK signal, thus improving BER performance. A closely related goal was evaluating various data-encoding schemes for low-data-rate systems.

BER tests were conducted using the digital MSK modulator and the coherent demodulator, for non-return to zero (NRZ), bi-phase, and Miller encoded data. The information rate in each case was 320 BPS. The results of these tests are shown in Figures 4-2 and 4-3. The results of these tests show close agreement between measured and theoretical performance (within 1.5 dB) for Miller and bi-phase data. Substantial variance exists for NRZ data, however. The degraded performance of NRZ data is caused mainly by failure of the mark/space phaselock loops to maintain the proper phase of the carriers at the low data rates. This problem is avoided with Miller or bi-phase data as a result of the more frequent transitions between the mark and space

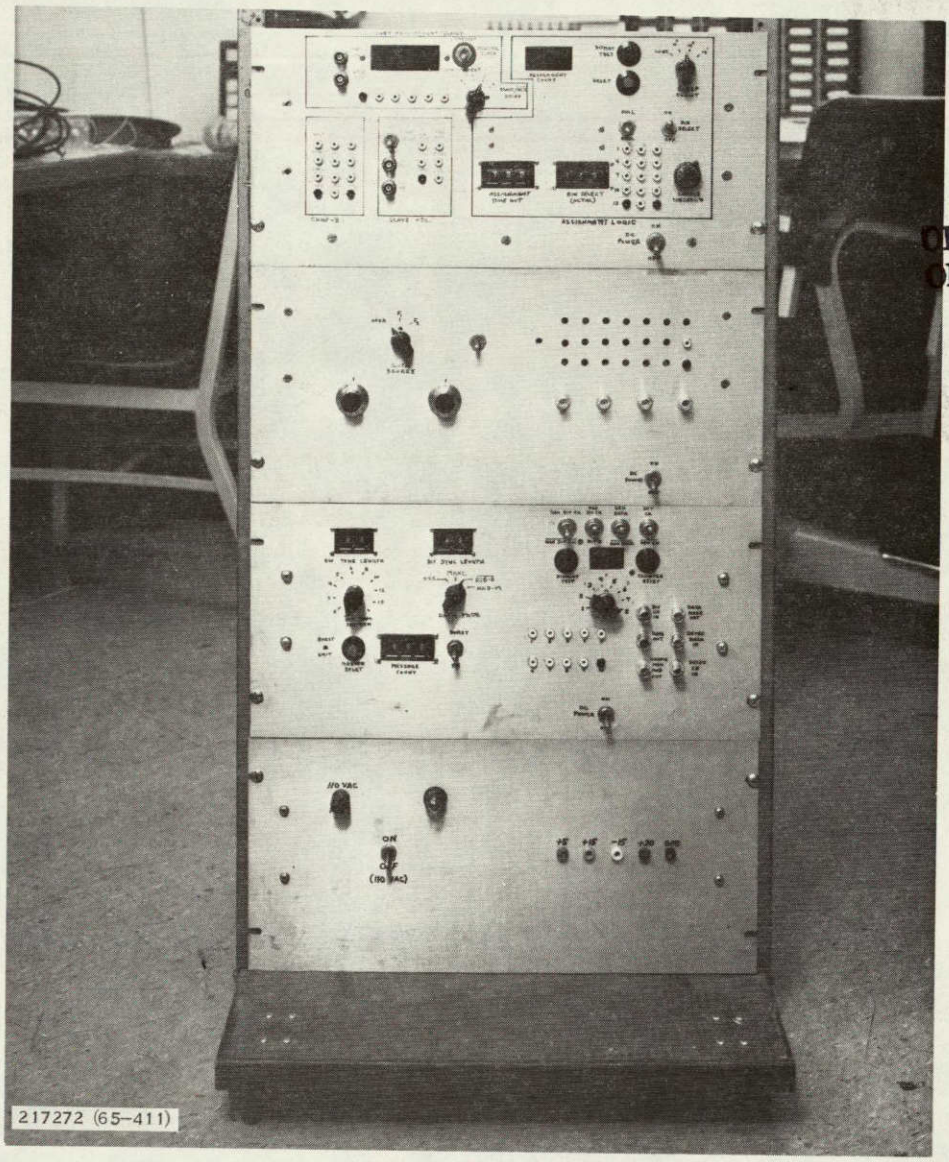

Figure 1-2. Breadboard Equipment for the Design and Evaluation of the ADC/Position Locating System

frequencies. Miller encoded data appears to have a 0.5 to 1.0 dB advantage over bi-phase encoded data.

The power spectral densities (PSD) of NRZ, bi-phase, and Miller data are compared in Figure 4-6. The measured PSD of NRZ/MSK data is similar to the calculated PSD. The PSD of Manchester/MSK data is similar to expected characteristics except for the existence of discrete tones. The PSD for Miller/MSK data is similar to that of NRZ/MSK for a data rate of 2R. The nonexistence of spectral lines in the PSD of Miller/MSK data probably accounts for the improved performance of Miller encoded data with respect to bi-phase encoded data. The lack of discrete frequencies in the sidelobes of Miller/MSK data would also indicate some reduction in adjacent-channel interference compared to that expected for Manchester/MSK data. Therefore, the use of

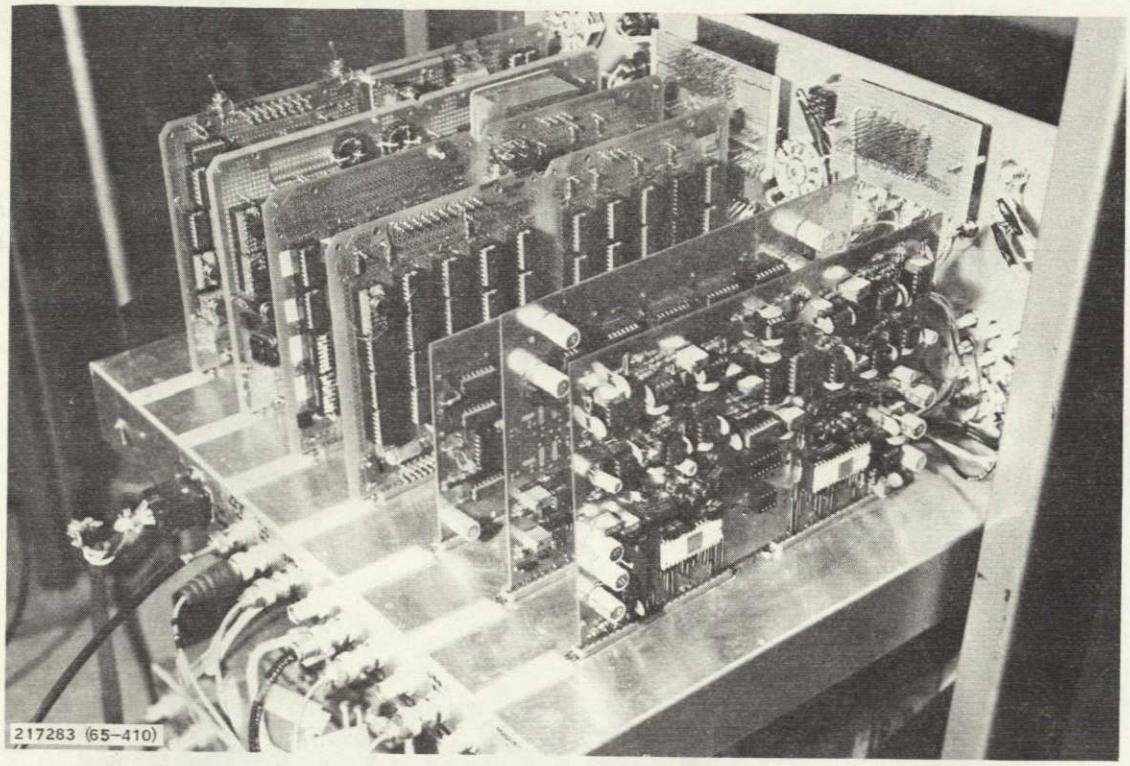

Figure 1-3. Shelf Containing Boards That Make Up the Receiver

Miller/MSK data encoding and modulation appears to offer the best performance in a low-data-rate data-collection system.

In this phase of the study, another key performance factor, a noncoherent MSK modulator with a coherent demodulator, was evaluated. A VCXO was used as the noncoherent modulator, and the modulation index of this modulator was adjusted for β 0.25. The VCXO operated at a frequency of 10.7 MHz and was in no way related in phase to the system clock. The data were applied directly to this modulator. The coherent demodulator was used to reconstruct the data and a set of BER performance data was obtained for this combination. The results showed *no measurable difference* in performance of this modulator and that of the digital modulator. This is an important data point in determining the feasibility of a low-cost data-collection system using MSK.

The frequency of the mark VCXO was used to measure the frequency of the received signal. The VCXO frequency was 100 times the intermediate frequency of the received mark frequency. The frequency measurement was made in a 100-ms time interval, beginning at various points in the message. The results of these tests show that a measurement accuracy of ±0.5 Hz is possible, following a 200-ms delay after the beginning of a message for Manchester or Miller encoded data. However, it is not possible to maintain this accuracy when NRZ data is used; errors up to 3 Hz were encountered with NRZ. Once again, the infeasibility of NRZ in a low-data-rate data-collection system is proved.

The test results show that a loop bandwidth of 16 Hz may be too narrow to provide good acquisition time with a 100-ms bit-sync code duration. Hence, it is suggested that the loops be designed to have an acquisition mode with a bandwidth of 100–200 Hz and a tracking mode with a bandwidth of 15–20 Hz. In addition, the overall performance can be improved by transmitting the mark frequency as the unmodulated preamble, thus allowing the mark PLL more time to acquire; this would improve the frequency measurement accuracy.

For the narrow-bandwidth IF filter, it is necessary to center the received signal within the passband of the filter. This requires a slight change in the mark PLL circuitry. This scheme requires mixing of the output from the mark VCXO with the other injection signals. The result is mixed with the incoming signal to produce the channel IF. The phase detector for the mark PLL is supplied with a stable reference at twice the mark frequency. The VCXO output frequency is thus controlled to center the received signal in the passband. This scheme requires a VCXO with a tuning range of at least ±0.1 percent. It also requires an acquisition loop bandwidth of 200 Hz if the search unit has a resolution of ±100 Hz. A 100-ms unmodulated preamble at the mark frequency must be used, requiring an offset in the digital oscillator frequency equal to 1/2 f_{ck}. However, this scheme will improve the overall acquisition performof the receiver.

To take advantage of the close-channel spacing obtainable with MSK, the search unit must provide good resolution. The 512-point CZT discussed in the main report can provide a resolution of 160 Hz over an analyzing bandwidth of 40 kHz. However, the device alone cannot adequately resolve all received frequencies and assign receive channels. Some logic is needed to distinguish between adjacent bin registrations to select the proper frequency. In addition, some decision logic is needed to prevent multiple assignments for the same signal and/or to select the strongest signal if there is mutual interference. Furthermore, it is desirable to provide some signal averaging to minimize the effects of noise. A microprocessor would be a major asset in accomplishing these tasks.

The increased dynamic range provided by MSK requires tight control over the receiver gain because of the threshold requirements of the search unit and the need for a fixed noise level at the baseband output of the receiver. This requires either very stable receiver gain or a means of automatically stabalizing that gain. The former is rather difficult to achieve on a long-term basis. Hence, some form of AGC is anticipated. One method would be to inject a stable-amplitude, stable-frequency tone into the receiver front-end. The output produced by this tone on one channel of the CZT could be used to set the receiver gain. The threshold could then be set relative to this reference.

The use of an AGC circuit for each receiver channel is desirable, although tests show that only a 1-dB loss in E_b/N_o is encountered if a limiter is used to maintain constant output from the receiver. The channel AGC is relatively easy to implement and ensures linear operation of all amplifiers and mixers. The only disadvantage to using channel AGC is that it requires some time (approximately 400 ms) to stabilize the gain. Furthermore, a limiter seemed to offer improved performance in the burst mode.

The above information can be formulated into a "strawman" design for a general-purpose data-collection system that would operate with either a low-orbiting satellite or a geostationary satellite. Such a system could service up to 1000 platforms within the field of view in the case of the low-orbiting satellite or up to several thousand, in the case of a geostationary satellite.

TABLE 1-1. PLATFORM 401-MHz TRANSMITTER CHARACTERISTICS

Item	Characteristics
Oscillator stability	
Long-term (uniform distribution)	±0.003 percent
Medium-term (drift rate)	1 Hz/minute maximum at 401 MHz
Short-term (fractional frequency deviation)	2×10^{-9} maximum
Power output	5 W, ERP
Spurious output	50 dB below 5 W
Antenna gain	3 dB
Polarization	Right-hand circular
VSWR tolerance	Infinite without damage
Modulation	MSK
Data encoding	Miller (from bi-phase data)
Error correcting code	Triple-error-correction Bose-Chaudhuri code
Message format	
CW preamble	102 ms
Bit sync code (all ones)	15 bits
Frame sync code	24 bits
User code	8 bits
ID code	24 bits
DATA*	64 bits
Parity code	21 bits
Transmission bit rate	128 BPS
Transmission time	1.32 seconds
Period between transmissions	48 seconds

*Multiplexing can provide up to 256 bits in four transmissions

The recommended design goals for a general-purpose data-collection platform transmitter are given in Table 1-1. It may be desirable to provide an optional power selection to provide for operation at either 1 W or 5 W power output. A small microprocessor can be used to collect, process, and format the data for transmission. If only a geostationary satellite is to be used, the period between transmissions can be extended; hence, provisions should be made to select the repetition rate for periods over a range of 48 seconds to perhaps an hour. A functional block diagram of a general-purpose data-collection platform is shown in Figure 1-4.

Satellite equipment characteristics are listed in Table 1-2. This table does not include all the design parameters. However, it does assume adequate baseband filtering and the use of a chirp-z transform for the search unit. Although not absolutely required, a microprocessor is highly desired to handle the density expected in the system, particularly in the geostationary system. The IF should be selected to accommodate filtering with crystal filters. A survey of present filter capability shows that the channel center IF should be approximately 400 kHz for conventional crystal filters and perhaps 5 MHz for monolithic crystal filters. Exact frequencies must be chosen to conform to a master frequency plan. Key considerations are the clock frequency of the CZT and the digital oscillator. A block diagram of a candidate system is shown in Figure 1-5.

