

701-
1N-52-CR

**FINAL REPORT
PROJECT NAG 9-597**

17 CIT.

TO:

49815

P. 72

**NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
SPACE BIOMEDICAL SCIENCES**

"CO₂-O₂ INTERACTIONS IN EXTENSION OF TOLERANCE TO ACUTE HYPOXIA"

(Period 1 April 1992 to 31 March 1995)

C. J. Lambertsen

Report No. 4-20-95

Institute for Environmental Medicine
1 John Morgan Building
University of Pennsylvania Medical Center
Philadelphia, PA 19104-6068

20 April 1995

(NASA-CR-198575) CO2-02
INTERACTIONS IN EXTENSION OF
TOLERANCE TO ACUTE HYPOXIA Final
Report, 1 Apr. 1992 - 31 Mar. 1995
(Pennsylvania Univ. Medical Center)
72 p

N95-29158

Unclass

G3/52 0049815

CONTENTS

- 1.0 OVERVIEW - RELATIONS OF CARBON DIOXIDE TO ATMOSPHERIC HYPOXIA AND HYPEROXIA
- 2.0 INTRODUCTION
- 3.0 OBJECTIVES OF PROJECT - HYPOXIA-CO₂ INTERACTIONS
- 4.0 PHYSIOLOGICAL BACKGROUND OF CO₂-HYPOXIA INTERACTIONS IN REST AND WORK (Relations to Acute and Sustained Adaptations)
 - 4.1 Relations to Previous Work: CO₂-Hypoxia Interactions in Brain Blood Flow Control
 - 4.2 Relations to Performance Functions
 - 4.3 CO₂-O₂ Interactions in Severe States of Hypoxia, Hypercapnia, and Physical Work
- 5.0 ORIGINAL HYPOTHESES
- 6.0 SIGNIFICANCE
- 7.0 SCOPE OF EXPERIMENT PROJECTS PERFORMED - Results During Four Phases of This Report Period
 - 7.1 Phase I - Methods and Procedures Development and Validation
 - 7.1.1 Development of Laboratory Underwater Ergometry System for Controlled Submerged Exercise
 - 7.1.2 Immersed Arterial Blood Sampling
 - 7.1.3 Establishment of Quantitative, On-Line Transcranial Doppler Method as Continuous Index of Brain Blood Flow
 - 7.1.4 Transcranial Doppler Blood Flow Measurement in Immersion
 - 7.2 Phase II - Effects of Partial and Total Immersion
 - 7.2.1 Effects of Partial and Total Immersion on Physiological Responses to Incremental Exercise (Breathing Air at 1.0 Atmosphere)
 - 7.2.2 Effects of Partial and Total Immersion on Physiological Responses to Progressive Hypercapnia (Breathing Oxygen at 1.0 Atmosphere)
 - 7.3 Phase III - Evaluation of Specific Mental Function Tests as Sensitive Indices of Hypoxic Effect, at Rest and in Exercise
 - 7.3.1 Test Evaluation and Performance Measurement System

- 7.3.2 Technical Descriptions of Six Mental Function Tests Evaluated for Use in Exposures to 10% O₂ Alone and (10% O₂ with CO₂)
 - 7.3.3 Effects of Breathing 10% Oxygen for 60 Minutes on Six Selected Tests of Mental Function (Inexperienced Subjects)
 - 7.4 Phase IV - CO₂ Effects During Hypoxia
 - 7.4.1 Effects of Breathing 10% Oxygen for 60 Minutes on Physiological and Mental Functions (Experienced Subjects)
 - 7.4.2 Contrast of Hypoxia Alone and Hypoxia with Restored Normocapnia, on Mental Performance and Physiological Responses
- 8.0 SUMMARY - SCIENTIFIC RELEVANCE OF RESULTS AND IMPACT ON FUTURE RESEARCH PLANS
- 9.0 PERSPECTIVE FOR FUTURE RESEARCH - Relevance of Carbon Dioxide/Hypoxia Interactions to NASA Biomedical Research Program and Operations
- 10.0 REFERENCES
- 11.0 REPORTS AND PUBLICATIONS—Present Grant Period
- 12.0 APPENDIXES
 - A. PERFORMANCE MEASUREMENT SYSTEM (BOUND SEPARATELY)
 - B. FIGURES. INDEX OF FIGURES

1.0 OVERVIEW - RELATIONS OF CARBON DIOXIDE TO ATMOSPHERIC HYPOXIA AND HYPEROXIA

Oxygen will always be the most critical life substance for human consciousness, mental capability, physical activity, and many physiologic support functions in space and underwater operations. Carbon dioxide is the critical agent linking respiration to the needs of oxidative metabolism. This continuing dual program has involved investigation of excessive oxygen pressure, and inadequate oxygen pressures.

Breathing high partial pressures of oxygen (hyperoxia), as for diving or therapy of aerospace decompression sickness, generates toxic effects upon organs and tissues. One component of the present oxygen research program has therefore been the measurement of rates of development of, and recovery from, toxic effects of oxygen upon specific functions of blood, lung, brain, visual systems, auditory and vestibular system, respiratory control, and cardiovascular system. These quantitative measurements in normal human subjects guide the use of oxygen in hyperoxic therapy, and determine its usefulness in new diving methods which allow prolonged underwater work by astronauts, using neutral buoyancy for training in the simulated weightless state. Since optimal use of oxygen is the key to effectiveness and safety in diving and decompression, determining the limits of human oxygen tolerance establishes the limits for improvement in safety across the full range of its usefulness (19,23,25).

In conditions of accidental or intentional exposure to lower than normal pressure of oxygen (hypoxia), the lowered oxygen supply to brain can result in partial or complete losses of mental and physical capability. The second major component of the dual oxygen research program is therefore the measurement of human tolerance to increasing degrees of sudden (acute) hypoxic stress. Measurements are made of effects on respiratory functions, arterial blood gases, physical exercise capability, and control of brain circulation, to the limits of consciousness. For both hyperoxia and hypoxia programs, measurements include calibrated on-line monitoring of blood flow rate through a major brain blood vessel, to allow calculation of changes in the brain oxygen flow and partial pressures.

The purposes of these two interlocking programs, involving toxic effects of excess oxygen pressure on the one extreme, and organ system failures in oxygen deficiency on the other, are to obtain the baselines for investigating the roles of carbon dioxide in modifying these different effects, at rest and at work, over the full range of useful or tolerable oxygen pressures.

The results accumulated over a many-year period have explained why exposures to an increase in atmospheric carbon dioxide can accelerate development of the convulsions produced by brain oxygen poisoning. The accelerated toxicity is due to the increase in brain blood (oxygen) flow produced by carbon dioxide dilation of brain blood vessels.

In hypoxia, carbon dioxide can also increase brain blood flow and oxygenation, by dilating brain blood vessels. This increased blood (oxygen) flow provides an acute, beneficial adaptation to otherwise intolerable degrees of hypoxia. The present stage of the hypoxia program concerns the

effectiveness of carbon dioxide-induced acute physiologic adaptations to hypoxia in maintenance of critical mental and psychomotor capabilities.

2.0 INTRODUCTION

The accomplishments described in this report are part of a continuing broad Program of the Institute for Environmental Medicine, concerned with potential advantageous and detrimental influences of deviations from natural respiratory environments. The overall NASA-related Program emphasizes human physical, physiological and mental activity in two extremes of physiologically related environmental oxygen stress (Fig. I-1).

One component, performed under other (USN) support, relates to interacting effects of carbon dioxide and hyperoxia in Underwater and Hyperbaric O₂ Therapy activities.

The component under this NASA Project relates to interacting effects of carbon dioxide and hypoxia (pertinent to aerospace adaptations in short and long duration operational and emergency atmospheres) (22).

The research therefore encompasses the extremes of tolerance to high and low oxygen partial pressures, in order to expand understanding of the basic physiological mechanisms affected by each (Fig. I-1). Biomedical implications in each component of the Program (hypoxia and hyperoxia) concern interrelations of respiratory control systems with arterial oxygenation, brain circulation, brain oxygenation and cognitive/psychomotor performance, and physical performance.

In hyperoxic exposures, an increase in arterial carbon dioxide pressure (hypercapnia) can accelerate, or a decrease (hypocapnia) can delay onset of the convulsions and unconsciousness of oxygen poisoning (25). In hypoxic exposures, an increase in arterial carbon dioxide pressure can sustain brain oxygenation and mental performance, or a decrease (hypocapnia) can induce hypoxic unconsciousness due to decreased brain blood (oxygen) flow and exaggerated fall in brain PO₂ (10,16,30).

The two different states of oxygenation and patterns of effect on central nervous system functions are linked physiologically by the fundamental influences of carbon dioxide (i.e., hydrogen ion activity) on neural sensors of respiratory control and on the smooth muscle cells of brain arterioles. Between the pathophysiologic effects of oxygen poisoning and the metabolic disruptions of hypoxia (40) the graded benign physiological influences of low and high oxygen pressures exist during important extremes of human activity environmental exposures.

Acute interactions of carbon dioxide with hypoxia and hyperoxia have been studied in resting states (2,27,29,30,36). They have not been well enough defined for either hypoxia or hyperoxia over the range of useful exercise tolerance, in which oxygen demand and carbon dioxide production are increased. However, the parallel investigations of graded hypoxia and hyperoxic environments within the present Program are providing quantitative predictive

descriptions of the physiological linkages relating to alterations of the internal respiratory environments, produced in adaptations to change in external atmospheres. The returns to each Program component are magnified by the other.

3.0 OBJECTIVES OF PROJECT - HYPOXIA-CO₂ INTERACTIONS

The Scope of Information desired included (a) physiological and performance consequences of exposures to simulated microgravity, in rest and graded physical activity, (b) separate influences of graded degrees of atmospheric hypercapnia and hypoxia, and (c) composite effects of hypoxia and hypercapnia.

The research Objectives were selected for close relevance to existing quantitative information concerning interactions of hypercapnia and hypoxia on respiratory and brain circulatory control. They include:

- To determine influences of normoxic immersion on interrelations of pulmonary ventilation, arterial PCO₂ and PO₂, and brain blood flow, in rest and physical work.
- To determine influence of normoxic immersion on respiratory reactivity to atmospheric hypercapnia at rest.
- To determine influence of atmospheric hypoxia on respiratory reactivity to hypercapnia at rest and in work.
- To provide physiological baselines of data concerning adaptations in acute exposures, to aid in investigation of rates of adaptation or deteriorations in physiological or performance capability during subsequent multi-day exposures.