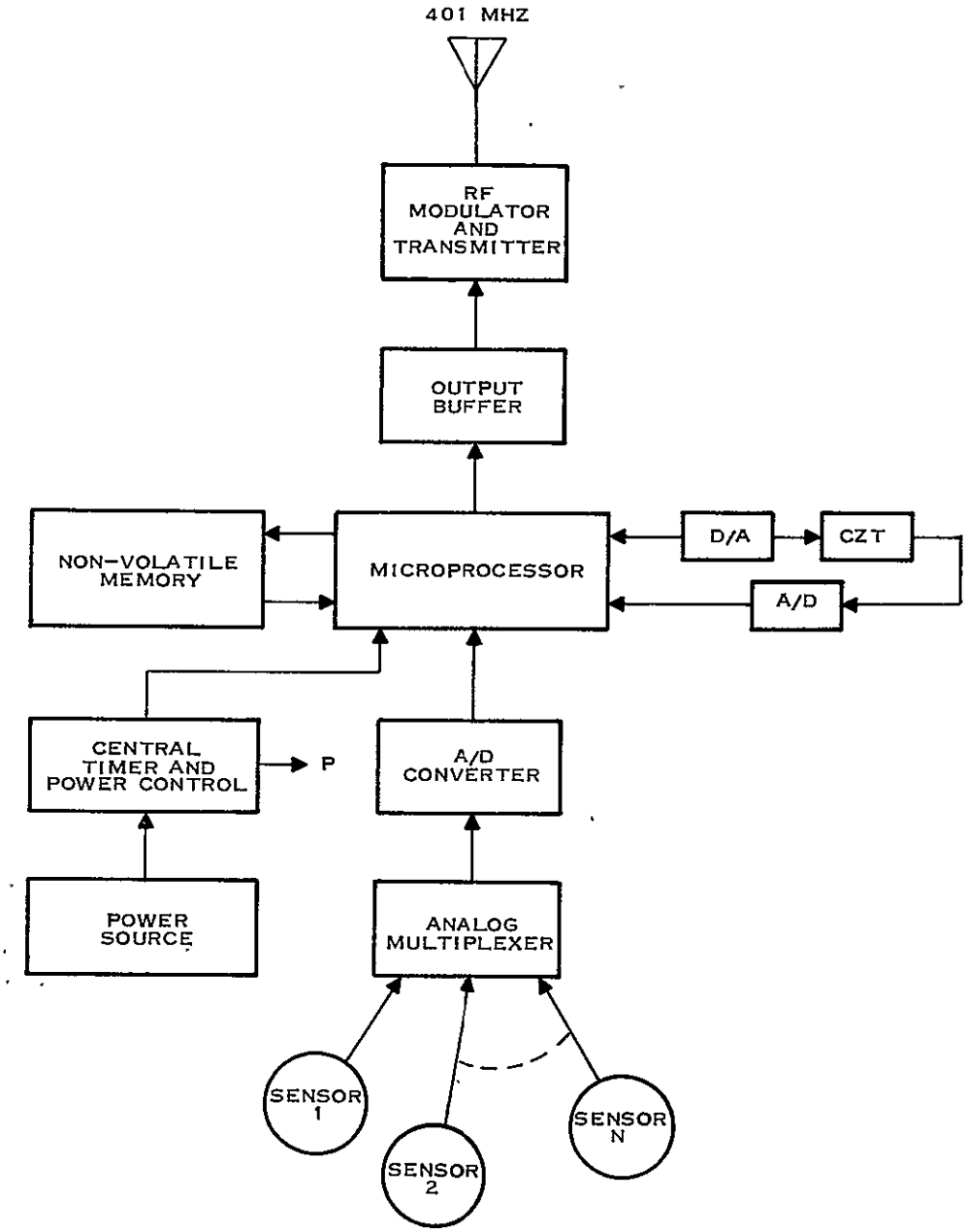

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Figure 1-4. General-Purpose Data Collection Platform

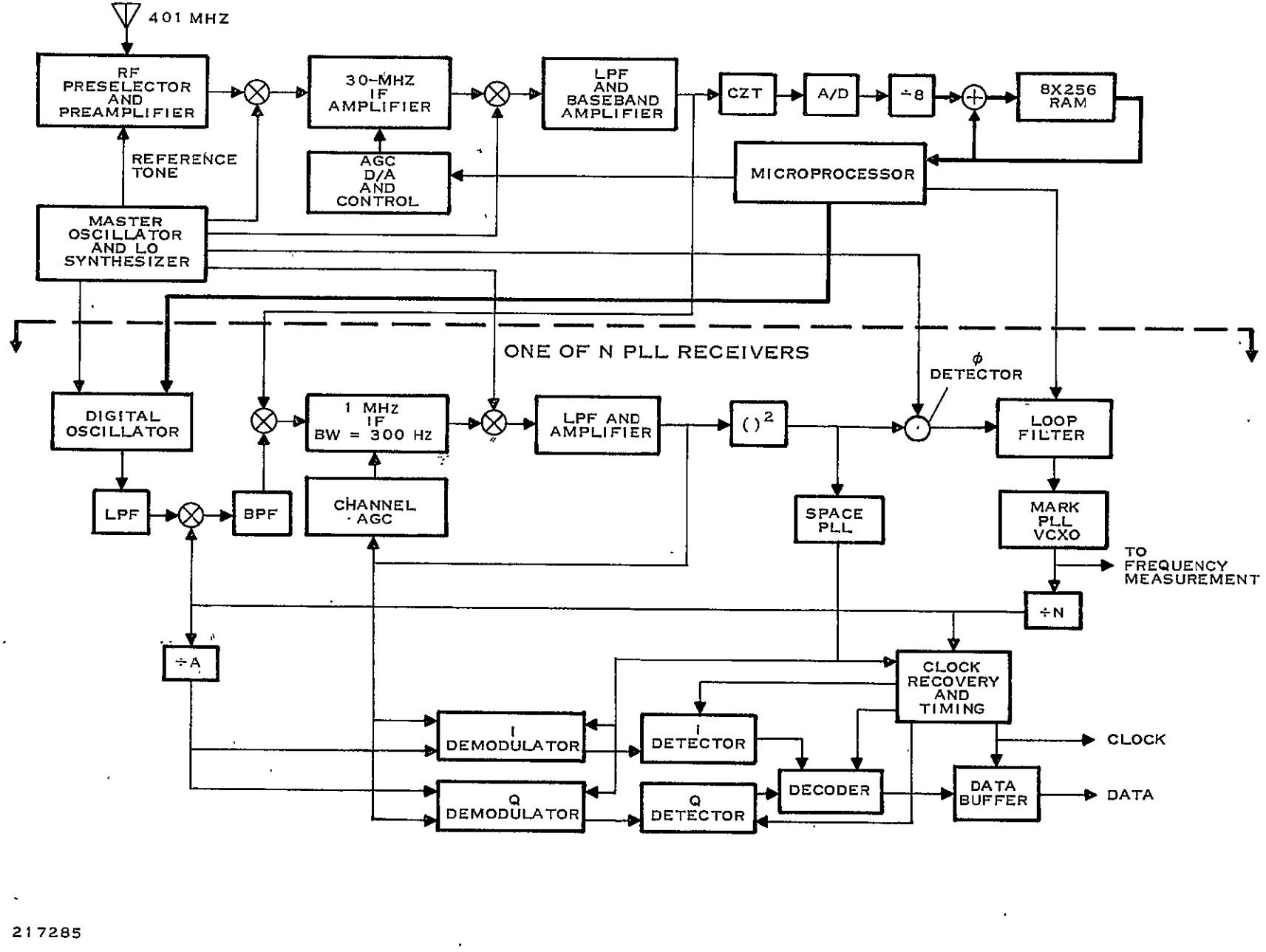


Figure 1-5. Satellite Receiver Block Diagram for ADC/PL System

TABLE 1-2. SATELLITE EQUIPMENT CHARACTERISTICS

Item	Characteristics
Low orbiter	
Antenna beam angle (3 dB)	120 degrees
Antenna gain	2.75 dB
Receiver noise figure	4 dB, maximum
Search bandwidth	40 kHz
Search/assignment time	25 ms, maximum
Receiver dynamic range	30 dB, minimum, at 4R
Number of channel demodulators	12
Channel bandwidth	300 Hz
Frequency measurement accuracy	±0.5 Hz
Suboptimum data detection allowance	3 dB E/n_o
Output data buffer size	1,024 bits
Output data rate	1,680 through 2,048 BPS
Geostationary, same as low orbiter, except:	
Antenna beam anble (3 dB)	20 degrees
Antenna gain	18 dB
Receiver noise figure	3 dB
Number of channel demodulators	16 through 20

SECTION 2
THEORETICAL CONSIDERATIONS

This section provides the theoretical basis for the design of the minimum shift keying (MSK) system described in Section 3. It also summarizes the results of major relevant papers on MSK and presents graphic comparisons of MSK with other popular data-modulation techniques. Although the emphasis is on applications to low-data-rate satellite data-collection systems, the results are generally applicable to other communication systems.

2.1 DEFINITION OF MSK

Minimum shift keying (MSK) is a member of the large category of modulation methods known as *exponential* modulation.[1] Exponential modulation is defined by the mathematical operation of multiplying a carrier function

$$e_c(t) = A_c \epsilon^{j(\omega_c t + \phi_c)} \tag{2-1}$$

by a transformed message function of the form

$$e_m(t) = \epsilon^{jm_p g(t)} \tag{2-2}$$

to yield a constant-amplitude angle-modulated signal:

$$e(t) = A_c \epsilon^{j[\omega_c t + m_p g(t) + \phi]} \tag{2-3}$$

For the above equations:

A_c = the peak carrier amplitude

ω_c = the carrier frequency (rad/s)

ϕ_c = the initial carrier phase offset

m_p = the modulation constant or phase-modulation index

$g(t)$ = the original message function.

The peak amplitude, A_c, of the phase-modulated signal is constant. However, output phase is made to vary with respect to a reference phase, ϕ_c, by the modulating function, g(t). The modulation constant, m_p, essentially converts from the units of g(t) (usually volts) to radians. The phase function for the modulated signal can also be expressed by:

$$\theta(t)_p = \omega_c t + \phi_c \, [1 + k_p \, g(t)] \tag{2-4}$$

This expression clearly shows that it is the phase that is operated on in a phase-modulated (PM) system. A similar expression for frequency modulation is:

$$\omega(t) = \omega_o [1 + k_f \, g(t)] \tag{2-5}$$

[1] Philip F. Panter, *Modulation, Noise, and Spectral Analysis*, McGraw-Hill, Inc. (New York, 1965).

The usual definition of frequency is applicable:

$$\omega(t) = d\,\theta\,(t)/dt$$

Then the phase function, i.e., the exponent in Equation (2-3), is obtained by integrating Equation (2-5):

$$\begin{aligned}\theta(t)_f &= \int \omega_o\ [1 + k_f\ f(t)]\ dt \\ &= \omega_o\ t + k_f \int g(t)\ dt + \phi_c\end{aligned} \tag{2-6}$$

This equation can be recast in a form similar to Equation (2-4):

$$\theta(t)_f = \omega_o\ t + \phi_c\ [1 + k_f\ \omega_o/\phi_c \int g(t)\ dt] \tag{2-7}$$

A comparison of Equations (2-4) and (2-7) shows that PM results in a phase function directly proportional to the modulating function, g(t), while FM results in a phase function directly proportional to the *integral* of the message function. This difference distinguishes between PM and FM.

It is obvious that PM requires a phase reference for demodulation, but no such reference is necessary for FM demodulation (Equation 2-5). However, if a phase reference is available for FM demodulation, it can be demodulated as a PM signal, with the output representing the integral of the original message function. Such a technique is not practical in most applications since linear phase detectors over phase ranges exceeding ±180 degrees cannot be made; however, as will be shown soon, MSK can be processed as a pseudo-PSK signal.

2.1.1 MSK Viewed as a Special Case of Continuous-Phase Frequency Shift Keying

When the message function, g(t), represents a binary data signal that can assume only one of two states, +1 or −1, the phase modulation (PM) of a carrier is termed phase shift keying (PSK). When it is used to frequency modulate a carrier, it is termed frequency shift keying (FSK). The phase deviation can range from a few degrees to the maximum of ±90 degrees in the case of antipodal PSK (also called binary PSK or BPSK). The frequency deviation in FSK can have any practical value. For an analog message function, the frequency deviation in FM is changing continuously. For a sine-wave modulating signal, the peaks of the sinusoid cause a *peak* frequency deviation of $\pm\Delta\Omega$. The modulation index, β, is defined for a single modulating frequency by:

$$\beta \equiv \frac{\Delta\Omega}{\omega_m} \tag{2-8}$$

where ω_m is the modulation frequency. When the modulating signal is a square wave, the modulation index is similarly defined, except that ω_m is the frequency of the square wave (or, more precisely, the frequency of the fundamental of the square wave). It should be noted that β depends on both the amplitude and the frequency of the modulating signal. Hence, it can approach any value from zero to infinity. However, the unique value of β used to characterize a system is that value of β, for the maximum amplitude and maximum frequency signal to be modulated:

$$\beta_s = \frac{\Delta\Omega_{max}}{\omega_{max}}$$

It is this unique value of β that is used to distinguish between narrowband FM and wideband FM. In general, narrowband FM is characterized by $\beta_s \leqslant 1$ while wideband FM is characterized by $\beta_s > 1$.

When data are transmitted, the frequency deviation is fixed at $\pm\Delta\Omega$. Therefore, the modulation index for data can be defined in terms of the data clock:

$$\beta_{ck} \equiv \frac{\Delta\Omega}{\omega_{ck}} \tag{2-9}$$

The clock frequency is defined as the clock used for NRZ data or the equivalent clock rate that makes other data appear as NRZ data. The instantaneous frequency can be expressed in terms of this modulation index:

$$\omega(t) = \omega_o + \beta_{ck}\ \omega_{ck}\ g(t) \tag{2-10}$$

where g(t) takes only values of ± 1. For the remainder of this report, the subscript for β will not be used and, unless otherwise indicated, the modulation index will be that defined by Equation (2-9).

FSK can be generated by switching between two frequencies. The higher frequency is usually called the mark frequency while the lower one is called the space frequency. The mark frequency is selected when g(t) = +1; the space frequency is selected when g(t) = −1. From Equation (2-10):

$$\omega_m = \omega_o + \beta\omega_{ck} \tag{2-11a}$$

$$\omega_s = \omega_o - \beta\omega_{ck} \tag{2-11b}$$

The value of β is unrestricted and the phase of the mark and space frequencies at the time of a data transition can be any random value. Therefore, the general case of FSK allows the phase to be discontinuous (Figure 2-1). If the phase is discontinuous, this modulation produces spectral spreading similar to that of PSK.

The power spectral density functions for both continuous- and discontinuous-phase FSK have been derived by Bennett and Rice.[2] The result for discontinuous-phase FSK, valid for $\omega > 0$ and $(\omega_m, \omega_s) \gg \omega_{ck}$, is:

[2] W.R. Bennett and S.O. Rice, "Spectral Density and Autocorrelation Functions Associated with Binary Frequency-Shift Keying," *Bell System Technical Journal*, September 1963.