4.0 PHYSIOLOGICAL BACKGROUND OF CO₂-HYPOXIA INTERACTIONS IN REST AND WORK (Relations to Acute and Sustained Adaptations)

The continuous linkage between the internal respiratory environment of metabolizing brain tissues and the external respiratory environments is precisely quantitative (in open or closed atmosphere systems). This linkage is largely by way of active influences of carbon dioxide and oxygen levels upon localized neural cells of respiratory control and smooth muscle cells of local vascular control.

Normally the authoritative role in regulating both the oxygenation and the level of tissue hydrogen ion activity, essential for the biological burning represented by our metabolism, belongs to carbon dioxide through its multiple effects. Oxygenation in the normal sea level environment follows adequately, but essentially passively, the events dictated by CO₂ (18). The fine adjustment of natural pulmonary function and respiration at rest (and evidently in physical work) are regulated by neural responses to local internal acid-base states. This function is not so much to efficiently eliminate metabolically produced carbon dioxide as

to do so with an inefficiency designed to assure maintenance of the essential elevated degree of carbon dioxide pressure in the internal environment (18).

In acute atmospheric hypoxia, the power of peripheral chemoreflex stimulation of respiratory central neural mechanisms lowers arterial carbon dioxide pressure, detracting in part from the cited peripheral chemoreflex respiratory drive (15,41,42,44,46). The quantitative interplay of these CO₂/O₂- related effects modifies the degree of hypoxia within the entire internal respiratory environment and the cell functions it supports.

4.1 Relations to Previous Work: CO₂-Hypoxia Interactions in Brain Blood Flow Control

The powerful importance of carbon dioxide in internal environmental control for the brain is via its influence upon the tone of brain arterial vessels (23,30,36,39). In an acute exposure to a hypoxic atmosphere, accidental or intentional, the resulting reduction in oxygen supply to the brain can produce impairment of mental and physical function, or loss of consciousness. The degree of functional impairment is only in part related to the degree of arterial hypoxemia. It is acutely also related to the degree of arterial hypocapnia resulting from the chemoreflex ventilatory stimulation which occurs in hypoxia (23,26). This hypoxic hyperventilation and its advantageous influence on alveolar PO₂ partially counters the lowered alveolar and arterial oxygen pressure (16,37a,46). However, the hypoxia-induced cerebral vasodilation and increased brain blood flow is partially limited or even overcome by the cerebral vasoconstrictor influence of hypocapnia (23,26). The lowered carbon dioxide pressure of arterial blood during acute exposure to hypoxic environments therefore can diminish central nervous system tolerance to atmospheric hypoxia, as well as disturbing the central neural acid-base environment. For such reasons, in human subjects a prevention of the prominent arterial hypocapnia associated with administration of hypoxic gas (e.g., 8% O₂ at 1.0 ATA, or during 100% O₂ breathing at 40,000 feet altitude), provides the brain with a better oxygen supply than it has when subnormal O₂ is inspired without preventing arterial hypocapnia (16,30).

4.2 Relations to Performance Functions

Interacting influences of hypoxia and CO₂ on ventilation, blood gas transport, brain blood flow, and brain metabolism have been described, as has study of visual and mental performance in hypoxic states (7,33,34,37,43,45). However, relatively little is known about concurrent effects of these important interactions of CO₂ and hypoxia on mental and psychomotor performance or on the quality of visual and other perceptual functions in physical exercise. Mental competence, neuromuscular coordination, and vision are critical functions in responding to a life-threatening emergency, or in maintaining the precise operational skills required for manned space operations. This Project hypothesizes maintenance of these critical functions during hypoxic work, by reversal of hypocapnia to sustain cerebral oxygenation. This effect may conceivably be modified during exposure to microgravity by central pooling of blood and other circulatory alterations. Simulation of

microgravity by immersion in water was therefore included in the Program to provide an initial step in evaluation of this possibility (9,28), at rest and at work.

4.3 CO₂-O₂ Interactions in Severe States of Hypoxia, Hypercapnia, and Physical Work

During any accidental or purposeful exposure to hypoxia, an ability to perform precise mental and psychomotor functions will be desirable or required during brief or extended periods of physical work, as well as in rest states. It was considered that the increased rates of whole body oxygen consumption and carbon dioxide production caused by physical work will decrease tolerance to hypoxemia alone (38,45) and could limit the beneficial effects of arterial PCO₂ elevation on brain oxygenation, mental, psychomotor, and visual functions that were hypothesized for hypoxic exposures at rest.

5.0 ORIGINAL HYPOTHESES

The originating Program hypothesis was that a tolerable increase in atmospheric carbon dioxide pressure during acute atmospheric hypoxia will accelerate adaptation and improve mental and physical performance, at rest and in work. It was desired to relate the concept also to immersion. Hypotheses were therefore extended to include:

- Respiratory (and brain vascular) reactivity to increased atmospheric carbon dioxide partial pressure during immersion microgravity simulation will not be different than in exposures to normal dry environment.
- Tolerable exposure to increased inspiratory hypercapnia will improve cognitive/psychomotor performance during atmospheric hypoxia in rest and physical work.
- Improvement in performance will result from CO₂-induced combined increases in respiration, arterial oxygenation, and brain blood O₂ flow.

6.0 SIGNIFICANCE

The special significance of the present research has been its contributions to a Program specifically concerned with interacting influences of non-inert respiratory atmospheric components in rest and in work, in acute and transient exposure, and in longer term exposures.

Especially significant from practical operational standpoints is that, different from effects of slow climbing to increased altitude, the spacecraft, module or station can experience abrupt accidental or intentional, slight or drastic changes in the contained atmosphere. The scope of potential accidental or intentional changes will increase with mission duration, and can involve loss of pressure, loss of inert gas, loss of oxygen, or defect in carbon dioxide

removal. Pressure reduction then becomes a means of reducing rate of gas loss or gas toxicity.

Significance related directly to physiologic responses to CO₂-Hypoxia interactions is that no exposure to atmospheric hypoxia, of either mild or severe degree, produces its measurable effects except in conjunction with parallel effects of intrinsic changes produced by carbon dioxide (16). Further special physiologic significance relates to the interlocked neurochemical central and peripheral chemoreflex System of Respiratory Control, affected in acute and in longer exposures to atmospheric changes. The System itself is constant, but alteration of the pressure of either or both oxygen or carbon dioxide results in immediate and reproducible shifts in control system equilibrium, modifying circulation and other systems affecting internal environments of brain and other critical organs.

Atmospheric control and adjustment for routine and contingency activity in prolonged space operations are more easily provided through engineering than are most other environmental requirements. They are also most critical, day-by-day, for ultimate long-term success. The many decades of expert physiological investigations of aviation, mountaineering and laboratory exposure to altered respiratory environments have provided much of the conceptual understanding and information concerning limits of tolerance for periods of minutes, days or weeks (15,16,38,42). Development of predictive capability for success in multi-month or multi-year exposures in space requires progressive accumulation of such data adaptable to predictive modeling of contingencies relating human and equipment function. The present Program is providing its organized data to the Environmental Biomedical Research Data Center (NASA NAGW-3628) for potential use in such predictive modeling (20).

7.0 SCOPE OF EXPERIMENT PROJECTS PERFORMED - Results During Four Phases of This Report Period

7.1 Phase I - Methods and Procedures Development and Validation

Work objectives centered upon study of interactions of physiologic responses to acute exposures to physical work, and to altered respiratory environment. These experiments therefore required simultaneous measurements of relevant facts, with special ability to measure rates of change, as well as "stable" dose-effect relationships.

Immersion was used as a partial simulation condition for null gravity. It is also the natural environment in Naval and NASA diving, and has been cited as contributory to development of convulsions in hyperoxic exposures (3,6,47). Special methods developments were required for the performance of physiologic studies in immersion and dry states. Pre-existing capability included essentially breath-by-breath measurement of respiratory ventilation functions, end-tidal gas composition, metabolic gas exchange, arterial % hemoglobin saturation (pulse oximeter), and physical work level.

Introduction of new required methods involved development and pre-experiment validation. Major Sub-Projects therefore encompassed:

7.1.1 Development of Laboratory Underwater Ergometry System for Controlled Submerged Exercise

An underwater work system was developed to measure physiological responses to equivalent exercise workloads under dry and immersed conditions (Fig. III-1). The system was calibrated in eight subjects by measuring oxygen uptake at underwater workloads of 30, 50, 75, 100, 125, and 150 watts and dry workloads of 30, 50, 100, 150, and 200 watts. Calculated regression lines fitted to oxygen uptake values obtained under wet and dry exercise conditions had nearly identical slopes, with the wet values about 0.550 L/min higher than the dry values at the same ergometer workload settings (Fig. III-2). The influence of pedal rate on oxygen uptake at equivalent workloads under wet and dry conditions was also evaluated in eight subjects (III-3). As expected, the change in oxygen uptake for different pedal rates underwater was much steeper than that found under dry conditions. A pedal rate control system with an underwater speaker that emitted low or high tones when pedal rate was too low or too high, respectively, was used to provide feedback that helped the subject to maintain a constant pedal rate/work rate underwater.

7.1.2 Immersed Arterial Blood Sampling

The requirement for obtaining arterial blood samples from an immersed subject imposed precautions to avoid contamination of the puncture wound over and in the radial artery. The skin puncture site was covered with antibiotic ointment prior to sealing the area with a large transparent Tegaderm dressing. Waterproofing of the puncture site was further assured by covering the periphery of the Tegaderm patch and adjacent skin with a layer of liquid silicone rubber.

7.1.3 Establishment of Quantitative, On-Line Transcranial Doppler Method as Continuous Index of Brain Blood Flow

Transcranial Doppler measurements of middle cerebral arterial (MCA) flow velocity were calibrated against simultaneous measurements of cerebral blood flow (CBF) perfusion rate by clearance of radioactive xenon (Fig. II-1). Eight subjects were studied over an arterial PCO₂ range of 25 to 50 mm Hg while breathing oxygen as the background gas. Relative to control values measured while breathing 100% O₂, percent changes in CBF at increased levels of arterial PCO₂ corresponded to changes in MCA velocity in a ratio of 2:1 (Fig. II-2). These relative changes are consistent with MCA vasodilation during exposure to hypercapnia.

7.1.4 Transcranial Doppler Blood Flow Measurement in Immersion

Transcranial Doppler (TCD) ultrasonography is a relatively new technique that was developed initially for use in patients with neurological diseases (1). Prior to the current application, it had not been used for underwater measurements. Problems were encountered related both to stabilization of the TCD probe and to the elimination of artifacts caused in immersion by multi-directional transmission of Doppler signals.

Stable orientation of the TCD probe with respect to the middle cerebral artery must be maintained at rest and during exercise throughout an experiment to obtain accurate measurements of blood flow velocity. The platform supplied by the manufacturer for long-term attachment of the TCD probe to an appropriate cranial "window" did not provide adequate stability during exercise even in a dry environment. This problem was solved by the design and fabrication of a new platform that could be attached to the subject's head with the aid of Stomahesive and double-backed tape. The new platform also provided a mechanism for clamping the TCD probe to maintain a constant angle of insonation with the middle cerebral artery. The softening of Stomahesive underwater occurred slowly enough that probe stability could easily be maintained for the duration of an experiment.

Multi-directional transmission of Doppler signals underwater caused problems that do not exist in an air background. Artifacts related to head or wave movements appeared as bursts of noise that distorted or obliterated the waveform of the blood velocity signal. Head movements were minimized by positioning the subject to provide mechanical stability that facilitated the performance of ergometer exercise without extraneous body movements. Measures were taken to reduce artifacts due to wave action during exercise.

7.2 Phase II - Effects of Partial and Total Immersion

Specific interests in immersion concerned effects of immersion on respiration and brain blood flow, respiratory and brain circulatory reactivity to CO₂, and responses to exercise.

7.2.1 Effects of Partial and Total Immersion on Physiological Responses to Incremental Exercise (Breathing Air at 1.0 Atmosphere)

Eight subjects were studied during incremental exercise under dry conditions, then during head-out immersion, and during total immersion. Water temperature was maintained at 32°C. Air was breathed rather than oxygen to facilitate measurements of oxygen uptake during exercise. During exposure to each condition, a 10-minute rest period was followed by three consecutive 6-minute workloads consisting of 60, 110, and 150 watts at a pedal rate of 60 rpm in the dry and 30, 80, and 110 watts at 50 rpm during immersion. Concurrent measurements at each workload included end-tidal PCO₂, MCA (middle cerebral artery) blood flow velocity, ventilation rate, tidal volume, respiratory rate, heart rate, and rates of O₂ uptake and CO₂ elimination.

Average ventilatory responses to incremental exercise were nearly identical under dry conditions and in both immersion states (Fig. IV-2). Average end-tidal PCO₂ values at rest and during incremental exercise for all three conditions are shown in Fig. IV-3. None of the differences in end-tidal PCO₂ values at equivalent workloads were statistically significant by analysis of variance across all workloads and all three conditions.

Average MCA blood flow velocities in all three conditions increased significantly by about 8 cm/sec on the average during the transition from rest to exercise, but remained essentially constant with further increments in workload (Fig. IV-1). There were no significant differences in MCA blood flow velocities measured under dry conditions or in either immersion state.

7.2.2 Effects of Partial and Total Immersion on Physiological Responses to Progressive Hypercapnia (Breathing Oxygen at 1.0 Atmosphere)

Eight subjects were studied at rest under dry conditions, then during head-out immersion, and finally during total immersion. Water temperature was maintained at 35°C. In each condition, the subject first breathed air, then 100% O₂, followed by consecutive periods of CO₂ administration at controlled end-tidal PCO₂ levels of 45, 50, and 55 mm Hg. Concurrent measurements on each gas included arterial PCO₂, PO₂, and pH, body temperature, MCA blood flow velocity, ventilation rate, tidal volume, respiratory rate, heart rate, and mean arterial blood pressure.

Average ventilatory responses to progressive hypercapnia were higher under dry conditions than in both immersion states, but the differences were small and not statistically significant (Fig. V-2). Neither head-out nor total immersion had a measurable effect on ventilatory responses to hypercapnia at rest.

Relationships of average body temperature to increasing arterial PCO₂ showed only small, but consistent, differences in the dry and during immersion. Hypercapnia-induced peripheral vasodilation with increased heat loss to the 25°C ambient air atmosphere probably accounted for the observed 0.4°C fall in body temperature under dry conditions. During head-out and total immersion in 35°C water, body temperature remained stable during exposure to hypercapnia. The observation that average body temperature was about 0.3°C higher during total than during head-out immersion probably reflects the fact that these measurements were obtained after an additional 90 minutes of immersion in warm water.

Average values for mean arterial blood pressure were consistently lower in both immersion states than under dry conditions at comparable levels of arterial PCO₂ (Fig. V-4). The lower arterial blood pressure and increased heart rate observed during immersion are consistent with a mild hypotensive response to heat-induced cutaneous vasodilation in 35°C water and a related reflex increase in heart rate. Mean blood pressure rose progressively in response to increasing arterial PCO₂ with essentially equal slopes in all three conditions, but average values during immersion were significantly lower by about 7-8 mm Hg.

Average heart rate also rose progressively in response to increasing arterial PCO₂ with nearly equal slopes in all three conditions (Fig. V-3). Average values in both immersion states were nearly identical during exposure to hypercapnia, and both curves were significantly higher by about 9-10 beats/minute than average values obtained under dry conditions at the same levels of arterial PCO₂. The nearly linear increments in heart rate and mean arterial blood pressure with increasing arterial PCO₂ probably reflect hypercapnia-induced sympathetic stimulation which may be modified to some extent by the superimposed vagal cardiac action of hyperoxia.

The average MCA blood flow velocity (Brain Blood Flow Index) responses to increasing arterial PCO₂ were nearly identical under dry conditions and in both immersion states (V-1). These results were obtained under resting conditions with virtually no Doppler signal interference related to immersion of the Doppler probe. The observed data indicate that cerebral circulatory responses to hypercapnia at rest are not significantly influenced by either head-out or total immersion.

7.3 Phase III - Evaluation of Specific Mental Function Tests as Sensitive Indices of Hypoxic Effect, at Rest and in Exercise

Effects of exercise and/or hypercapnia during acute exposures to atmospheric hypoxia depend upon the degree and duration of the hypoxia.

An intent of the Program therefore has to be to establish sustained decrements in hypoxia-sensitive mental functions, in order then to investigate effects of superimposed hypercapnia and/or exercise. Such ancillary effects must be interpreted in relation to degree and duration of hypoxic exposure. Therefore an initial step in the Program required selecting atmospheric O₂ concentrations tolerably detrimental over the desired hour-long exposure period.

Prior experiments showed that .08 ATA O₂ inspired at rest for periods of 20 to 30 minutes induced significant decreases in brain O₂ consumption, which was largely reversible by preventing arterial hypocapnia (16,23). Exposure to .10 ATA O₂ causes only small decrements in brain O₂ consumption of resting subjects (13).

7.3.1 Test Evaluation and Performance Measurement System

In the present Program, the search for sensitive measures of hypoxic effects on mental function was greatly facilitated by the availability of the Performance Measurement System, that was designed, constructed, and evaluated at the Institute for Environmental Medicine (Appendix A). The Appendix contains descriptions of Tests and System.

Exposures to .12 ATA O₂ and 10% in N₂ were used initially to explore capability of graded uninterrupted physical work of 50 and 100 watts, while responding to mental and psychomotor function tests. Significant changes in psychomotor functions were found with

10% O₂, but not found with 12% O₂ in continuous rest/work exposure (Annual Report 1993) (21).

Specific tests evaluated with 10% O₂ during and following studies of 12% O₂ exposures were a Time Reproduction Test of time estimation ability, a Visual Digit Span Test of span memory ability, a Choice Reaction Time Test of response orientation ability, and an Operations Test of numeric reasoning ability. These tests were selected for evaluation by the following criteria: (a) a level of difficulty such that a stable performance plateau could be readily achieved within a reasonable training period; (b) a wide range of possible scores achievable within an administration time of less than 5 minutes; (c) randomly presented test items having uniform degrees of difficulty; (d) sensitivity of test scores to hypoxia sufficient that significant decrements occur consistently during exposure to inspired oxygen concentrations of 10% or higher.

Effects of exposure to 10% O₂ (0.1 ATA O₂) for up to 60 minute on the four selected tests in five trained normal men are summarized in the prior Annual Report (21). Both the choice reaction time and the operations test were significantly reduced by 19.6% and 19.0%, respectively. However, these observed decrements were relatively small in magnitude, and therefore not satisfactory for assessing the capacity for restoring function by carbon dioxide administration.

Accordingly, effects of hypoxia on six additional tests of mental function were evaluated for potential larger sensitivity to hypoxia.

7.3.2 Technical Descriptions of Six Mental Function Tests Evaluated for Use in Exposures to 10% O₂ Alone and (10% O₂ with CO₂)

The simpler tests evaluated initially were studied in subjects trained to baseline plateaus in the technical performance of these tests. More complex tests were selected for this next phase of investigation, and the effects of hypoxic exposure on recently learned skills were also evaluated. The selected Performance Measurement System Tests and mental functions involved are briefly as follows:

Word-Number Test of Associative Memory Ability. The subject is given one minute to memorize a video monitor display of six vertically oriented side-by-side word-number pairs (Appendix A). This display is then replaced by another with the same words in a different (scrambled) order, without the numbers. The response is to indicate (with the stylus) the two digits remembered as paired with each word, working from top to bottom of the display. The complete test consists of two six-paired displays, with the principal score the number of correct responses (24 maximum).

Letter Order Test of Inductive Reasoning Ability. The object is find the rule that makes four of five letter groups (identified with the digits "1" to "5") alike in some way, and to report which one does not fit the rule. The response is to put the stylus into the

appropriate cell "1" through "5". The test lasts four minutes, and the principal score is the number of correct responses minus one fourth the number of incorrect responses.

Surface Development Test of Visualization Ability. An isometric drawing of a three dimensional object with eight edges numbered "1" through "8" and a principal side identified with the letter "X" is displayed to the right of the same object shown folded out in two dimensions, with the same edges identified by the letters "A" through "H" and the side "X" identified. The response is to match each lettered edge on the foldout with its corresponding numbered edge on the isometric drawing by placing the stylus in the appropriate cell "1" through "8". The test lasts three minutes, and the principal score is the number of correct responses minus one seventh the number of incorrect responses.

Choosing-a-Path Test, of Spatial Scanning Ability. The display shows five boxes side-by-side. Each box has indicated start (S) and finish (F) points along with a maze of intersecting lines. Small closed circles indicate those intersections where turns can be made. One large open circle must be included in the single complete path originating at the "S" in a box and terminating at the "F" in the same box. Each box is identified with a digit ("1" through "5"). The response is to indicate the box that has a complete path from "S" to "F" by placing the stylus in the appropriate cell "1" through "5". The test lasts four minutes, and the principal score is the number of correct responses minus one fourth the number of incorrect responses.