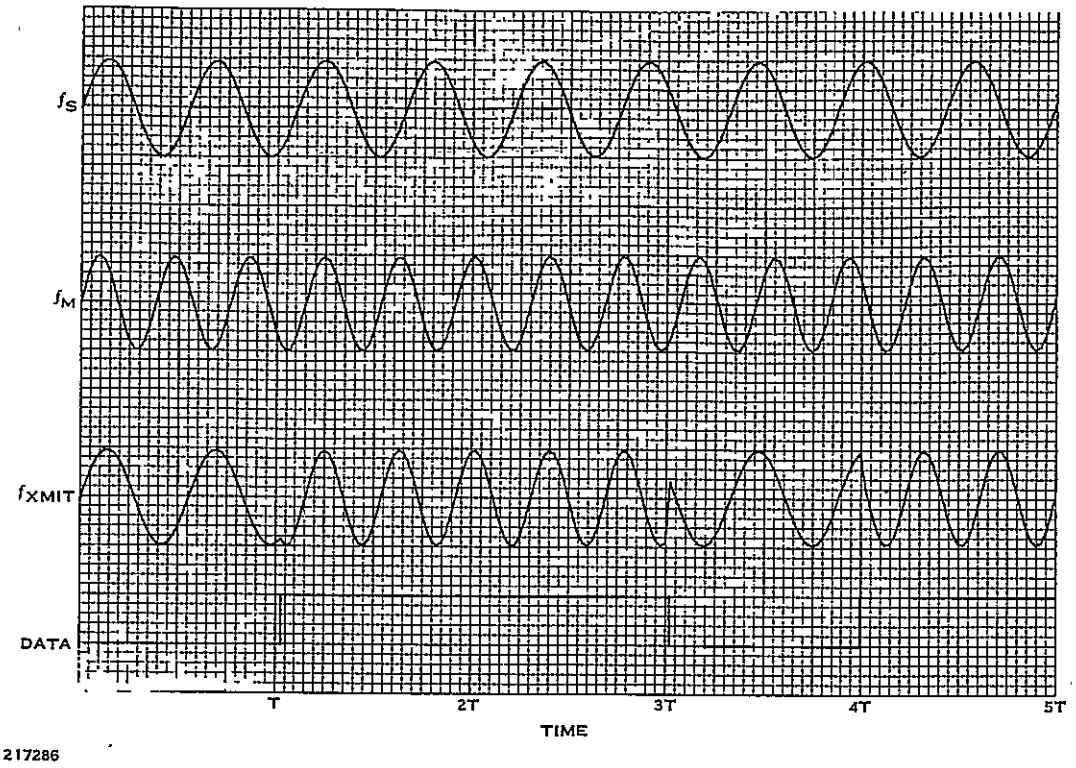


Figure 2-1. Timing/Phase Diagram for Discontinuous-Phase FSK, B = 0.4

$$W(f) = A^2\delta(f - f_s)/8 + A^2\delta(f - f_m)/8 + A^2 G(\omega - \omega_s)/2T(\omega - \omega_s)^2 + A^2 G(\omega - \omega_m)/2T(\omega - \omega_m)^2 \quad (2\text{-}12a)$$

where

$$G(\omega - \omega_s) = \sin^2[(\omega - \omega_s)T/2]$$

$$G(\omega - \omega_m) = \sin^2[(\omega - \omega_m)T/2]$$

This is the power spectral density function when the mark and space signals are defined by:

$$f(t)_m = A \cos(\omega_m t + \phi_m)$$

$$f(t)_s = A \cos(\omega_s t + \phi_s)$$

Note that the rms value of each signal is 0.707A; hence, the power in each wave is proportional to $0.5A^2$. The two delta-functions show the existence of single spectral lines at f_m and f_s, each containing power equal to $0.125A^2$. Hence, 25 percent of the transmitted power is contained in the mark frequency, 25 percent in the space frequency, and 50 percent in the data sidebands. It is this power distribution that governs the signal-to-noise performance of the general case of

FSK. Rearrangement of the last two terms in Equation (2-12a) yields the following interesting result:

$$W(f) = A^2\delta(f - f_s)/8 + A^2\delta(f - f_m)/8 + \frac{A^2T}{8}\left[\frac{\sin^2[\omega - \omega_s)T/2]}{[(\omega - \omega_s)T/2]^2} + \frac{\sin^2[(\omega - \omega_m)T/2]}{[(\omega - \omega_m)T/2]^2}\right] \quad (2\text{-}12b)$$

The third and fourth terms are observed to be similar to the baseband power spectral density of NRZ data as derived by Fredrick L. Chapman:[3]

$$W(f)_{NRZ} = T \sin^2 (\omega T/2)/(\omega T/2)^2 \quad (2\text{-}13)$$

The frequencies have been shifted from $\omega_o = 0$ for NRZ to ω_s and ω_m for FSK. Except for this and an amplitude constant, both are of the form $\sin^2(x)/x^2$. For ω_s and ω_m widely separated, the spectrum is simply two NRZ spectra translated to ω_s and ω_m. However, as ω_s and ω_m become close together, the two spectra overlap. A similar result is produced by Manchester or Miller data.

A second important quality to be observed in Equation (2-12) is that the magnitude of the PSD envelope falls off as the inverse square of $(f - f_o)$ for frequencies far removed from f_o. This is the same as PSK and should be expected since the phase is discontinuous. This will be shown by plotting Equation (2-12) for the modulation index $\beta = 0.4$.

First, the amplitude will be normalized for $A = \sqrt{2}$ which will make the total power equal to unity. The ordinate is plotted as 10 log [W(f)/T]. The horizontal axis is plotted as a parameter x defined by:

$$f = f_o + X f_{ck} \quad (2\text{-}14)$$

for $-5 \leqslant X \leqslant 5$. This causes the spectrum to be symmetrical about f_o, which occurs at $X = 0$; the mark and space frequencies are at $X = \beta$ and $X = -\beta$, respectively. The power spectral density for discontinuous-phase FSK with $\beta = 0.4$ ($f_m - f_s = 0.8\ f_{ck}$) is plotted in Figure 2-2. The total power contained in the spectrum is represented by the reference level of 0 dB. The two spectral lines at the mark and space frequencies have areas equal to 0.25, representing the power content relative to the total power. The first sidelobe is approximately 13 dB down with respect to the primary data lobe. The other sidelobes continue to decrease proportional to the inverse square of $(f - f_{ck})$.

The next group of FSK signals to be considered are continuous-phase FSK. The restriction here is that the phase must be continuous between frequency transitions (Figure 2-3). There are two basic subgroups:

(1) The subgroup in which the mark and space frequencies differ by an integer number of cycles at the end of each bit period and the initial phase of each is arbitrary

[3] F.L. Chapman, "Proposal to Design, Develop, Fabricate, and Test Data Storage Subsystem Electronics and Subsystem Support Equipment for Viking Orbiter 1975," Volume I, Appendix B, Texas Instruments Proposal EG72-095, 1972.

(2) The subgroup in which the mark and space frequencies differ by non-integer multiples of the clock frequency, but the initial phase of the mark or space frequency is controlled at the beginning of each bit period to maintain phase continuity.

The time-domain characteristics of members of subgroup one are shown in Figure 2-3 for continuous-phase FSK (CPFSK) with $\beta = 0.5$. Note that, although the initial phase of f_s is shown as zero, this is not a restriction. It is only necessary that f_m have the same phase as f_s at the beginning of each bit time; i.e., only the relative initial phase is restricted. The key feature to be observed is that there are no phase discontinuities in the modulated output signal even though the frequency is changed; this is in contrast to the waveform of Figure 2-1. The spectral characteristics of this CPFSK signal are shown in Figure 2-4. Note that the magnitude of the spectral sidebands decreases as the inverse of the fourth power of $(f - f_o)$. The mark and space carriers remain, each containing 25 percent of the total power, just as for discontinuous-phase FSK. The primary data energy is observed to occupy a bandwidth of twice the clock frequency. The sidelobes are observed to have peaks occur at multiples of the clock frequency displaced from f_o. The first pair have peaks at $f_o \pm 2f_{ck}$ and are 23 dB down. Note the similarity in the distribution of frequencies for the discontinuous-phase case with $\beta = 0.4$ (Figure 2-2) and the continuous-phase case with $\beta = 0.5$ (Figure 2-4).

It is interesting to compare the spectrum for the CPFSK case with $\beta = 0.4$ with that of the discontinuous-phase case with $\beta = 0.4$. The CPFSK spectrum for $\beta = 0.4$ is plotted in Figure 2-5. Note that the spectrum has no discrete spectral lines, but only a peaking in the PSD near the mark and space frequencies. The primary data lobe is also observed to be compressed, with the first sidelobe pair being located at approximately $\pm f_{ck}$ and 11 dB down with respect to the center of the spectrum. Therefore, continuous-phase FSK has obvious advantages in bandwidth economy.

The case just described ($\beta = 0.4$) is representative of CPFSK signals in subgroup two. The phase is maintained constant when the transition between mark and space frequencies is made. This is easily accomplished with a digital oscillator. There is, however, a special case for signals that have mark and space frequencies differing by odd-integer multiples of one-half the clock frequency. For these cases, the transmit signal is selected from one of four signals:

$$\begin{aligned} &(1)\ A\cos(\omega_m t + \phi) \\ &(2)\ A\cos(\omega_m t + \phi + n\pi) \\ &(3)\ A\cos(\omega_s t + \phi) \\ &(4)\ A\cos(\omega_s t + \phi + n\pi) \end{aligned} \qquad (2\text{-}15)$$

where n is an *odd* integer and $(\omega_m - \omega_s) = n\omega_{ck}/2$. MSK is the special case where $\beta = 0.25$ and $n = 1$. For this case, the PSD is compressed the maximum amount so that most of the energy is contained in a bandwidth equal to the clock frequency and no discrete spectral components are produced. In addition, the spectral sidelobes are suppressed the maximum amount. These two properties are the result of a minimum frequency shift ($0.5\ f_{ck}$) and continuous phase at the time of frequency transitions. The time-domain characteristics of MSK are shown in Figure 2-6. Note that the inverse of the mark signal is selected at $t = T$ to maintain phase continuity.

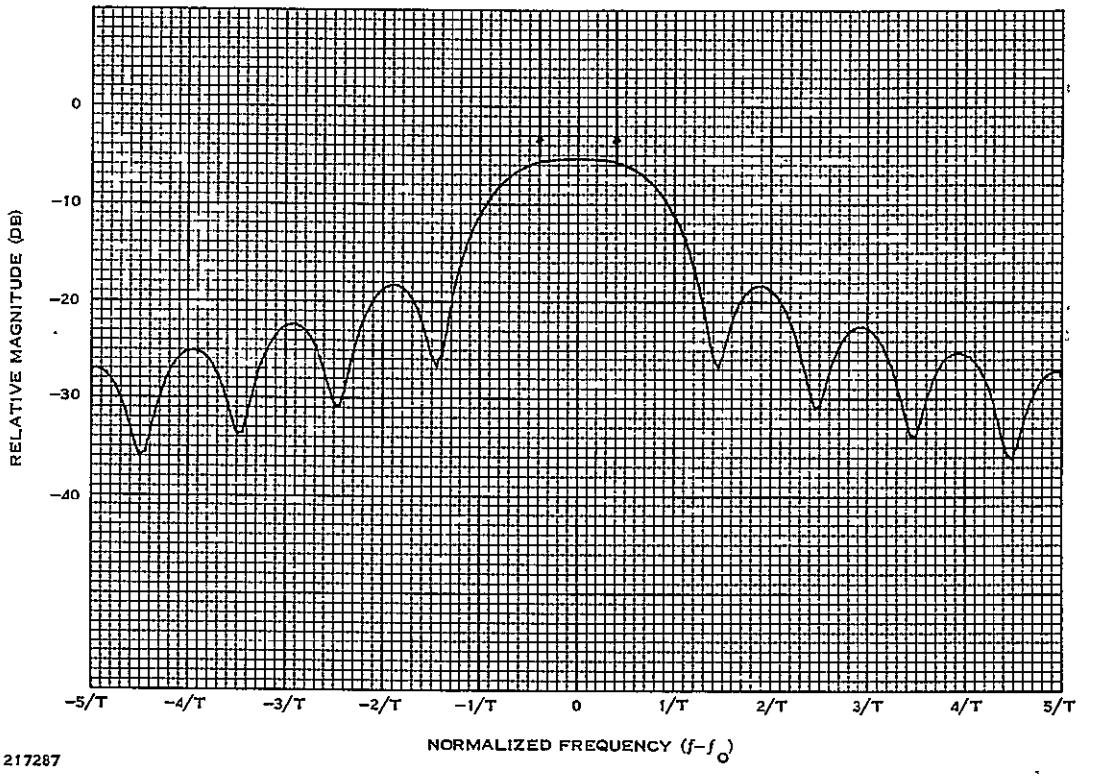


Figure 2-2. Power Spectral Density of Discontinuous-Phase FSK, B = 0.4

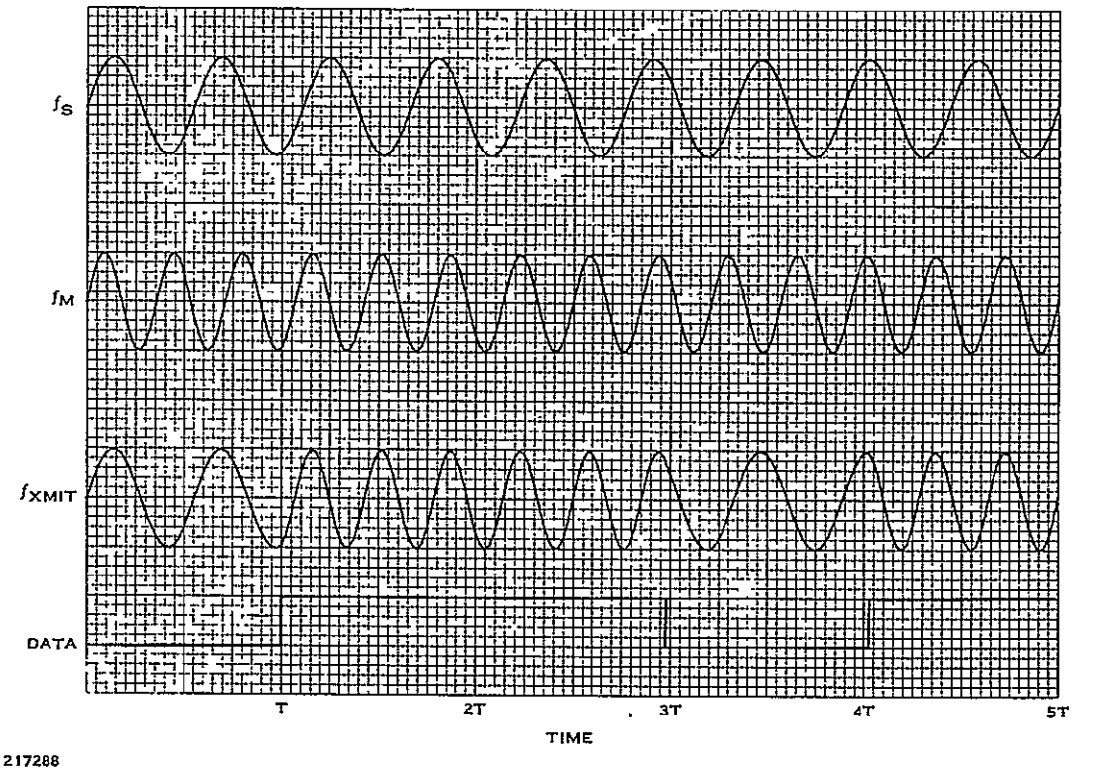


Figure 2-3. Timing/Phase Diagram for Continuous-Phase FSK, B = 0.5

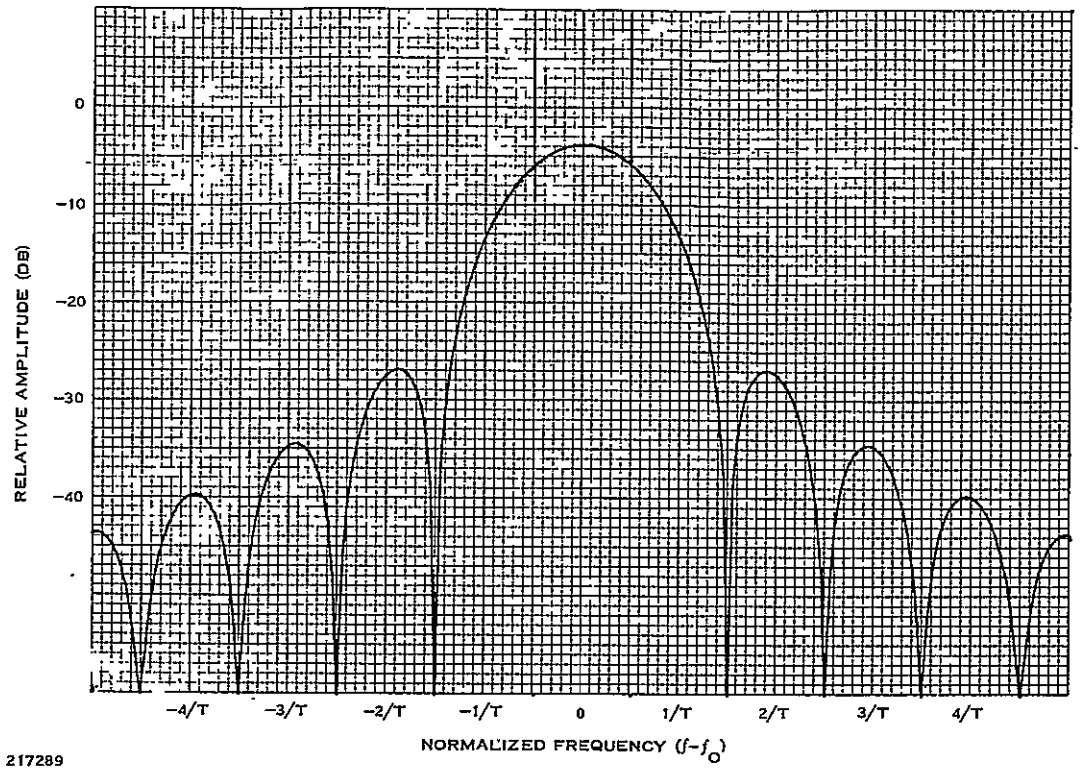


Figure 2-4. Power Spectral Density of Continuous-Phase FSK, B = 0.5

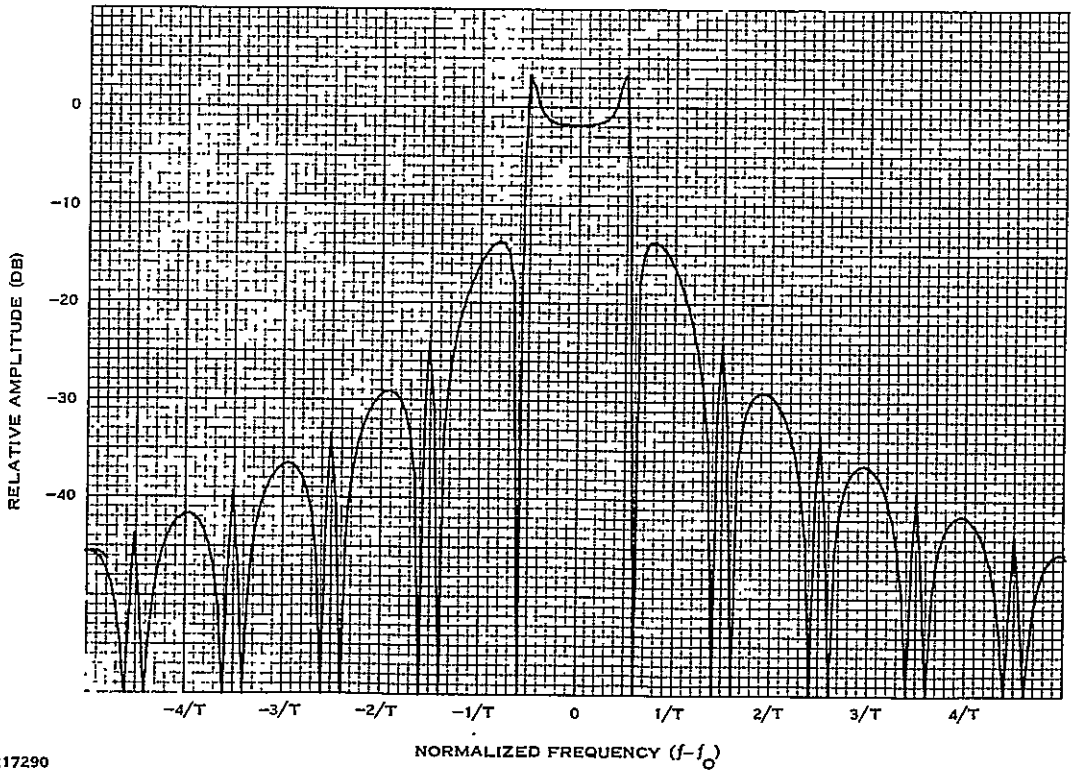


Figure 2-5. Power Spectral Density of Continuous-Phase FSK, B = 0.4

The spectral properties of MSK are shown in Figure 2-7. Note the very dense primary spectral lobe. The first pair of sidelobes at $f_o \pm f_{ck}$ are down 23 dB. When compared with Figure 2-4, the bandwidth is observed to be only one-half that required for $\beta = 0.5$. This is very important from the viewpoint of adjacent-channel interference when the transmitter output cannot be filtered. Comparison with Figure 2-5 shows that the first pair of sidelobes for MSK is attenuated approximately 10 dB more than those for CPFSK with $\beta = 0.4$. In addition, the primary data sidelobe is denser for MSK. The advantage of MSK in terms of spectral efficiencies is obvious.

All continuous-phase FSK power spectral densities were obtained from the following:[4]

$$W(f) = \frac{2A^2 \sin^2[(\omega - \omega_s)T/2] \sin^2[\omega - \omega_m)T/2]}{T[1 - 2\cos(\omega t - \alpha T)\cos(2\pi\beta) + \cos^2(2\pi\beta)]} \cdot \left[\frac{1}{(\omega - \omega_s)} - \frac{1}{(\omega - \omega_m)}\right]^2$$
$$+ \frac{2A^2 \sin^2[\omega + \omega_s)T/2] \sin^2[(\omega + \omega_m)T/2]}{T[1 - 2\cos(\omega T + \alpha T)\cos(2\pi\beta) + \cos^2(2\pi\beta)} \cdot \left[\frac{1}{(\omega + \omega_s)} - \frac{1}{(\omega + \omega_m)}\right]^2 \quad (2\text{-}16)$$

where

$$T = 1/f_{ck}$$

$$\alpha = 1/2\,(\omega_s + \omega_m) = \omega_o$$

and all other symbols are as previously defined.

All plots were normalized for $A = \sqrt{2}$ to make the 0 dB reference represent the total power of a single continuous tone. The vertical scale was normalized to represent:

$$Y = 10 \log [W(f)/T]$$

The horizontal scale was normalized to f_o, with each major division being equal to f_{ck}. Therefore, all spectra are normalized and represent the modulation characteristics, regardless of the center frequency or clock frequency.

The characteristics of MSK as a special case of FSK have been presented in both the time domain and the frequency domain. The definition of MSK in terms of FSK is now stated:

> MSK is a special case of FSK having a modulation index of 0.25 ($f_m - f_s = f_{ck}/2$) and continuous phase at the transitions.

This is a necessary and sufficient restriction to characterize MSK. In particular, it is not necessary for the mark or space frequencies to be integer multiples of the clock frequency.

The above definition is necessary and sufficient, but it does not provide a rigorous mathematical definition relating the transmitted MSK signal to the data and bit clock. By

[4] Bennet and Rice.

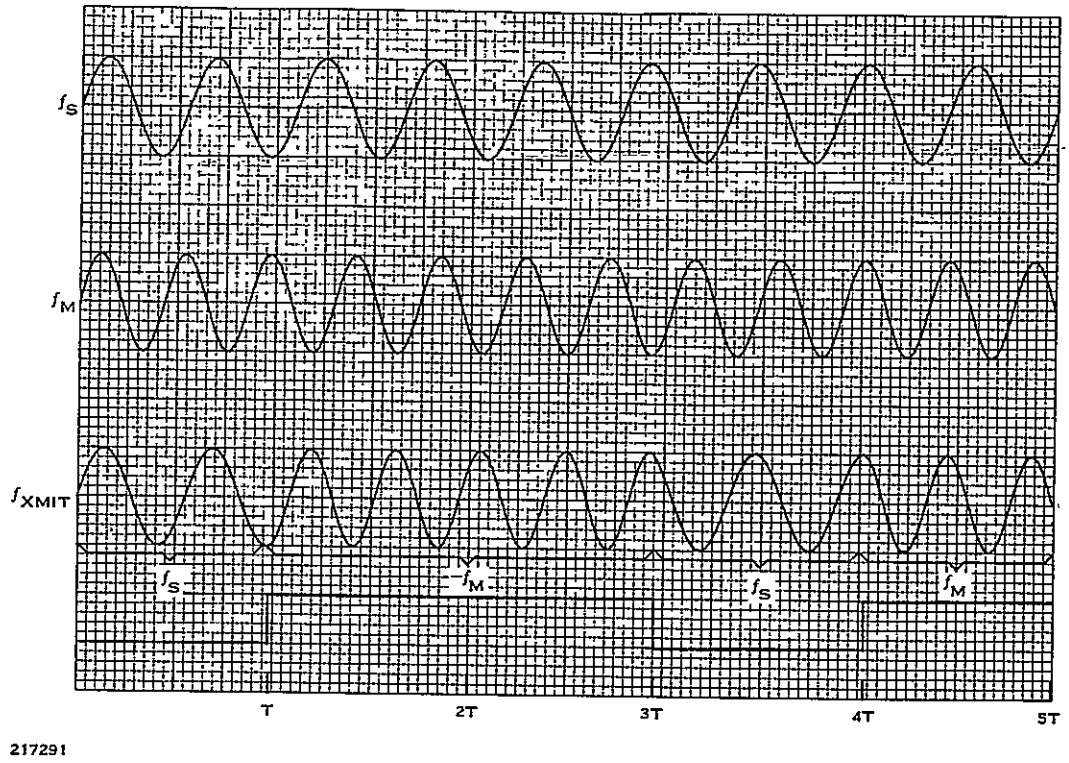


Figure 2-6. Timing/Phase Diagram for MSK

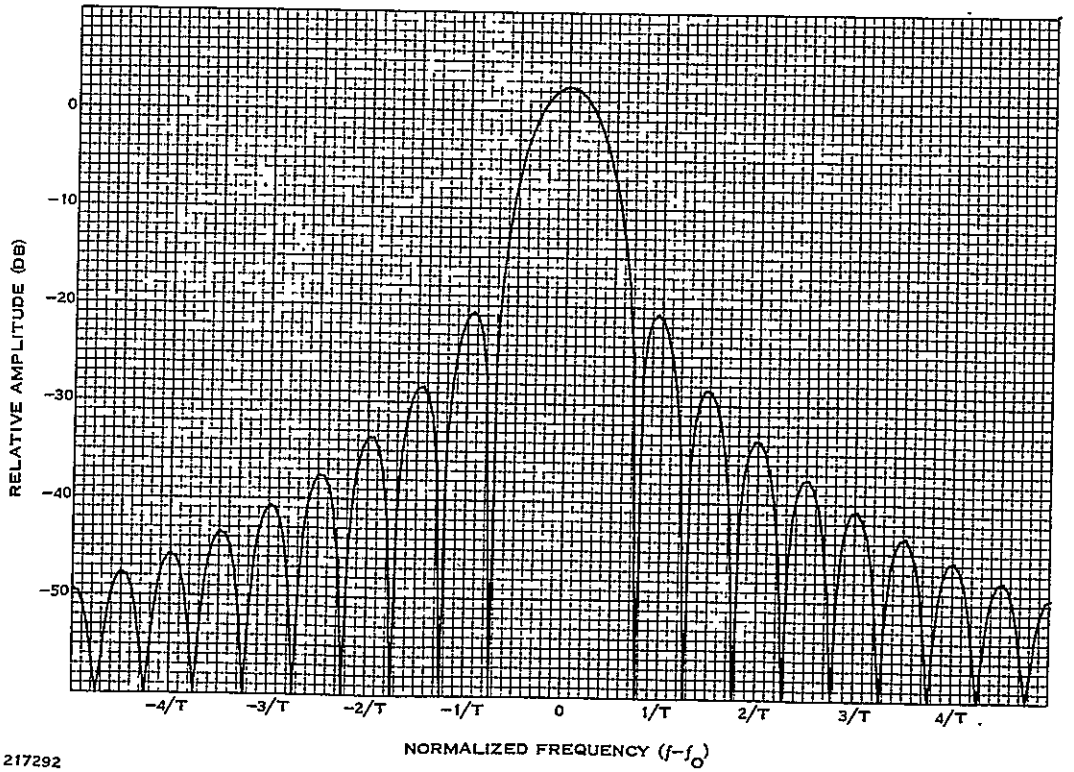


Figure 2-7. Power Spectral Density of MSK

beginning with Equation (2-15) and letting the arbitrary initial phase, ϕ equal zero and n = 1 for MSK, the transmit signal is selected by the data from a set of four signals:

$$\begin{aligned} S1(t) &= A \cos \omega_m t \\ S2(t) &= -A \cos \omega_m t \\ S3(t) &= A \cos \omega_s t \\ S4(t) &= -A \cos \omega_s t \end{aligned} \tag{2-17}$$

Since the signal is selected only at the time of a data transition and the selected signal is maintained for one bit period, the modulator output signal is given by:

$$y(t) = S(nT)$$

for

$$nT \leqslant t < (n + 1)T$$

The selection of one of the four signals is mutually exclusive; i.e., only one signal can be selected at a given time.