Multiplication (or Division) Test, of Numerical Ability. A multiplication (or division) problem is displayed. The response is to indicate the correct answer to the arithmetical operation by placing the stylus sequentially into the appropriate cells numbered "0" through "9", and then indicating that the answer is complete by placing the stylus into the "star" cell. The test lasts two minutes, and the principal score is the number of correct digits in the 2- or 3-digit solutions to the randomly presented problems.

7.3.3 Effects of Breathing 10% Oxygen for 60 Minutes on Six Selected Tests of Mental Function (Inexperienced Subjects)

Previous studies (5,8,12) have indicated that recently learned information may be more sensitive than well established skills, to impairment by hypoxia. In order to determine whether any of the six selected tests were unusually sensitive to hypoxia in inexperienced subjects, they were initially screened in eight subjects who were taught how to perform each test, but had not achieved a stable learning plateau. On each of two different days at least two days apart, each subject performed three mental function tests under normoxic control conditions and then repeated the tests starting at 35, 45, and 55 minutes of a 60-minute exposure to 10% O₂. The Word-Number, Choosing-a-Path, and Multiplication Tests were administered in that order on the first day. On the second day, the test sequence consisted of the Letter Order, Surface Development, and Division Tests. Because performance of each test during exposure to hypoxia always followed testing under normoxic conditions, any learning that occurred with repetition of the same test would reduce rather than increase its apparent sensitivity to hypoxia.

Average scores for each of the six selected tests under normoxic and hypoxic conditions are summarized in Fig. VI-1. None of the tests provided an exceptionally sensitive indication of hypoxia in the eight relatively inexperienced subjects. Average scores were reduced during exposure to hypoxia for all but the Letter Order Test. Distinct decrements of performance were found for several tests. The observed decrements were statistically significant for the Word-Number, Choosing-a-Path, and Division Tests. With respect to the air breathing control value, average scores for these tests were reduced by 38.1, 58.6, and 28.5%, respectively. Despite the observed large decrement, the Choosing-a-Path Test was not selected for further evaluation, because the excessive time required for each response greatly reduced the range of possible scores that could be achieved within a limited time. Conversely, the Letter Order Test was selected for further evaluation, despite its apparent insensitivity to hypoxia, because it was considered that its sensitivity could be improved with more experienced subjects.

Accordingly, the Word-Number, Letter Order, and Division Tests were selected for final evaluation in subjects who were trained to achieve baseline plateaus in all three tests.

7.4 Phase IV - CO₂ Effects During Hypoxia

7.4.1 Effects of Breathing 10% Oxygen for 60 Minutes on Physiological and Mental Functions (Experienced Subjects)

Mental Function Responses

Results of mental function testing under normoxic and hypoxic conditions in nine experienced subjects are summarized in Fig. VI-2. Average air breathing control scores for the Word-Number, Letter Order, and Division Tests were 21, 61, and 14% higher, respectively, than the corresponding scores achieved by inexperienced subjects. Scores of all three tests were significantly reduced during exposure to hypoxia by average decrements of 37.9, 47.6, and 21.6%, respectively. Percent decrements in all three tests in relation to hypoxic exposure duration are shown in Appendix Fig. VI-3.

All three tests were selected as mental function indices for the planned evaluation of carbon dioxide administration as an effective means for increasing CNS tolerance to hypoxia.

Physiological Responses

Average physiological responses to prolonged hypoxia in the nine experienced subjects are summarized graphically in Figs. VII-1 to VII-4. Upon abrupt administration of 10% O₂, end-tidal PO₂ fell rapidly, as the initiating event, to about 35 mm Hg, with little individual variability during the first half of the hypoxic exposure and some variation in association with mental function testing during the second half (Fig. VII-1). Average arterial oxyhemoglobin saturation decreased from about 98% during air breathing to 62-67% (Fig. VII-2). Ventilation increased from an average control value of 7.2 L/min to 9-13 L/min (Fig. VII-4). Reflecting the hypoxic stimulation of ventilation, estimated arterial PCO₂ (from end-tidal values) fell

from an average value of 42.5 mm Hg during air breathing to about 36 mm Hg during the first half of the exposure, declining further to about 34 mm Hg during the second half of the hypoxic exposure (Fig. VII-3). Average heart rate increased from 72 beats/min to about 89 beats/min within the first 10 minutes and then declined gradually to about 78 beats/min during the rest of the exposure to hypoxia (Fig. VII-2).

7.4.2 Contrast of Hypoxia Alone and Hypoxia with Restored Normocapnia, on Mental Performance and Physiological Responses

CO₂ Effects Upon Mental Function during Hypoxia

The potential for amelioration of hypoxic effects on mental function by concurrent administration of carbon dioxide was evaluated in eight subjects exposed continuously to 10% O₂ for 60 minutes. 4.1% CO₂ was added over the final 23 minutes of 10% O₂ exposure. Following air control measurements, the Word-Number, Letter Order, and Division Tests were administered to each subject beginning at about 28 minutes of 10% O₂ exposure (see Fig. VII-1 for timing). All three tests were then repeated beginning at 10 minutes after the start of CO₂ addition. Average scores for all three mental function tests during air breathing, exposure to 10% O₂, and exposure to 10% O₂ with 4.1% CO₂ are summarized in Fig. VI-2 and percent decrements in relation to exposure duration in Fig. VI-3.

Average scores of the word-number and division tests were significantly reduced by 25.5 and 34.4%, respectively, during exposure to hypoxia alone and were not significantly different from normoxic control scores after addition of 4.1% CO₂ to the breathing gas. In contrast, average scores of the Letter Order Test were not significantly reduced during hypoxia alone, with an average decrement of only 10.1%, but were significantly decreased by 32.4% after addition of CO₂ to the breathing gas. Upon detailed evaluation of this test to determine why the corresponding pattern of responses differed so markedly from those found for the other two tests, it was discovered that the degrees of difficulty among test items varied randomly. Therefore, the relative scores achieved during repeated Letter Order Testing were influenced as much by the random variation in degree of difficulty as they were by concurrent changes in the mental competence of the subject. By comparison, repeated presentations of the Word-Number and Division Tests were more uniform in degree of difficulty.

CO₂ Effects upon Physiologic Responses to Hypoxia

Physiological responses to hypoxia with restored normocapnia are summarized in Figs. VII-1 to VII-2. End-tidal PO₂ fell from an average control value of 108 mm Hg to level off at about 36 mm Hg during exposure to hypoxia alone (Fig. VII-1). After addition of CO₂, end-tidal PO₂ rose to stabilize at about 55 mm Hg. Arterial oxyhemoglobin saturation decreased from an average control value of 98% to about 68-72% during exposure to hypoxia, and rose to level off at about 91% after addition of CO₂ (Fig. VII-2). Ventilation increased from an average control value of 7.7 L/min to 10-12 L/min while breathing 10.0% O₂ and increased further to 21-23 L/min after addition of 4.1% CO₂ (Fig. VII-4). Estimated arterial PCO₂ fell from an average value of 43.2 mm Hg during air breathing to about 36 mm Hg

during exposure to hypoxia alone and rose to plateau at about 44.5 mm Hg after addition of CO₂ (Fig. VII-3). Average heart rate increased from a control value of 75 beats/min to 88-93 beats/min during exposure to hypoxia alone. After addition of CO₂ to the inspired gas, heart rate actually declined, to range from average values of 76-84 beats/min, with an overall average of about 80 beats/min (Fig. VII-2).

8.0 SUMMARY - SCIENTIFIC RELEVANCE OF RESULTS AND IMPACT ON FUTURE RESEARCH PLANS

The Performance Measurement System (Appendix A) proved highly adaptable to studies of changing hypoxic stress. Of the six tests of mental function that were evaluated during the second Program year, a word-number test of associative memory ability and a division test of numerical ability were consistently impaired during exposure to 10% O₂. Scores of both tests were restored to normoxic control levels when 4.1% CO₂ was added to the inspired gas while continuing the exposure to hypoxia. This represented an acute adaptation to acute atmospheric hypoxia.

Physiological responses to restored normocapnia in hypoxia included an increment in ventilation that was sufficient to raise arterial oxyhemoglobin saturation to about 91% from a level of about 70% during exposure to hypoxia alone. The improvement in arterial oxygen content was associated with reversal of a mild tachycardia that was present during exposure to hypoxia alone.

During immersion studies measurement of the Transcranial Doppler index of brain blood flow was possible. The initial research plan included the use of immersion to simulate microgravity influences on ventilatory and brain circulatory adaptations to hypoxia with and without restored normocapnia. The immersion studies, done in the presence of normoxic or hyperoxic levels of oxygenation, showed no detectable influences of total immersion on ventilatory or cerebral circulatory responses to either incremental exercise or progressive hypercapnia. This indicates that the influence of restored normocapnia on CNS tolerance to hypoxia is also unlikely to be affected by immersion.

The results provided in Graphic Summary are all consistent with our initial hypothesis that CNS tolerance to atmospheric hypoxia can be increased promptly and significantly by purposeful elevation of inspired carbon dioxide partial pressures. The influence of carbon dioxide in limiting the degree of arterial hypoxemia is expected to produce further advantage through improvement in brain blood (oxygen) flow. At the time these experiments were performed, the techniques for measuring MCA blood flow velocity by Transcranial Doppler (TCD) ultrasonography were not fully developed in our laboratory. The important measurement of brain oxygenation will be included in the planned extension of the Program. The physiological influence of exercise on CNS tolerance to hypoxia alone and with restored

normocapnia will also be determined, along with specific study of the rates of adaptation of physiologic responses.