From the definition of MSK, a logic "1" selects a mark signal, either S1(t) or S2(t). A logic "0" selects either S3(t) or S4(t). However, the phase of the mark or space signal is selected to meet the phase continuity requirements of MSK. Now make the following definitions:

$S1_n = 1$ represents the logic selection of the S1(t) signal at data time t = nT

$S1_n = 0$ represents the logic deselection of the S1(t) signal at data time t = nT

Similar definitions are made for $S2_n$, $S3_n$, and $S4_n$. It is also appropriate to represent the logic state of each Boolean variable at an earlier time by decrementing the subscript; e.g., $S1_{n-1}$ represents the state of the S1 variable at time (n − 1)T. (Of course, the value assigned a variable at data time is maintained for one bit period.) Furthermore, define the input data (NRZ-L) as:

D_n = the logic variable for the input data

As an arbitrary reference, at n = 0, let $\cos \omega_m t = \cos \omega_s t$ and also require the derivatives to be equal. Then, the following states are dictated by teh requirements stated in the definition of MSK:

$S1_n = 1$ if $D_n = 1$ and $S1_{n-1} = 1$
or $D_n = 1$ and $S3_{n-1} = 1$ and n is even
or $D_n = 1$ and $S4_{n-1} = 1$ and n is odd

$S1_n = 0$ otherwise

$S2_n = 1$ if $D_n = 1$ and $S2_{n-1} = 1$
or $D_n = 1$ and $S3_{n-1} = 1$ and n is odd
or $D_n = 1$ and $S4_{n-1} = 1$ and n is even

$S2_n = 0$ otherwise

$S3_n = 1$ if $D_n = 0$ and $S3_{n-1} = 1$

or $D_n = 0$ and $S1_{n-1} = 1$ and n is even

or $D_n = 0$ and $S2_{n-1} = 1$ and n is odd

$S3_n = 0$ otherwise

$S4_n = 1$ if $D_n = 0$ and $S4_{n-1} = 1$

or $D_n = 0$ and $S1_{n-1} = 1$ and n is odd

or $D_n = 0$ and $S2_{n-1} = 1$ and n is even

$S4_n = 0$ otherwise

Now, since n is a binary signal (even or odd), a square-wave signal at one-half the data clock frequency can be used to determine whether n is odd or even. The time function is defined by:

$$n(t) = 1 \text{ for } nT < t < (n + 1)T, \text{ n even}$$

$$N(t) = 0 \text{ for } nT < t < (n + 1)T, \text{ n odd}$$

Therefore, N(t) = N for n even while N(t) = $\overline{N}$ for n odd. The period of this signal is 2T.

The above logic statements can now be converted to Boolean logic equations:

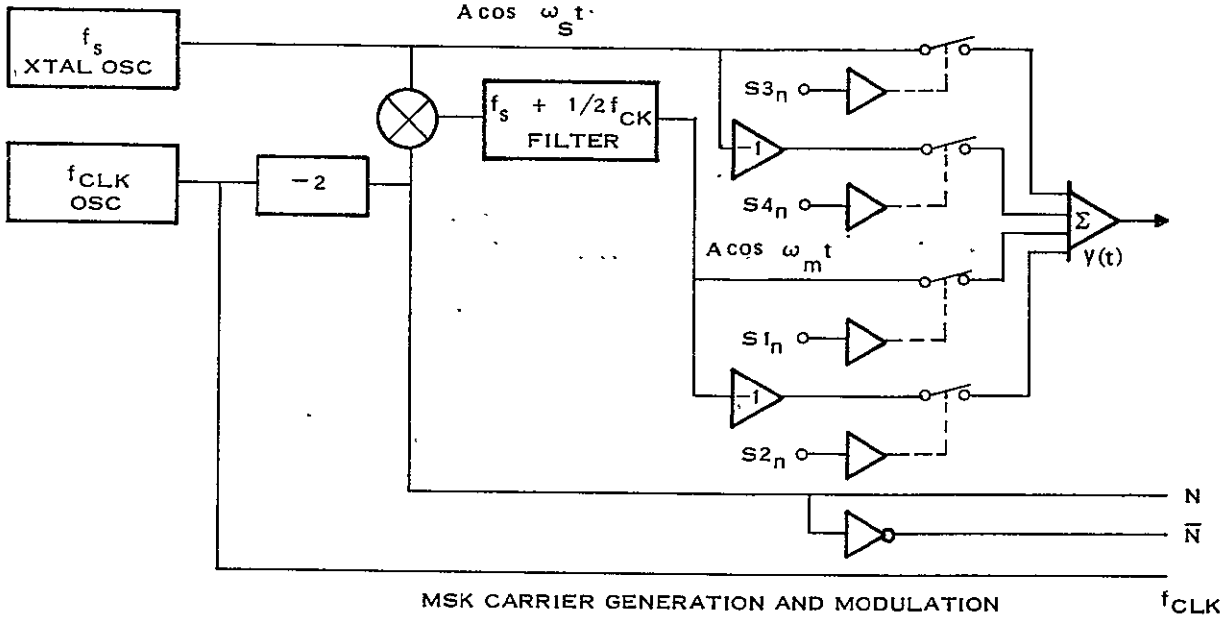
$$\begin{aligned} S1_n &= D_n S1_{n-1} + D_n S3_{n-1} N + D_n S4_{n-1} \overline{N} \\ S2_n &= D_n S2_{n-1} + D_n S3_{n-1} \overline{N} + D_n S4_{n-1} N \\ S3_n &= \overline{D}_n S3_{n-1} + \overline{D}_n S1_{n-1} N + \overline{D}_n S2_{n-1} \overline{N} \\ S4_n &= \overline{D}_n S4_{n-1} + \overline{D}_n S1_{n-1} \overline{N} + \overline{D}_n S2_{n-1} N \end{aligned} \qquad (2\text{-}18)$$

These equations can be used to implement a *direct* approach to MSK generation. They also represent the implicit relationships between the data, clock, and output signal when a digital modulation scheme is used to generate MSK. A schematic of the direct approach is shown in Figure 2-8.

The direct approach shown generates a space carrier frequency from a stable source. The data clock signal is divided by two and mixed with the space carrier; the sum is selected by the (f_s + 1/2 f_{ck}) bandpass filter to produce the mark carrier. The correct phase mark or space frequency is selected by the analog switches driven by the encoder logic. The encoder logic implements Equation (2-18). Note that the 1/2 f_{ck} signal used to generate the mark signal is also used for N; it is derived from f_{ck}.

Although the direct approach shown uses analog switches to perform the modulation function, ring modulators can be used when operating at high RF frequencies. However, the design of the bandpass filter for the mark carrier generation places a lower limit on the data rate for a given carrier frequency. Therefore, it may be necessary to perform the modulation at a low frequency (for low data rates) and then translate to the desired output frequency.

When the data rate is sufficiently low and translation is to be used, an all-digital means of generating MSK is both feasible and attractive. An all-digital modulator is described in detail in the main body of this report (Subsection 2.D.3). The modulator was built and used to perform tests described in Section 4 of this addendum. For the sake of completeness, a generalized block diagram of this approach is shown in Figure 2-9.



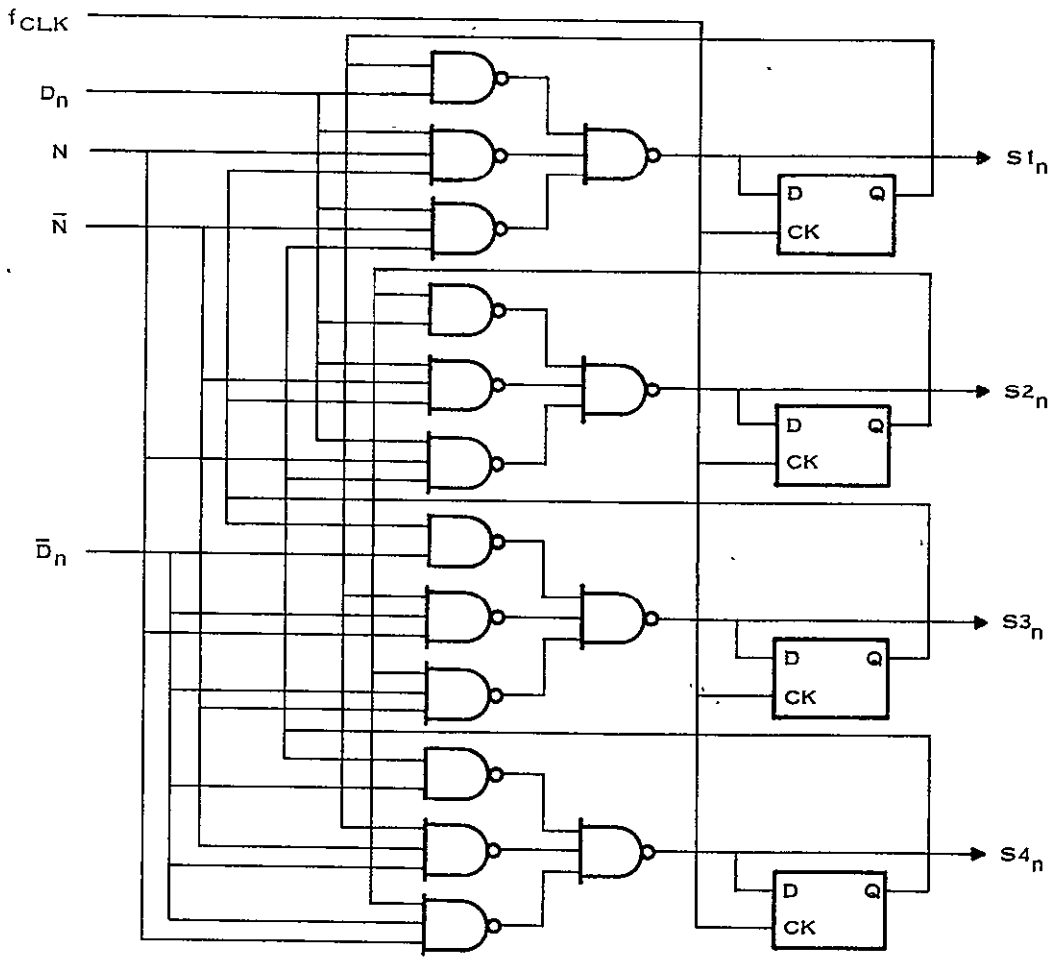


Figure 2-8. Schematic Diagram of Direct Approach of MSK Generation

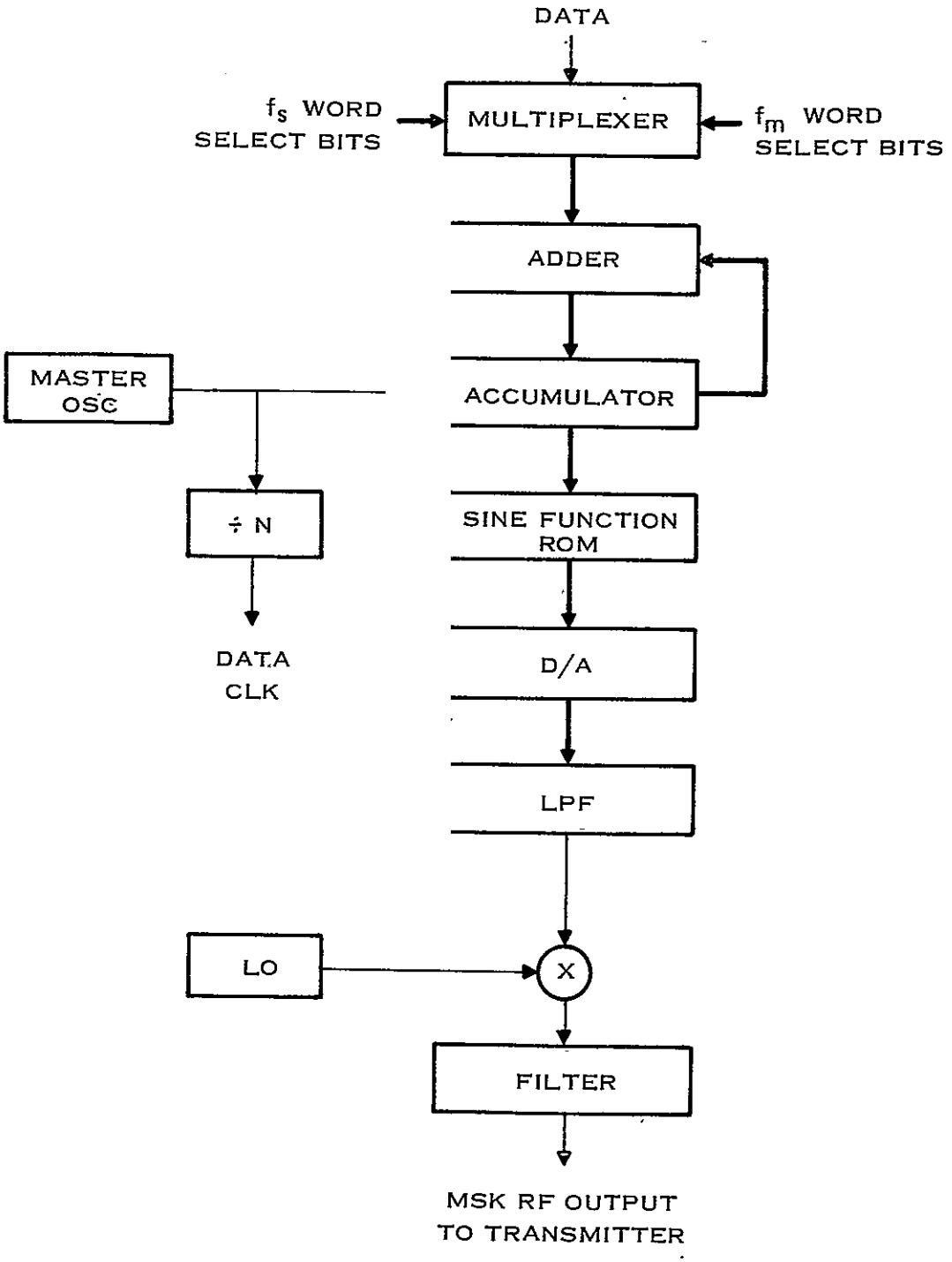


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Figure 2-9. Digital MSK Generator

This implementation is based on stepping-through samples of the sine function stored in a ROM. The number of samples taken is varied to determine the desired output frequency. A D/A converter, followed by a filter, converts the sampled sine wave into a continuous sine wave of the same frequency. The rate at which the ROM is scanned is determined by the rate at which the ROM address is incremented and the size of each address increment. This, in turn, is determined by the size of the f_s or f_m words added to the contents of the accumulator. The accumulator is clocked by a signal many times the data clock frequency. The output frequency is determined by the relationships between the accumulator clock, the data clock, the size of the mark/space increment words added, and the number of words used to represent the sine function stored in the ROM.

This scheme is attractive when the data rates are relatively low (<100 KPBS). It will easily provide output frequencies up to about 1 MHz. For higher transmit frequencies, the output must be translated as shown in Figure 2-9.

A final consideration for generating MSK as an FSK signal involves applying the data to a VCO and using a modulation index of 0.25. In this case, the VCO frequency is increased to $f_o + f_{ck}/4$ for a one and decreased to $f_o - f_{ck}/4$ for a zero. Phase continuity is maintained since a single oscillator is used to generate both the mark and the space frequencies. The major problem in implementing this approach is maintaining a modulation index of precisely 0.25.

2.1.2 MSK Viewed as a Special Case of Offset-Keyed QPSK

The previous discussions have treated MSK as a special case of FSK. This subsection will describe the mathematical link that enables MSK to be treated as a special case of QPSK and, hence, the basis for the coherent demodulator built and tested as a part of this study.

Returning to Equation (2-17), make the following definitions:

V_n = selection of the mark frequency

$\overline{V}_n$ = selection of the space frequency

U_n = selection of the noninverted signal

$\overline{U}_n$ = selection of the inverted signal.

then

$$\begin{aligned} S1_n &= U_n V_n \\ S2_n &= \overline{U}_n V_n \\ S3_n &= U_n \overline{V}_n \\ S4_n &= \overline{U}_n \overline{V}_n \end{aligned} \tag{2-19}$$

It is obvious that the data determines V_n, i.e.

$$V_n = D_n$$

The question is: What is U_n? To solve this problem, considerable use will be made of modulo-2 algebra. Only the results are sketched here, but a complete derivation is contained in Appendix A. The symbol $\oplus$ is used to indicate modulo-2 addition or the exclusive-OR function.

To begin, perform the following:

$$S1_n \oplus S3_n = U_n V_n \oplus U_n \overline{V}_n = U_n \ (V_n + \overline{V}_n) = U_n$$

or

$$U_n = S1_n \oplus S3_n$$

Now, making use of Equation (2-18):

$$\begin{aligned} U_n = \ & (D_n S1_{n-1} + D_n S3_{n-1} N + D_n S4_{n-1} \overline{N}) \oplus \\ & (\overline{D}_n S3_{n-1} + \overline{D}_n S1_{n-1} N + \overline{D}_n S2_{n-1} \overline{N}) \end{aligned}$$

But since $D_n = V_n$, then:

$$\begin{aligned} S1_{n-1} &= U_{n-1} D_{n-1} \\ S2_{n-1} &= \overline{U}_{n-1} D_{n-1} \\ S3_{n-1} &= U_{n-1} \overline{D}_{n-1} \\ S4_{n-1} &= \overline{U}_{n-1} \overline{D}_{n-1} \end{aligned}$$

Therefore,

$$\begin{aligned} U_n = \ & (D_n D_{n-1} U_{n-1} + D_n \overline{D}_{n-1} U_{n-1} N + \overline{D}_n \overline{D}_{n-1} \overline{U}_{n-1} \overline{N}) \oplus \\ & (\overline{D}_n \overline{D}_{n-1} U_{n-1} + \overline{D}_n D_{n-1} U_{n-1} N + \overline{D}_n D_{n-1} \overline{U}_{n-1} \overline{N}) \end{aligned}$$

After considerable algebraic manipulations and reduction, it can be shown that:

$$U_n = U_{n-1} \oplus \overline{N} \ (D_n \oplus D_{n-1}) \tag{2-20}$$

The U,V equations defined by Equation (2-19) apply to the FSK definition of MSK. Now, define the mark/space frequencies in terms of a center frequency, ω_o:

$$\begin{aligned} \omega_m &= \omega_o + \frac{\pi}{2T} \\ \omega_s &= \omega_o - \frac{\pi}{2T} \end{aligned} \tag{2-21}$$

where T is the bit period as before. [These equations are obtained from Equation (2-11) with β = 0.25 for MSK.] Substitution into Equation (2-17) results in:

$$
\begin{aligned}
S1(t) &= A \cos \left(\omega_o + \frac{\pi}{2T}\right) t \\
S2(t) &= -A \cos \left(\omega_o + \frac{\pi}{2T}\right) t \\
S3(t) &= A \cos \left(\omega_o - \frac{\pi}{2T}\right) t \\
S4(t) &= -A \cos \left(\omega_o - \frac{\pi}{2T}\right) t
\end{aligned}
\tag{2-22}
$$

Trigonometric expansion yields:

$$
\begin{aligned}
S1(t) &= A \cos(\omega_o t) \cos\left(\frac{\pi t}{2T}\right) - A \sin(\omega_o t) \sin\left(\frac{\pi t}{2T}\right) \\
S2(t) &= -A \cos(\omega_o t) \cos\left(\frac{\pi t}{2T}\right) + A \sin(\omega_o t) \sin\left(\frac{\pi t}{2T}\right) \\
S3(t) &= A \cos(\omega_o t) \cos\left(\frac{\pi t}{2T}\right) + A \sin(\omega_o t) \sin\left(\frac{\pi t}{2T}\right) \\
S4(t) &= -A \cos(\omega_o t) \cos\left(\frac{\pi t}{2T}\right) - A \sin(\omega_o t) \sin\left(\frac{\pi t}{2T}\right)
\end{aligned}
\tag{2-23}
$$

For simplicity, let A = 1. Now, the four components can be expressed as the algebraic sum of two quadrature phasor components. The sign of each phasor component is determined by the data. The output signal, y(t), can be expressed in general form by the following I,Q equation:

$$
y(t) = I(t) \cos(\omega_o t) \cos\left(\frac{\pi t}{2T}\right) + Q(t) \sin(\omega_o t) \sin\left(\frac{\pi t}{2T}\right) \tag{2-24}
$$

where I(t) = ±1 and Q(t) = ±1 are determined by the data. Now, form the following definitions:

$$
\begin{aligned}
I_n &= 1 \\
\overline{I}_n &= 0 \\
Q_n &= 1 \\
\overline{Q}_n &= 0
\end{aligned}
$$

From Equation (2-23), the following logic equations can be formed:

$$
\begin{aligned}
S1_n &= I_n \overline{Q}_n \\
S2_n &= \overline{I}_n Q_n \\
S3_n &= I_n Q_n \\
S4_n &= \overline{I}_n \overline{Q}_n
\end{aligned}
\tag{2-25}
$$

Combining Equations (2-19) and (2-25):

$$\begin{aligned} U_n V_n &= I_n \bar{Q}_n \\ \bar{U}_n V_n &= \bar{I}_n Q_n \\ U_n \bar{V}_n &= I_n Q_n \\ \bar{U}_n \bar{V}_n &= \bar{I}_n \bar{Q}_n \end{aligned}$$

Once again, using modulo-two algebra:

$$\begin{aligned} UV \oplus U\bar{V} &= I\bar{Q} \oplus IQ \\ U(V \oplus \bar{V}) &= I(\bar{Q} \oplus Q) \\ U &= I \end{aligned}$$

and

$$\begin{aligned} UV \oplus \bar{U}V &= I\bar{Q} \oplus \bar{I}Q \\ V(U \oplus \bar{U}) &= (I\bar{Q})(I + \bar{Q}) + \bar{I}Q(\bar{I} + Q) \\ V &= I\bar{Q} + I\bar{Q} + \bar{I}Q + \bar{I}Q \\ &= I\bar{Q} + \bar{I}Q \\ \therefore V &= I \oplus Q \end{aligned} \qquad (2\text{-}26)$$

Thus

$$\begin{aligned} U &= I \\ V &= I \oplus Q \end{aligned}$$

Conversely, simple algebraic (modulo-2) manipulation yields:

$$\begin{aligned} I &= U \\ Q &= U \oplus V \end{aligned} \qquad (2\text{-}27)$$

Substituting for U and V in Equation (2-27) and suitable manipulation yields:

$$\begin{aligned} I_n &= I_{n-1} \oplus \bar{N}(D_n \oplus D_{n-1}) \\ Q_n &= Q_{n-1} \oplus N(D_n \oplus D_{n-1}) \end{aligned} \qquad (2\text{-}28)$$

Equations (2-26) and (2-27) show the link between the FSK (U,V) symbols and the PSK (I,Q) symbols. Equation (2-28) defines the I,Q symbols in terms of the data and the $f_{ck}/2$ signal, N. Note that I_n and Q_n are permitted to change only every other bit time since, when N = 1, $\bar{N}$ = 0, and $I_n = I_{n-1} \oplus 0 = I_{n-1}$ while $Q_n = Q_{n-1} \oplus (D_n \oplus D_{n-1})$. Then, at $t = (n + 1)T$, N = 0, and N = 1; $Q_{n+1} = Q_n$ and $I_{n+1} = I_n \oplus (D_{n+1} \oplus D_n)$. Therefore, these I and Q symbols are

precisely those used in an offset-keyed QPSK modulation system. However, in OK-QPSK, the symbols are applied directly to the in-phase and quadrature carriers while in MSK, the I and Q symbols are weighted by the cos ($\pi t/2T$) and sin ($\pi t/2T$) functions respectively, as shown by Equation (2-24).

These equations provide the basis for the design of both a modulator and a demodulator. Although the MSK coherent demodulator is the primary objective of this study, a brief description of a modified OK-QPSK modulation scheme is in order. Such a modulator is shown in Figure 2-10.

The input data is in NRZ-L format. The first encoder differentially encodes the data with itself, thus implementing the ($D_n \oplus D_{n-1}$) term in Equation (2-28). The bit clock is divided by two to produce the signal, N, of Equation (2-28). This signal is used as a sampling clock to steer alternate bits to the I and Q channels. The I and Q channels each contain a differential encoder to complete the implementation of Equation (2-28).

A stable carrier generator (e.g., crystal oscillator) produces the mean frequency, f_o, that is shown as cos $\omega_o t$. A −90-degree phase shift (produced by a simple integrator circuit) provides the quadrature carrier, sin $\omega_o t$. Antipodal PSK modulation of both the I and Q carriers is achieved by a double-balanced modulator in each channel. The two outputs, I cos $\omega_o t$ and Q sin $\omega_o t$, would produce OK-QPSK if summed together at this point. MSK is produced by respectively mixing the I and Q modulated carriers with cos ($\pi t/2T$) and sin ($\pi t/2T$) signals before adding. The two signals are derived from the bit clock as suggested by the PLL; the output frequency of the loop is $f_{ck}/4$.

Equation (2-24) provides the relationship for coherent demodulation of MSK. First, assume that a carrier, ω_o, is available. Then, sin $\omega_o t$ and cos $\omega_o t$ are available. Multiplication of y(t) by cos $\omega_o t$ yields:

$$y(t)\cos(\omega_o t) = I(t)\cos^2(\omega_o t)\cos\left(\frac{\pi t}{2T}\right) + Q(t)\sin(\omega_o t)\cos(\omega_o t)\sin\left(\frac{\pi t}{2T}\right)$$

$$= I(t)\left[1 - \sin^2(\omega_o t)\right]\cos\left(\frac{\pi t}{2T}\right) + Q(t)\sin\left(\frac{\pi t}{2T}\right)\left[1/2\sin(2\omega_o t)\right]$$

$$= I(t)\cos\left(\frac{\pi t}{2T}\right)\left[1/2 - 1/2\cos(2\omega_o t)\right] + Q(t)\sin\frac{\pi t}{2T}\left[1/2\sin(2\omega_o t)\right]$$

$$= 1/2I(t)\cos\left(\frac{\pi t}{2T}\right) - 1/2I(t)\cos\left(\frac{\pi t}{2T}\right)\cos(2\omega_o t) + 1/2Q(t)\sin\left(\frac{\pi t}{2T}\right)\sin(\omega_o t)$$

The first term on the right contains the demodulated I-channel information; the other terms contain only carrier/sideband components at $2\omega_o$. Therefore, suitable filtering can remove the carrier components, leaving only the I-channel information term, 1/2I(t) cos ($\pi t/2T$). Similar multiplication of y(t) by sin ($\omega_o t$) yields the Q-channel information term, 1/2Q(t) sin($\pi t/2T$).