9.0 PERSPECTIVE FOR FUTURE RESEARCH - Relevance of Carbon Dioxide/Hypoxia Interactions to NASA Biomedical Research Program and Operations

In the self-contained spacecraft, vehicles, stations, suits or other human containments in space activity, the desired safety and functional capability depend primarily, both acutely and in prolonged missions, upon the respiratory environmental, carbon dioxide, nitrogen, and oxygen. Normal individuals adapt gradually on earth to a considerable range of prolonged exposure to atmospheres of reduced oxygen pressure, having no appreciable carbon dioxide content. Natural states of prolonged human exposure to elevated carbon dioxide pressures, with or without concurrent hypoxia, do not exist on earth except in chronic pulmonary disease. Nevertheless, potentially useful, acutely hazardous, or chronically detrimental exposures to carbon dioxide and/or hypoxia are all considered important possibilities within forthcoming prolonged human activity in space. Enhanced ability to adapt to accidental or purposeful atmospheric changes in oxygen and/or carbon dioxide could save lives of crew members, sustain performance capability, or avoid premature termination of missions. Reduction of ambient pressure in spacecraft or station reduces decompression hazard, suit pressure requirement, reduces leakage rate, and, without oxygen supplementation, reduces fire hazard (14,17,22,35). Elevation of inspired carbon dioxide in acute (possibly accidental) hypoxic states sustains consciousness and mobility for emergency action (10,30). For such reasons, experiment and analysis providing for prediction of physiological tolerance and performance capability in unusual respiratory environments is an essential component of NASA Biomedical Research and Operations programs.

The data obtained concerning CO₂-O₂ interactions in hypoxia will continue to be paralleled by data obtained concerning CO₂-Hyperoxia interactions (Fig. VIII-1).

/
CORRECT

10.0 REFERENCES

1. Aaslid, R., T. M. Markwalder, and H. Nornes. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. *J. Neurosurg.* 57: 769-774, 1982.
2. Asmussen, E., and M. Nielsen. Ventilatory response to CO₂ during work at normal and at low oxygen tensions. *Acta Physiol. Scan.* 39(1): 27-35, 1957.
3. Butler, F. K., Jr., and E. D. Thalmann. Central nervous system oxygen toxicity in closed circuit scuba divers II. *Undersea Biomed. Res.* 13: 193-223, 1986.
4. Clark, J. M., and C. J. Lambertsen. Extension of central nervous and visual system oxygen tolerance in physical work. Combined final report for Navy Contract Nos. N00014-88-K-0270 and N00014-88-K-0318. Philadelphia, PA: Institute for Environmental Medicine, University of Pennsylvania, 1990.
5. Denison, D. M., F. Ledwith, and E. C. Poulton. Complex reaction times at simulated cabin altitudes of 5000 feet and 8000 feet. *Aerosp. Med.* 37: 1010-1013, 1966.
6. Donald, K. W. Oxygen poisoning in man. *Brit. Med. J.* 1: 667-672, 712-717, Parts I and II, 1947.
7. Dyer, F. N. Effects of low and high oxygen tensions and related respiratory conditions on visual performance: A literature review. USAARL Report No. 88-7. Fort Rucker, AL: United States Army Aeromedical Research Laboratory, 1988.
8. Ernsting, J. Prevention of hypoxia-acceptable compromises. *Aviat. Space Environ. Med.* 56: 1004-1008, 1985.
9. Gauer, O. H. Recent advances in the physiology of whole body immersion. *Acta Astronaut.* 2: 31-39, 1975.
10. Gibbs, F. A., E. L. Gibbs, W. G. Lennox, and L. F. Nims. The value of carbon dioxide in counteracting the effects of low oxygen. *J. Aviat. Med.* 14: 250-261, 1943.
11. Harvey, T. C., M. E. Raichle, M. H. Winterborn, J. Jensen, N. A. Lassen, N. V. Richardson, and A. R. Bradwell. Effect of carbon dioxide in acute mountain sickness: A rediscovery. *Lancet* 639-641, 1988.
12. Kelman, G. R., and T. J. Crow. Impairment of mental performance at a simulated altitude of 8000 feet. *Aerosp. Med.* 40: 981-982, 1969.

13. Kety, S. S., and C. F. Schmidt. The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. *J. Clin. Invest.* 27(4): 484-492, 1948.
14. Knight, D. R. The medical hazards of flame-suppressant atmospheres. NSMRL Report No. 1167. Naval Submarine Medical Research Laboratory, 1991.
15. Lahiri, S. Physiological responses and adaptations to high altitude. *Intl. Rev. Physiol.: Environ. Physiol. II*, Vol. 15. Robertshaw, D. (Ed.) Baltimore, MD: University Park Press, 1977.
16. Lambertsen, C. J. Hypoxia, altitude, and acclimatization. *Medical Physiology* (14th ed.). Mountcastle, V. B. (Ed.). St. Louis, MO: Mosby, 1980.
17. Lambertsen, C. J. A philosophy of extremes for the gaseous environment of manned closed ecological systems. *Aerosp. Med.* 34: 291-299, 1963a.
18. Lambertsen, C. J. Chemical control of respiration at rest. *Medical Physiology* (14th ed.) Mountcastle, V. B. (Ed.). St. Louis, MO: Mosby, 1774-1827, 1980.
19. Lambertsen, C. J. Effects of excessive pressure of oxygen, nitrogen, helium, carbon dioxide, and carbon monoxide. *Medical Physiology* (14th ed.). Mountcastle, V. B. (Ed.) St. Louis, MO: Mosby, 1901-1944, 1980.
20. Lambertsen, C. J. *The Environmental Biomedical Research Data Center. Institute For Environmental Medicine*, Report No. 6-1-93. Philadelphia, PA: Institute for Environmental Medicine, University of Pennsylvania, 1993.
21. Lambertsen, C. J. *CO₂-O₂ Interactions in Extension of Tolerance to Hypoxia*. NASA Project NAG 9-597 Annual Report. Philadelphia, PA: Institute for Environmental Medicine, University of Pennsylvania, 1993.
22. Lambertsen, C. J. Physiological interactions and gaseous environment in manned exploration of space. *Fed. Proc.* 22: 1046-1050, 1963.
23. Lambertsen, C. J. Effects of hyperoxia on organs and their tissues. *Extrapulmonary Manifestations of Respiratory Disease*. Robin, E. D. Ed. Vol. 8 of *Lung Biology in Health and Disease*. Lenfant, C. (Ed.) New York: Dekker, 239-303, 1978.
24. Lambertsen, C. J., J. H. Ewing, R. H. Kough, R. A. Gould, and M. W. Stroud, 3rd. Oxygen toxicity. Arterial and internal jugular blood gas composition in man during inhalation of air, 100% O₂ and 2% CO₂ in O₂ at 3.5 atmospheres ambient pressure. *J. Appl. Physiol.* 8: 255-263, 1955.

25. Lambertsen, C. J. Extension of oxygen tolerance: The philosophy and significance. *Symposium on Extension of Oxygen Tolerance: Exper. Lung. Res.* 14: 1035–1058, 1988.
26. Lennox, W. G., and E. L. Gibbs. The blood flow in the brain and the leg of man, and the changes induced by alteration of blood gases. *J. Clin. Invest.* 11: 1155–1177, 1932.
27. Loeschcke, H. H., and K. H. Gertz. Einfluss des O₂-Druckes in der Einatemungsluft auf die Atemtatigkeit des Menschen, gepruft unter Konstanthaltung des Alveolaren CO₂-Druckes. *Pflugers Arch.* 267: 460–477, 1958.
28. Nicogossian, A. E., and L. F. Dietlein. Microgravity: Simulation and analogs. *Space Physiology and Medicine* (2nd ed.). Nicogossian, A. E., C. L. Huntoon, and S. L. Pool (Eds.). Philadelphia, PA: Lea and Febiger, 240–249, 1989.
29. Nielsen, M., and H. Smith. Studies on the regulation of respiration in acute hypoxia. With an appendix on respiratory control during prolonged hypoxia. *Acta Physiol. Scan.* 24: 293–313, 1951.
30. Pierce, E. C., C. J. Lambertsen, M. J. Strong, S. C. Alexander, and D. Steele. Blood PCO₂ and brain oxygenation at reduced ambient pressure. *J. Appl. Physiol.* 17: 899–908, 1962.
31. Raichle, M. E., and F. Plum. Hyperventilation and cerebral blood flow. *Stroke* 3: 566–575, 1972.
32. Raichle, M. E., J. B. Posner, and F. Plum. Cerebral blood flow during and after hyperventilation. *Arch. Neurol.* 23: 394–403, 1970.
33. Sayers, J. A., R. E. A. Smith, R. L. Holland, and W. R. Keatinge. Effects of carbon dioxide on mental performance. *J. Appl. Physiol.* 63(1): 25–30, 1987.
34. Shock, N. W., and R. Scow. The effect on learning of repeated exposures to lowered oxygen tension of the inspired air. *J. Comp. Phys. Psychol.* 34: 55–63, 1942.
35. Shvartz, E. Advantages of a low-oxygen environment in space cabins. *Aviat. Space Environ. Med.* 61: 272–276, 1990.
36. Siesjo, B. K., H. Johannson, K. Norberg, and L. Salford. Brain function, metabolism, and blood flow in moderate and severe arterial hypoxia. *Brain Work. Alfred Benzon Symposium VIII.* Munksgaard, 101–125, 1975.

37. Slobodnik, B., M. T. Wallick, and J. M. Chimiak. Effectiveness of oxygen-nitrogen gas mixtures in inducing hypoxia at 1 ATA. U.S. Navy Experimental Diving Unit Report 4-91, 1991.
- 37.a. Sorensen, S. C., and J. W. Severinghaus. Respiratory sensitivity to acute hypoxia in man at sea level and at high altitude. *J. Appl. Physiol.* 25: 211-216, 1968.
38. Squires, R. W., and E. R. Buskirk. Aerobic capacity during acute exposure to simulated altitude. *Med. Sci. Sports Exer.* 14: 36-40, 1982.
39. Thomas, S. N., T. Schroeder, N. H. Secher, and J. H. Mitchell. Cerebral blood flow and maximal dynamic exercise in humans. *J. Appl. Physiol.* 67(2): 744-748, 1989.
40. Torbati, D., J. Greenberg, and C. J. Lambertsen. Regional cerebral glucose metabolic rate during thirty minute hypoxia of 7% oxygen in adult conscious rats. *Neurosci. Lett.* 65: 253-258, 1986.
41. Vacher, J. M., and A. T. Miller, Jr. Altitude acclimatization: Its effects on hypoxia-induced performance decrements. *Psychopharmacol. (Berlin)* 12: 250-257, 1968.
42. Ward, M. P., J. S. Milledge, and J. B. West. *High Altitude Medicine and Physiology*. Philadelphia, PA: University of Pennsylvania Press, 1989.
43. Webster, A. P., and O. E. Reynolds. High altitude, high velocity flying with special reference to the human factors. II. Time of consciousness during exposure to various pressure altitudes. *Aviat. Med.* 237-245, 1950.
44. Weil, J. V., E. Byrne-Quinn, I. E. Sodal, G. F. Filley, and R. F. Grover. Acquired attenuation of chemoreceptor function in chronically hypoxic man at high altitude. *J. Clin. Invest.* 50: 186-195, 1971.
45. Welch, H. G. Effects of hypoxia and hyperoxia on human performance. *Exer. Sport Sci. Rev.* 15: 191-221, 1987.
46. West, J. B., S. J. Boyer, D. J. Graber, P. H. Hackett, K. H. Manet, J. Milledge, R. M. Peters, Jr., C. J. Pizzo, M. Sainaja, F. H. Sarnquist, R. B. Schoene, and R. M. Winslow. Maximal exercise at extreme altitudes on Mount Everest. *J. Appl. Physiol.: Respir. Environ. Exer. Physiol.* 55: 688-698, 1983.
47. Yarbrough, O. D., W. Welham, E. J. Brinton, and A. R. Behnke. Symptoms of oxygen poisoning and limits of tolerance at rest and at work. Navy Experimental Diving Unit Report 01-47, 1947.

11.0 REPORTS AND PUBLICATIONS—Present Grant Period (Cumulative; *=Abstracts)

1995

*Clark, J. M., R. Gelfand, C. J. Lambertsen, G. Beck, Jr., K. R. Hardy. Ventilatory, arterial PCO₂, and cerebral circulatory responses to incremental exercise during O₂ breathing at 2.0 ATA (Abstract). Philadelphia, PA: Institute for Environmental Medicine, University of Pennsylvania, 1995. (Submitted to *Undersea Hyperbaric Med.* In press.)

Clark, J. M., R. Gelfand, C. J. Lambertsen, W. C. Stevens, G. Beck, Jr., and D. F. Fisher. Human tolerance and physiological responses to exercise while breathing oxygen at 2.0 ATA. *Aviat. Space Environ. Med.* 66: 336–45, 1995.

*Gelfand, R., C. J. Lambertsen, G. Beck, Jr., and J. M. Clark. Dynamic responses of S_aO₂ and "CBF" to abrupt exposure to inhaled 10% O₂ / 4% CO₂ at rest, followed by 50 and 100 watts exercise (Abstract). Philadelphia, PA: Institute for Environmental Medicine, University of Pennsylvania, 1995. (Submitted to *Undersea Hyperbaric Med.* In press.)

Thom, S. R., R. L. Taber, I. I. Mendiguren, J. M. Clark, K. R. Hardy, and A. B. Fisher. Delayed neuropsychological sequelae following carbon monoxide poisoning and its prophylaxis by treatment with hyperbaric oxygen. *Ann. Emer. Med.* 25: 474–480, 1995.

1994

*Beck, G., R. Gelfand, T. S. Pak, R. D. Fagan, and J. M. Clark. Immersion effects on bicycle ergometry: O₂ vs. load and pedal rate (Abstract). *Undersea Hyperbaric Med.* 21(5): 37, 1994.

Clark, J. M. Modification of oxygen tolerance in rats by adaptation to combined hypoxia and hypercapnia. *Undersea Hyperbaric Med.* 21: 251–264, 1994.

*Clark, J. M., R. Gelfand, G. Beck, Jr., B. A. Youdelman, E. J. Hopkin, and C. J. Lambertsen. Effects of head-out and total immersion at 1.0 ATA on ventilatory and cerebral circulatory responses to progressive hypercapnia (Abstract). *Undersea Hyperbaric Med.* 21(Supp.): 36, 1994.

*Gelfand, R., B. Youdelman, J. Clark, and C. Lambertsen. Mental performance and physiological effects of hypoxia (0.1 ATA O₂ insp.): Effects of restored normocapnia (Abstract). *FASEB J.* 8(4): A554, 1994.

*Gelfand, R., J. M. Clark, G. Beck, Jr., B. A. Youdelman, E. J. Hopkin, and C. J. Lambertsen. Effects of head-out and total immersion on ventilatory and cerebral circulatory responses to progressive exercise at 1.0 ATA in humans (Abstract). *Undersea Hyperbaric Med.* 21(Supp.): 37, 1994.

*Lambertsen, C. J. Acute physiologic adaptation to acute hypoxia. Relation to fire prevention and extinguishment in closed spaces (Abstract). *Undersea Hyperbaric Med.* 21(Supp.): 38, 1994.

Lambertsen, C. J. *Final Report: Safety Analysis of NOAA Nitrox I and NOAA Nitrox II Decompression Tables*. Related to NOAA Project NA36 RU 0422. EBRDC Report 10-30-94. Philadelphia, PA: Environmental Biomedical Research Data Center, Institute for Environmental Medicine, University of Pennsylvania, 1994.

1993

*Clark, J. M., B. E. Skolnick, R. Gelfand, R. E. Farber, M. Steirheim, W. C. Stevens, G. Beck, Jr., and C. J. Lambertsen. Correlation of middle cerebral artery blood flow velocities with ¹³³Xe cerebral blood flow perfusion rates during oxygen breathing over a wide range of arterial PCO₂ values (Abstract). *Undersea Hyperbaric Med.* 20(Supp.): 12, 1993.

Farber, R. E., J. M. Clark, B. Skolnick, R. Gelfand, M. Stierheim, G. Beck, W. C. Stevens, and C. J. Lambertsen. Transcranial Doppler (TCD) Flow monitoring at varying levels of CO₂: Direct correlation with xenon blood flows over a wide range of PCO₂ values. *Neurology* 43(4): A342, 1993.

*Gelfand, R., J. M. Clark, and C. J. Lambertsen. Hypoxic and hypercapnic ventilatory responses following intermittent hyperoxia (30 min. O₂: 0 min. normoxia) at 2.0 ATA in man (Predictive Studies VI) (Abstract). *Undersea Hyperbaric Med.* 20(Supp.): 46-47, 1993.

Lambertsen, C. J. *Carbon Dioxide, Hypoxia, Epinephrine and Cardiac "Sensitization" Tests*. EBRDC Report 5-20-93. Philadelphia, PA: Institute for Environmental Medicine, Environmental Biomedical Research Data Center, University of Pennsylvania, 1993.

Stevens, W. C., J. M. Clark, R. Gelfand, G. Beck, M. Stierheim, R. E. Farber, and C. J. Lambertsen. Relationships of brain blood velocity to arterial PCO₂ at rest and during exercise. *Med. Sci. Sports Exer.* 25(5): S55, 1993.

1992

Lambertsen, C. J., M. L. Gernhardt, R. G. Miller, and E. J. Hopkin. *Development of Decompression Procedures. Air Diving with Surface Decompression Using Oxygen*. EBRDC Report 7-28-92. Philadelphia, PA: Institute for Environmental Medicine, Environmental Biomedical Research Data Center, University of Pennsylvania, 1992. (Preliminary basis for formal grant).