Unfortunately, the MSK spectrum contains no discrete spectral line at ω_o; neither do any of the other classes of CPFSK signals using NRZ data. This makes recovery of a coherent carrier

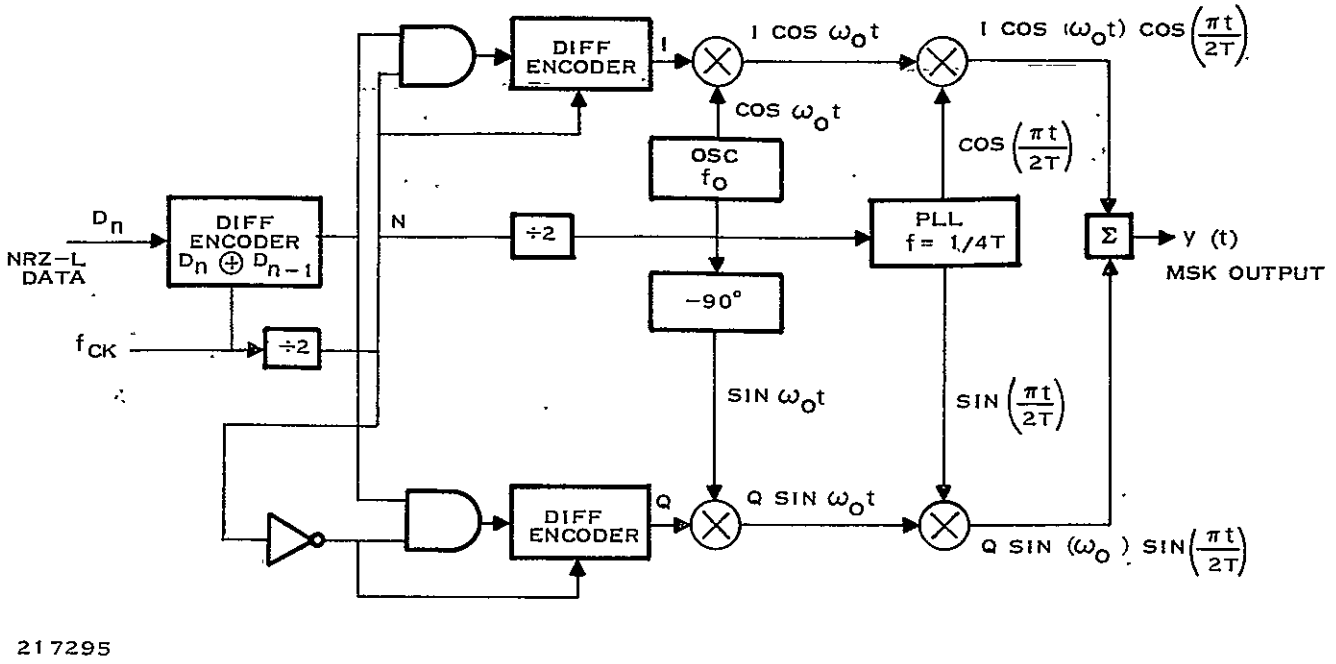

Figure 2-10. PSK Method of Generating MSK

impossible. In addition, MSK/NRZ contains *no* discrete spectral lines at *any* frequency as shown in Figure 2-7. If the MSK frequency is doubled, however, the modulation index is also doubled; i.e., $\beta = 0.5$. The frequency-doubled signal is equivalent to a CPFSK signal with $\beta = 0.5$ at twice the signaling frequency. Thus, the frequency-doubled spectrum contains a spectral line at twice the *mark* frequency and a spectral line at twice the *space* frequency (Figure 2-4). This makes recovery of a mark and a space carrier frequency possible.

A simple method for doubling the frequency is squaring the input signal, $[y(t)]^2$. The mark signal, from Equation (2-17), is:

$$Y_m(t) = \pm A \cos(\omega_m t)$$

Squaring this signal yields:

$$[y_m(t)]^2 = 1/2A^2 \ [1 + \cos(2\omega_m t)]$$

This operation produces twice the mark frequency and a DC term. A similar result is produced for the space frequency. A PLL or ringing filter recovers each frequency; division by 2 produces a mark carrier and a space carrier..

The problem now is to use these carriers to coherently demodulate the MSK signal. From Equation (2-21):

$$\cos(\omega_m t) = \cos\left(\omega_o t + \frac{\pi t}{2T}\right) = \cos(\omega_o t)\cos\left(\frac{\pi t}{2T}\right) - \sin(\omega_o t)\sin\left(\frac{\pi t}{2T}\right)$$

$$\cos(\omega_s t) = \cos\left(\omega_o t - \frac{\pi t}{2T}\right) = \cos(\omega_o t)\cos\left(\frac{\pi t}{2T}\right) + \sin(\omega_o t)\sin\left(\frac{\pi t}{2T}\right)$$

Adding these signals yields:

$$\cos(\omega_m t) + \cos(\omega_s t) = 2\cos(\omega_o t)\cos\left(\frac{\pi t}{2T}\right) \tag{2-29}$$

Subtraction yields:

$$\cos(\omega_s t) - \cos(\omega_m t) = 2\sin(\omega_o t)\sin\left(\frac{\pi t}{2T}\right) \tag{2-30}$$

Multiplying Equation (2-24) by Equation (2-29) yields:

$$\begin{aligned} y(t)\,[\cos(\omega_m t) + \cos(\omega_s t)] &= 2I(t)\cos^2(\omega_o t)\cos^2\left(\frac{\pi t}{2T}\right) \\ &\quad + 2Q(t)\cos(\omega_o t)\sin(\omega_o t)\cos\left(\frac{\pi t}{2T}\right)\sin\left(\frac{\pi t}{2T}\right) \\ &= 2I(t)\,[1/2 + 1/2\cos(2\omega_o t)]\left[1/2 + 1/2\cos\left(\frac{\pi t}{T}\right)\right] \\ &\quad + 2Q(t)\,[1/2\sin(2\omega_o t)]\left[1/2\sin\left(\frac{\pi t}{T}\right)\right] \\ &= 1/2I(t)\left[1 + \cos\left(\frac{\pi t}{T}\right) + \cos(2\omega_o t) + \cos(2\omega_o t)\cos\left(\frac{\pi t}{T}\right)\right] \\ &\quad + 1/2Q(t)\left[\sin(2\omega_o t)\sin\left(\frac{\pi t}{T}\right)\right] \end{aligned}$$

Simple filtering will remove all RF components, leaving only the demodulated I-channel data:

$$I'(t) = 1/2I(t)\left[1 + \cos\left(\frac{\pi t}{T}\right)\right] \tag{2-31}$$

Similar multiplication of Equations (2-24) and (2-30) yields:

$$Q'(t) = 1/2Q(t)\left[1 - \cos\left(\frac{\pi t}{T}\right)\right] \tag{2-32}$$

Recalling that the I and Q symbols exist for two bit times, $I'(t)$ and $Q'(t)$ can be integrated over time 2T. The integral of $\cos(\pi t/T)$ over period 2T is zero. Therefore, the integral of $I'(t)$ and $Q'(t)$ over 2T is:

$$\int_0^{2T} I'(t)\,dt = \int_0^{2T} 1/2I(t)\,dt = T\,I(t) \tag{2-33}$$

since I(t) = ±1. Also, integration of Equation (2-32) yields:

$$\int_0^{2T} Q'(t)\,dt = T\,Q(t) \tag{2-34}$$

A simple decision circuit and sampling device following each integrator completes the data demodulation and detection circuitry, except for bit-clock recovery.

Recovery of the bit clock is relatively simple. The bit-clock frequency is related to the mark/space frequencies by:

$$f_{ck}/2 = f_m - f_s$$

This difference frequency is easily obtained from the recovered mark/space carriers by multiplication:

$$\cos(\omega_m t)\cos(\omega_s t) = \cos(\omega_m + \omega_s)t + \cos(\omega_m - \omega_s)t \tag{2-35}$$

Simple filtering extracts the difference frequency, $f_{ck}/2$. This signal is then used as a reference for a PLL that multiplies this signal to provide f_{ck} and other timing signals.

The final decoding operation needed to reconstruct the original NRZ data is given by Equation (2-26). Since $V_n = D_n$:

$$D_n = I_n \oplus Q_n \tag{2-36}$$

Note that this is a simple differential decoder and that, if both I_n and Q_n are inverted, the correct data polarity is maintained. Also note that for NRZ data, the clock polarity is unimportant for decoding.

The final consideration for a coherent demodulator is phase ambiguities in the recovered carriers and clock. Ambiguities arise because of the squaring (doubling) of the input signal and subsequent division by 2 of the recovered carriers. This division by 2 causes the recovered mark/space signals to have a phase ambiquity of 180 degrees; i.e., the recovered carriers can be either in phase with the input or 180 degrees out of phase with the input. In addition, either one or both may be inverted. If both are inverted, Equations (2-29) and (2-30) are inverted. This results in both the I and the Q symbols [Equations (2-33) and (2-34)] being complemented. This has no effect on the recovered data as shown by Equation (2-36). This also has no effect on the recovered clock as given by Equation (2-35).

However, it is possible for only *one* recovered carrier to be inverted. This results in the following changes in Equations (2-29) and (2-30). If only the mark carrier is inverted:

$$-\cos(\omega_m t) + \cos(\omega_s t) = 2\sin(\omega_o t)\sin\left(\frac{\pi t}{2T}\right)$$

$$\cos(\omega_s t) + \cos(\omega_n t) = 2\cos(\omega_o t)\cos\left(\frac{\pi t}{2T}\right)$$

Hence, the I and Q channels are interchanged. The $f_{ck}/2$ signal is also inverted and thus there is no net effect. The other possibility is for the space carrier to be inverted. Similar analysis shows that the I and Q channels change places *and* both are inverted (both the I data and Q data are complemented). The recovered $f_{ck}/2$ is also inverted and once again the recovered data is unaffected. In all cases, the NRZ bit clock, f_{ck}, is unaffected since it is twice the recorded $f_{ck}/2$ signal. Therefore, there are no ambiguities in the recovered NRZ data, even though there are some ambiguities in the recovered carriers. The I and Q channels can change roles (i.e., the designation of I and Q channels is arbitrary as long as the relative polarities are maintained) and the I *and* Q bits can be complemented, but the output data polarity is correctly maintained. This is not the case when codes other than NRZ are used and special precautions must be taken in these cases to maintain correct data and clock polarities.

A block diagram of the basic coherent demodulator described by the above equations is shown in Figure 2-11. The input signal is squared and used as a reference for mark/space carrier recovery phaselocked loops. The sum of the mark/space carrier is applied to the mixer

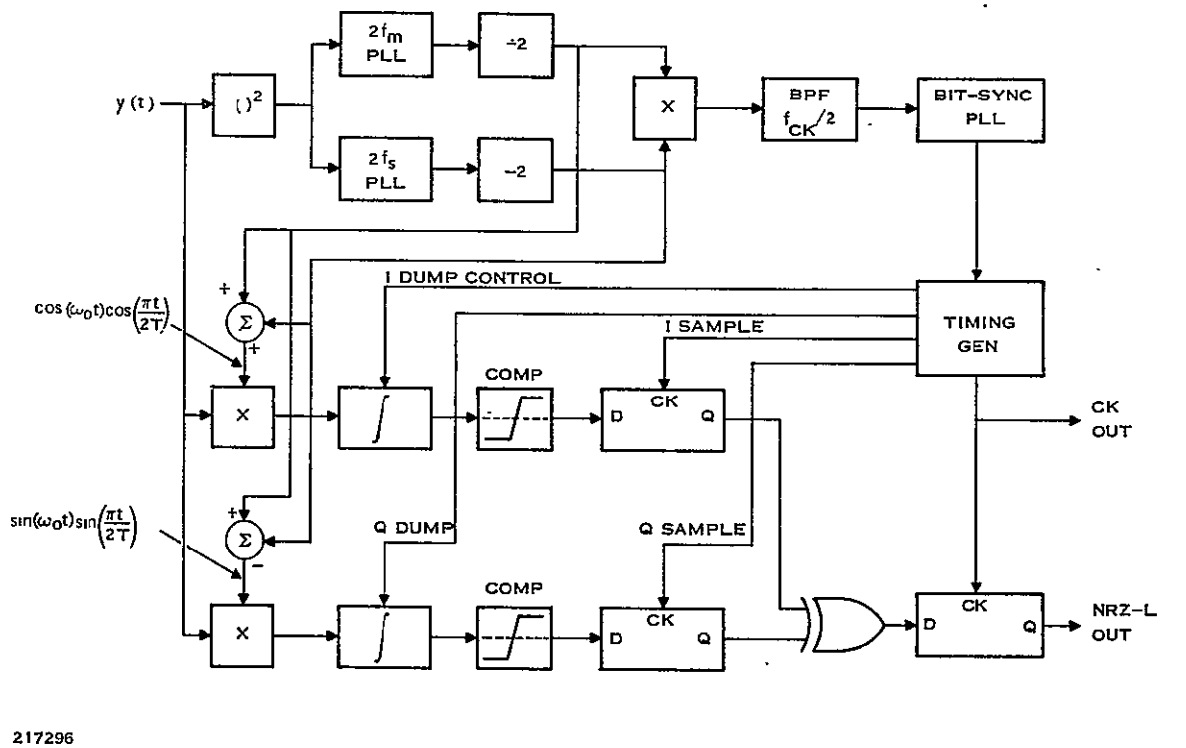

Figure 2-11. Simplified Block Diagram of Coherent Demodulator for MSK

(multiplier) for the I-channel demodulator; the difference is applied to the Q-channel demodulator. The MSK signal, y(t), is demodulated by the I and Q demodulators. The output of each demodulator is integrated for two bit periods, but the I and Q integrators are staggered or offset by one bit period. Following the relationships given by Equation (2-28), the I-channel is integrated over two bit times beginning and ending on the negative edge of N; the Q-channel is integrated over two bit times; beginning and ending on the positive edge of N. Note that this phasing must be maintained *relative* to the definitions of the I and Q signals; however, the designation of the I and Q channels is arbitrary. The designation of the I and Q channels by the polarity of the $f_{ck}/2$ clock at the transmitter can be arbitrary; either will be correctly demodulated and decoded if the phasing of the demodulator signals is properly chosen for a given designation.

The output of each integrator is either positive or negative. A threshold comparator selects either a logic one or a logic zero, depending on the polarity of the integrator output. At the end of each 2T integration period, the output of the comparator is sampled and stored in a one-bit register and the integrator is reset. The I and Q channel outputs are applied to an exclusive-OR gate that decodes the I and Q data into NRZ data. The NRZ data is buffered by an output register. The recovered clock frequency is synchronous with this data.

The above statement provides the basis for the coherent demodulator to be described in detail later. The mathematical relationships for MSK as a pseudo-PSK signal presented in this subsection are the theoretical foundation for the coherent demodulator. The performance advantages of the coherent demodulator are clarified in the following subsections.

2.2 BIT ERROR RATE VERSUS E_b/N_o FOR MSK

The primary advantage of MSK over CPFSK is the possibility of coherently demodulating the signal as a PSK signal. This advantage is the realization of a 3-dB improvement in E_b/N_o performance since PSK provides 3 dB better performance over CPFSK. It should be noted that all performance is relative to NRZ data.

The coherent demodulator described in the previous subsection is analogous to the frequency-doubling carrier-recovery coherent demodulator for antipodal PSK. A typical circuit [5] is shown in Figure 2-12. The input BPSK signal is represented as $A(t)\sin(\omega_o t)$ where $A(t) = \pm 1$ represents the binary data modulation. This corresponds to the binary I(t) and Q(t) data symbol modulation of the MSK phasors of Equation (2-24). The mixing (multiplication) operation results in:

$$2A(t)\sin^2(\omega_o t) = A(t)\,[1 - \cos(2\omega_o t)]$$

Subsequent integration removes the carrier and results in the integral of the DC term, T A(t). Note that this is precisely analogous to the T I(t) and T Q(t) values obtained for the coherent demodulator for MSK. (This results from the fact that the I and Q symbols can be integrated for *two* bit times; this compensates for the energy division between the I and Q phasors over a single-bit time.) The conclusion from this analysis is that the energy content of the recovered I and Q symbols for the coherent MSK demodulator is precisely the same as that for antipodal binary PSK, using the frequency-doubling carrier recovery loop. Therefore, the BER performance should be the same.