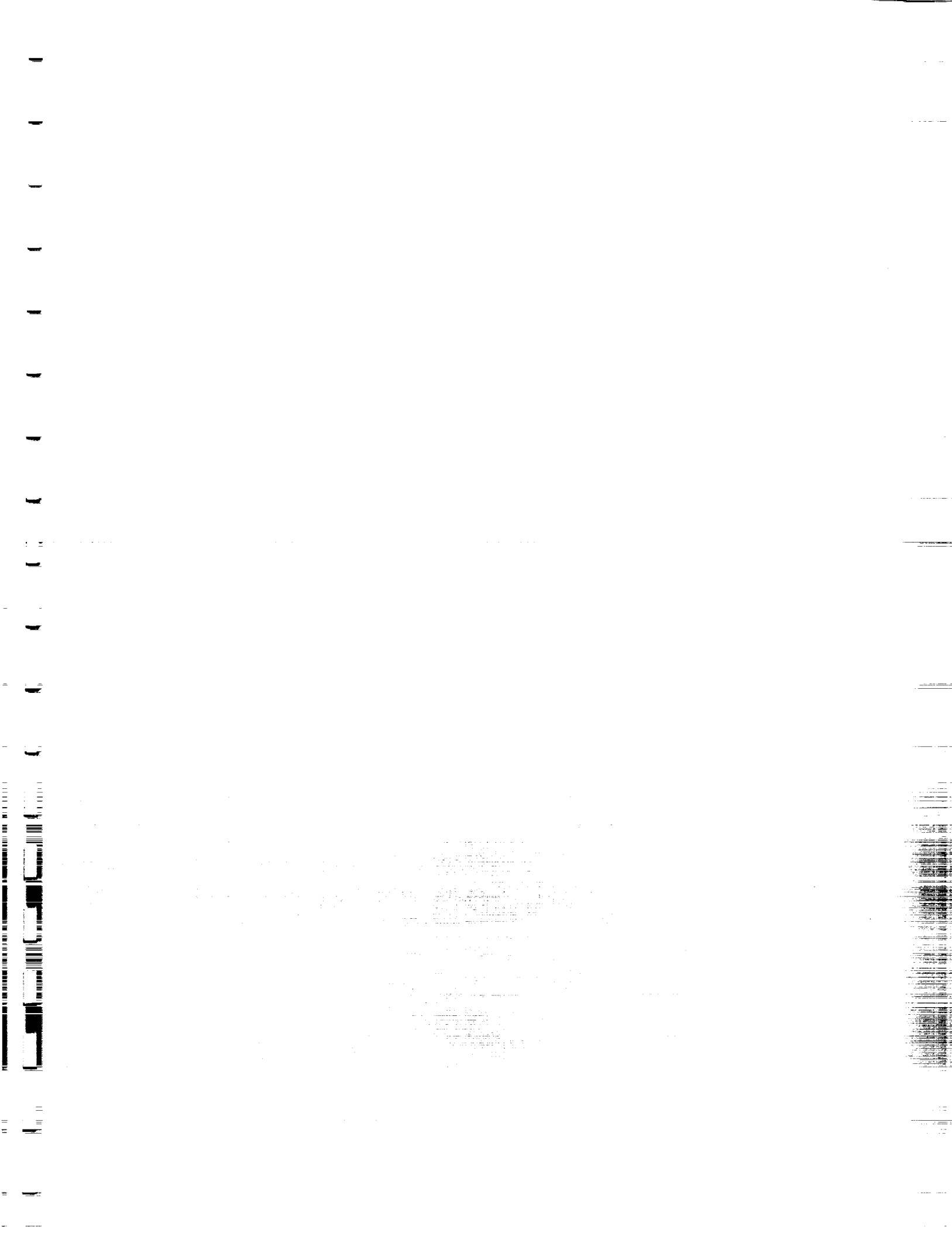
12.0 APPENDIXES

A. PERFORMANCE MEASUREMENT SYSTEM (BOUND SEPARATELY)

B. FIGURES. INDEX OF FIGURES

- I-1 • The Oxygen Program
- II-1 • Correlation of ¹³³Xenon Brain Volume Flow vs. Middle Cerebral Artery Flow Velocity (TCD)
- II-2 • Relations of % Change in CBF and % Change in Flow Velocity vs. Arterial PCO₂
- III-1 • Immersion Ergometer System
- III-2 • Immersion Ergometer. Effect of Pedaling Rate on O₂ Consumption
- III-3 • Immersion Ergometer. Relation of O₂ Consumption to Ergometer Work Load
- IV-1 • Immersion/Exercise. Brain Circulation
- IV-2 • Immersion/Exercise. Ventilation
- IV-3 • Immersion/Exercise. End-Tidal PCO₂
- V-1 • Immersion/Hypercapnia. Brain Circulation
- V-2 • Immersion/Hypercapnia. Ventilation
- V-3 • Immersion/Hypercapnia. Cardiac Rate
- V-4 • Immersion/Hypercapnia. Arterial Blood Pressure
- VI-1 • Mental Function / 10% O₂. Six Test Comparison With Inexperienced Subjects
- VI-2 • Mental Function / 10% O₂. Experienced Subjects. Three Selected Tests.
- VI-3 • Acute Adaptation to Hypoxia (4.1% CO₂ with 10% O₂)
- VII-1 • Physiological / CO₂ in Hypoxia. %Hb Saturation and Cardiac Rate
- VII-2 • Physiological / CO₂ in Hypoxia. End-Tidal PO₂
- VII-3 • Physiological / CO₂ in Hypoxia. End-Tidal PCO₂
- VII-4 • Physiological / CO₂ in Hypoxia. Ventilation
- VIII-1 • Arterial Hypercapnia Induced by Exercise in Hyperoxia

/
CORRECT



ATMOSPHERIC AND ENVIRONMENTAL PHYSIOLOGY AND TOXICOLOGY PROGRAM
(EXTERNAL AND INTERNAL ENVIRONMENT)

NAVY EMPHASIS
(on HYPEROXIA)

NASA EMPHASIS
(on HYPOXIA)

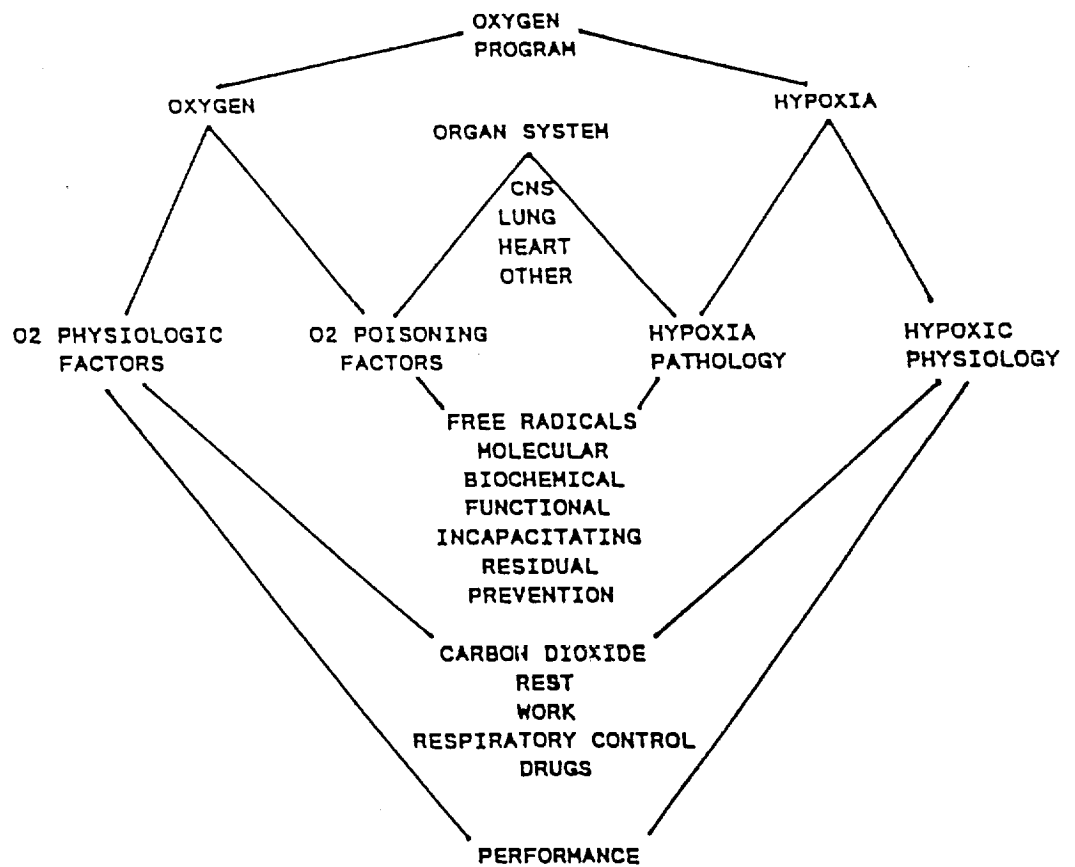
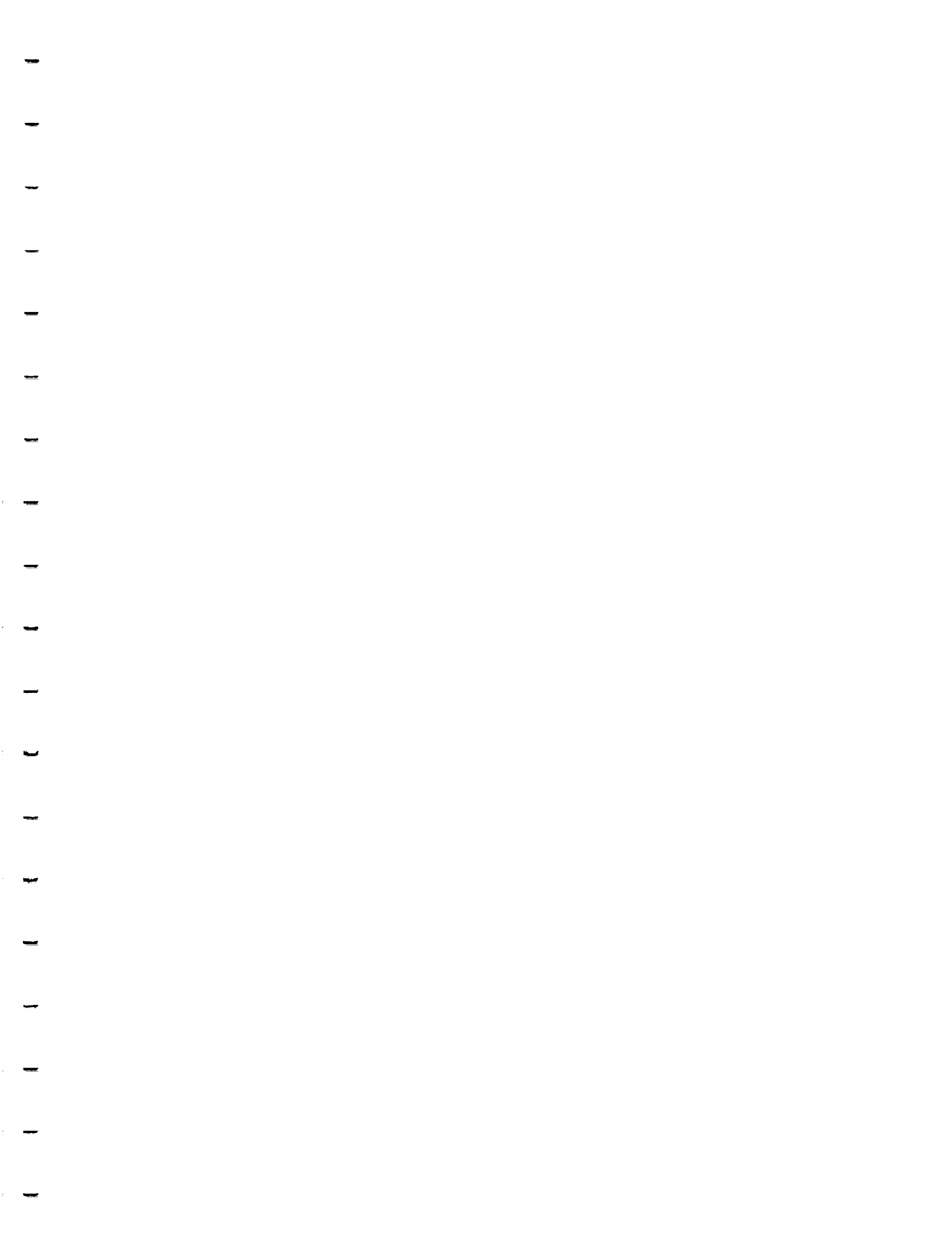


Fig. I-1



**Correlation of CBF (^{133}Xe) With MCA Blood Flow Velocity (TCD)
Over Arterial Pco_2 Range of 25 to 50 mm Hg
(Individual Values in 8 Subjects)**