[5] James J. Spilker, Jr., *Digital Communications by Satellite*, Prentice-Hall, Inc. (New Jersey, 1977).

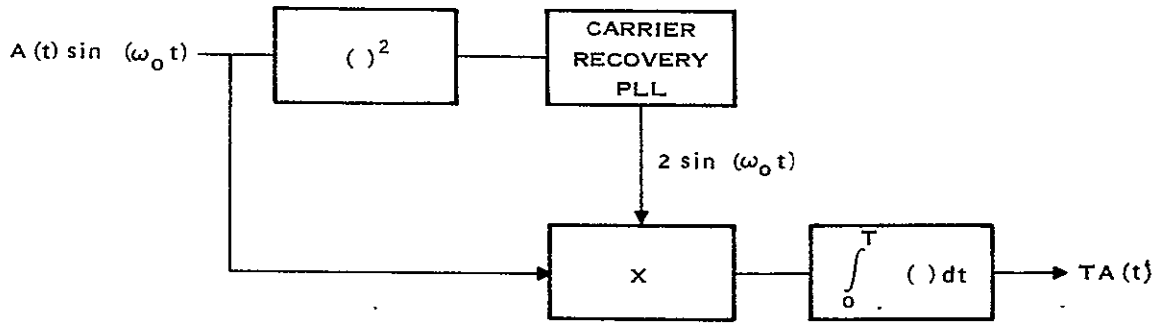

Figure 2-12. Simplified Block Diagram of a BPSK Demodulator

The BER performance would be exactly the same as BPSK, except for the decoding of the I and Q symbols to form binary NRZ-L data. The differential decoder causes errors to occur in *pairs;* this essentially doubles the BER performance of MSK with respect to BPSK. Therefore, the theoretical BER performance of MSK is twice that of BPSK. The theoretical BER performance of MSK as a function of E_b/N_o is shown in Figure 2-13.

2.3 POWER SPECTRAL DENSITY OF MSK

A key advantage of MSK, as the name implies, is its minimum bandwidth occupancy. A plot of the PSD for MSK/NRZ is shown in Figure 2-7. It is obvious that most of the energy is contained in a channel bandwidth equal to the data rate, R. Mathwich[6] has shown that in fact very little degradation in E_b/N_o performance occurs with a bandwidth of only 0.75R; the limiting case is approximately 0.55R, causing a loss of 1.15 dB in E_b/N_o performance. He has also shown that the effects of nonlinear phase (introduced by a channel filter) have little effect on link performance. This is comparable to the performance of OK-QPSK where transmit filtering is used to limit bandwidth occupancy.[7] However, for data collection systems, no transmit filter is to be used; hence, performance improvement of MSK over QPSK (or BPSK) is realized by the rapid decrease in the sidelobes for MSK. Futhermore, amplitude limiting can be used in a receiver without increasing the sidelobes beyond those shown in Figure 2-7.

A comparison of the normalized PSD of MSK and offset QPSK (SQPSK) is given in Figure 2-14.[8] Note the rapid decrease in the sidelobes of MSK. Although OK-QPSK decreases more rapidly out to 0.7R, a transmit filter is needed to take advantage of this roll-off. In addition, if the QPSK signal undergoes amplitude limiting after filtering, the sidelobes are restored.

The general equation for CPFSK used to produce Figure 2-7 is Equation (2-16). A simpler equation for MSK expressed in decibels relative to carrier power, P_c, is:

$$W_{msk}(f) = 10 \log \left[\frac{8P_cT\,(1 + \cos 4\pi fT)}{\pi^2(1 - 16\,T^2f^2)^2} \right] \qquad (2\text{-}37)$$

[6]H. Robert Mathwich, "The Effect of Tandem Band and Amplitude Limiting on the E_b/N_o Performance of Minimum (Frequency) Shift Keying (MSK)," *IEEE Transactions on Communications,* Vol. COM-22, No. 10, October 1974.

[7]Spilker.

[8]S.A. Gronemeyer and A.L. McBride, "Theory and Comparison of MSK and Offset QPSK Modulation Techniques Through a Satellite Channel," presented at the National Telecommunications Conference, December 1974, New Orleans, Louisiana.

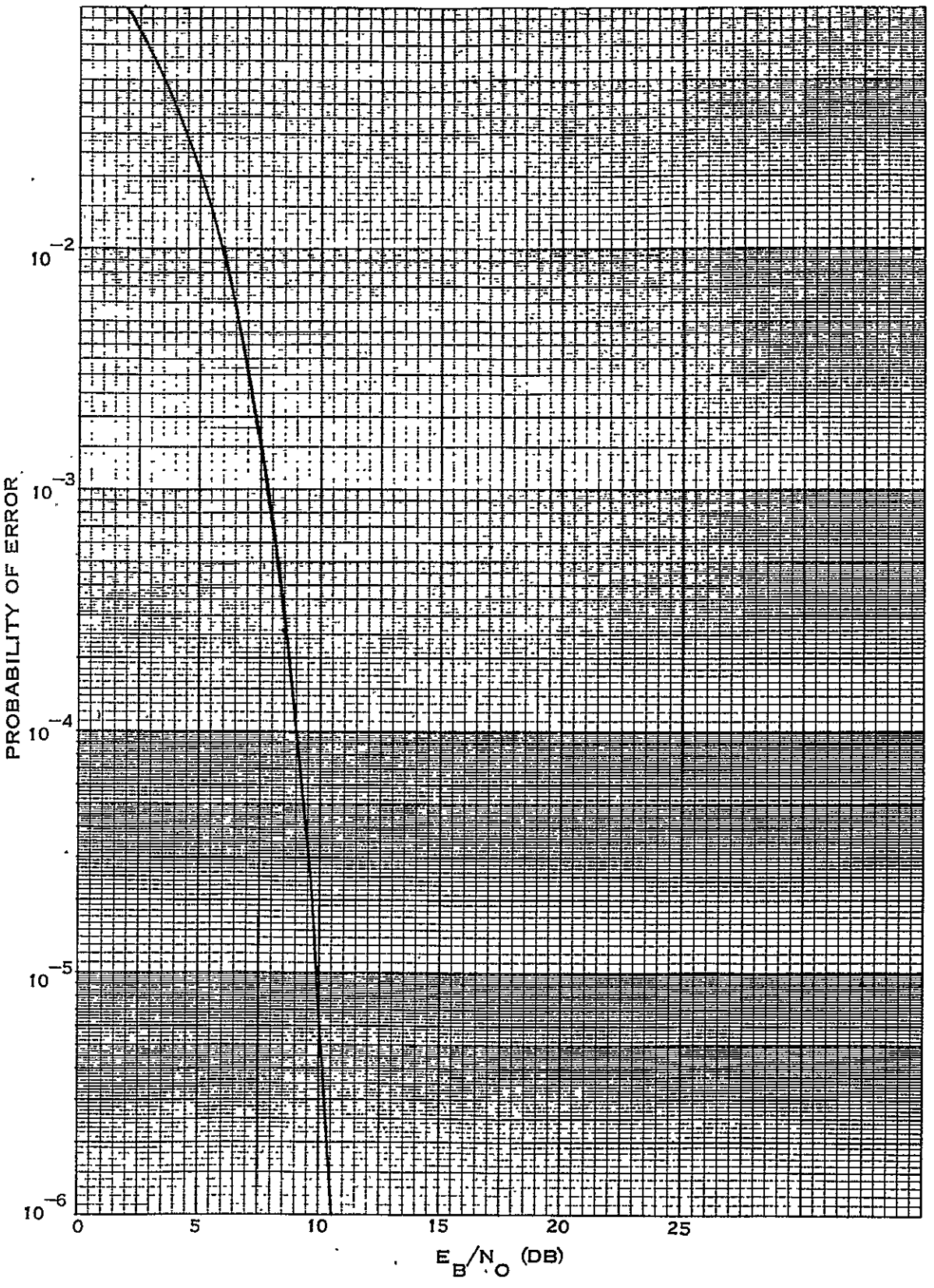

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Figure 2-13. Theoretical BER Performance of NRZ/MSK Data

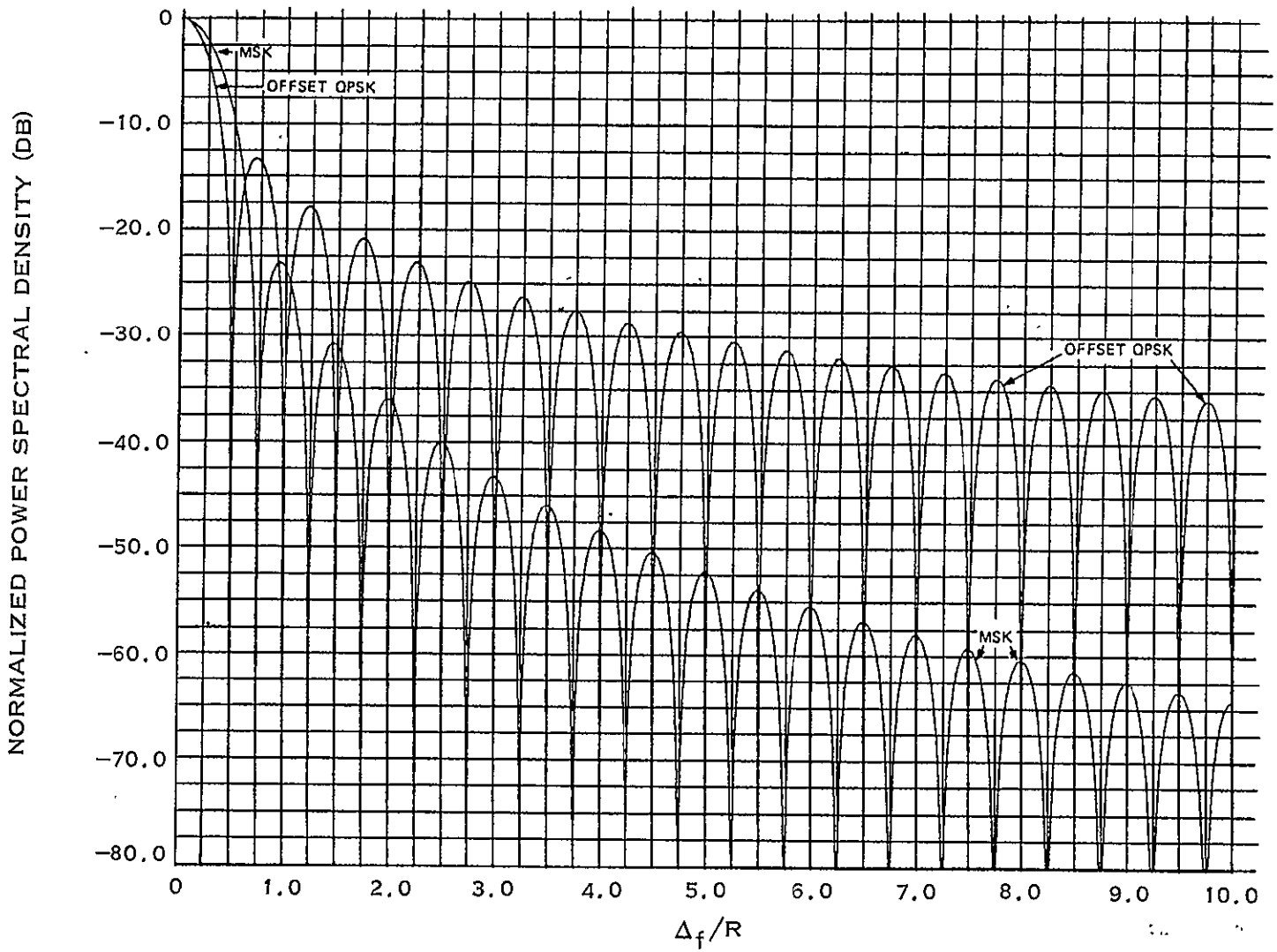

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Figure 2-14. Comparison of Power Spectral Density of MSK and Offset QPSK

A similar expression for SQPSK is:

$$W_{sqpsk}(f) = 10 \log \left[2P_c T \frac{\sin^2 (2\pi fT)}{(2\pi fT)^2} \right] \quad (2\text{-}38)$$

where

f = frequency offset from carrier

P_c = power in the modulated carrier

T = the bit period.

These equations were used to produce Figure 2-14.

All forms of PSK (BPSK, QPSK, SQPSK, etc.) have similar spectra. The spectrum of MSK is similar to all forms of CPFSK with two notable exceptions: (1) there are no discrete spectral lines and (2) the main data lobe occupies a minimum amount of bandwidth. These two features make MSK the best choice for maximizing the number of data collection transmitters in a given bandwidth while minimizing mutual interference.

2.4 CROSSTALK AND ADJACENT-CHANNEL INTERFERENCE FOR MSK

The key requirement for improving the dynamic range of data collection systems of the RAMS type is reduction of adjacent-channel crosstalk and mutual interference. This reduction is contingent on minimizing both the bandwidth of the main data lobe and the amplitude of the sidelobes. The use of MSK modulation significantly reduces the energy contained in the sidelobes while keeping the bandwidth of the mainlobe about the same as BPSK.

It has been shown that the amplitudes of MSK sidelobes decrease at the rate of 40 dB/decade of normalized frequency while the sidelobes of BPSK decrease at the rate of 20 dB/decade. Furthermore, the first pair of sidelobes of MSK are down 23 dB with respect to the peak, while, for PSK, they are down only 11 dB. Therefore, it is obvious that MSK will provide superior crosstalk performance, but the improvement needs to be quantized. Fortunately, this has been considered by McBride,[9] Kalet,[10] and White.[11]

Gronemeyer and McBride[12] used out-of-band power as a measure of potential interference from an adjacent channel. Their results are duplicated in Figure 2-15. This curve sets a bound on the maximum power available for interference and, therefore, indicates minimum dynamic range as a function of normalized frequency offset from an adjacent channel.

Kalet[13] has computed the mean-square crosstalk as a function of normalized frequency displacement for MSK. His equation is:

$$E_s(C^2) = 1/8 \left[\frac{A^2 (k^2 + 0.25)}{16\pi^2 k^2 (k^2 - 0.25)} \right] \quad (2\text{-}39)$$

[9] Gronemeyer and McBride.

[10] Irving Kalet, "A Look at Crosstalk in Quadrature-Carrier Modulation Systems," *IEEE Transactions on Communications*, Vol. Com-25, No. 9, September 1977.

[11] Brian E. White, "A Worst Case Crosstalk Comparison Among Several Modulation Schemes," *IEEE Transactions on Communications*, Vol. Com-25, No. 9, September 1977.

[12] Gronemeyer and McBride.

[13] Kalet.