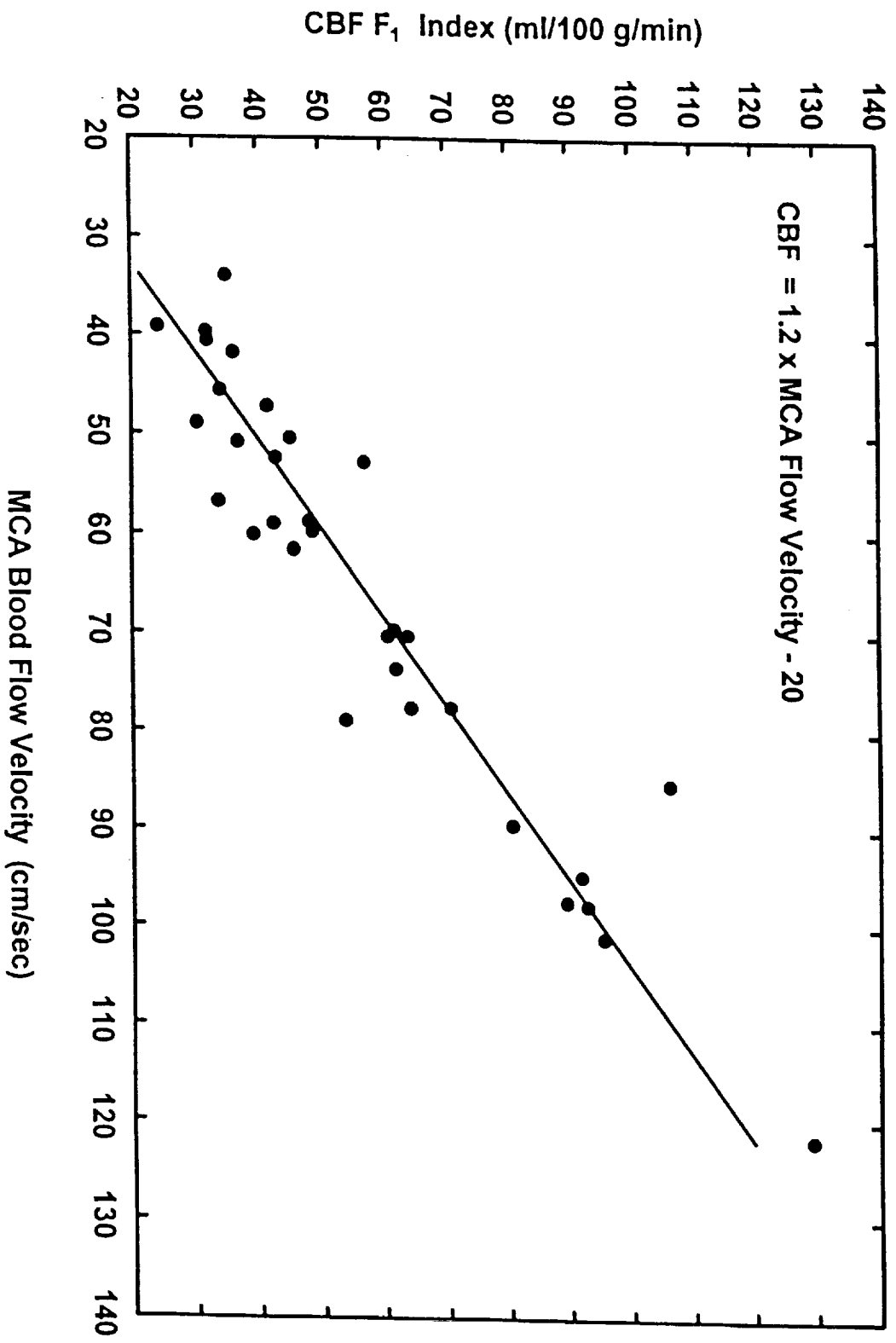


Fig. II-1

Relationships of Percent Changes in CBF F_1 Index and MCA Blood Flow Velocity to Arterial P_{CO_2} During Oxygen Breathing at 1.0 ATA

(Mean Values \pm SEM in 8 Subjects)

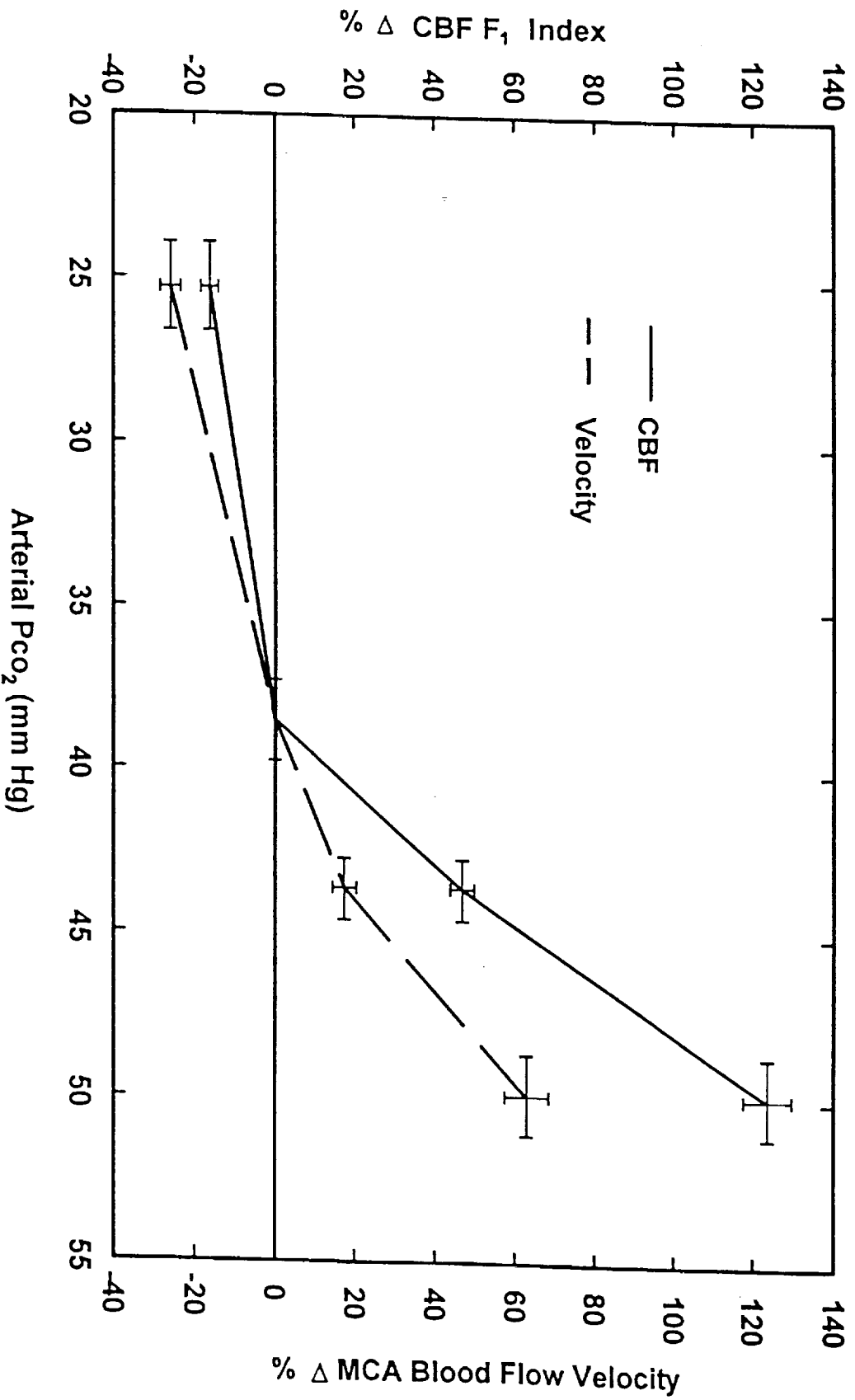
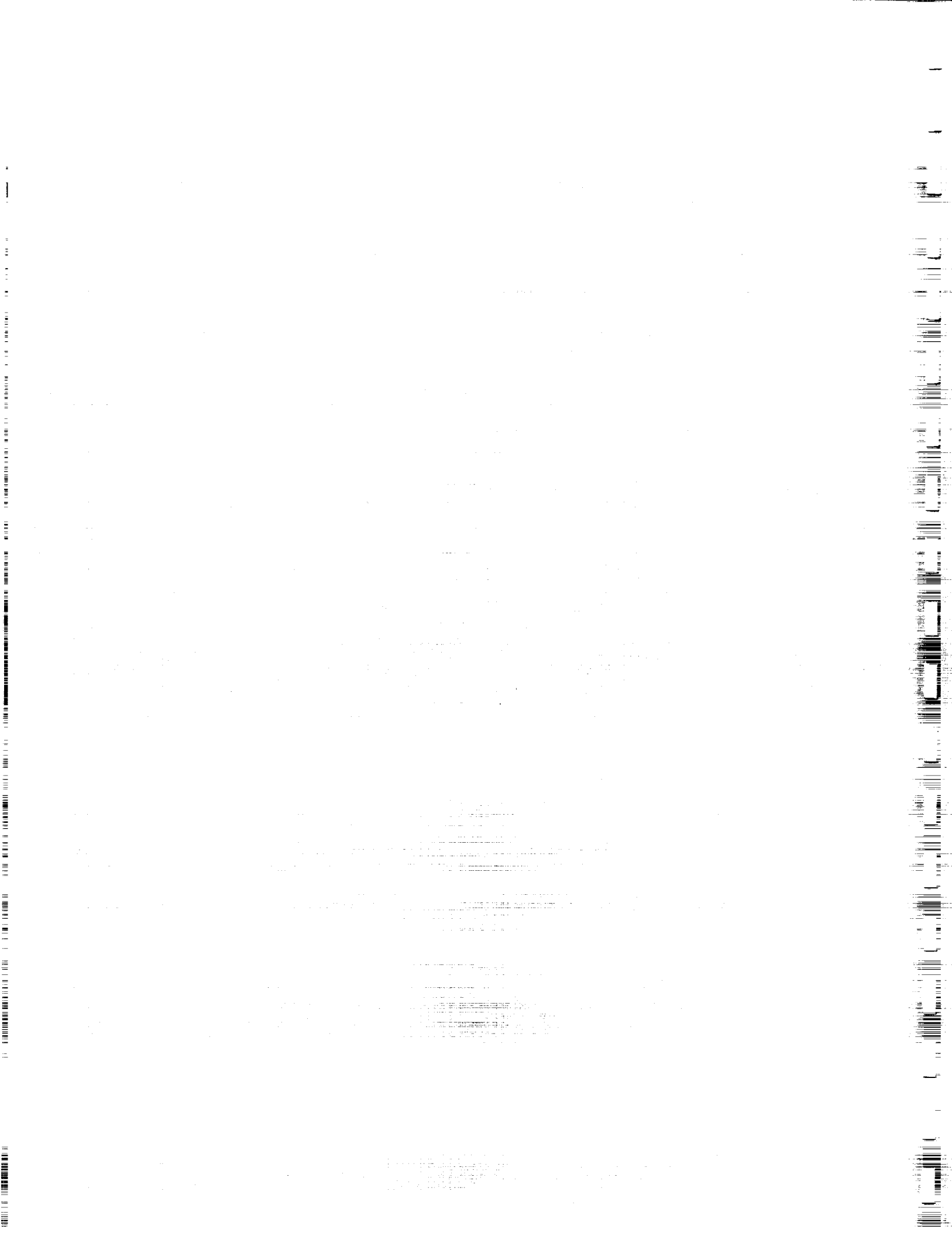


Fig. II-2



IMMERSION ERGOMETER

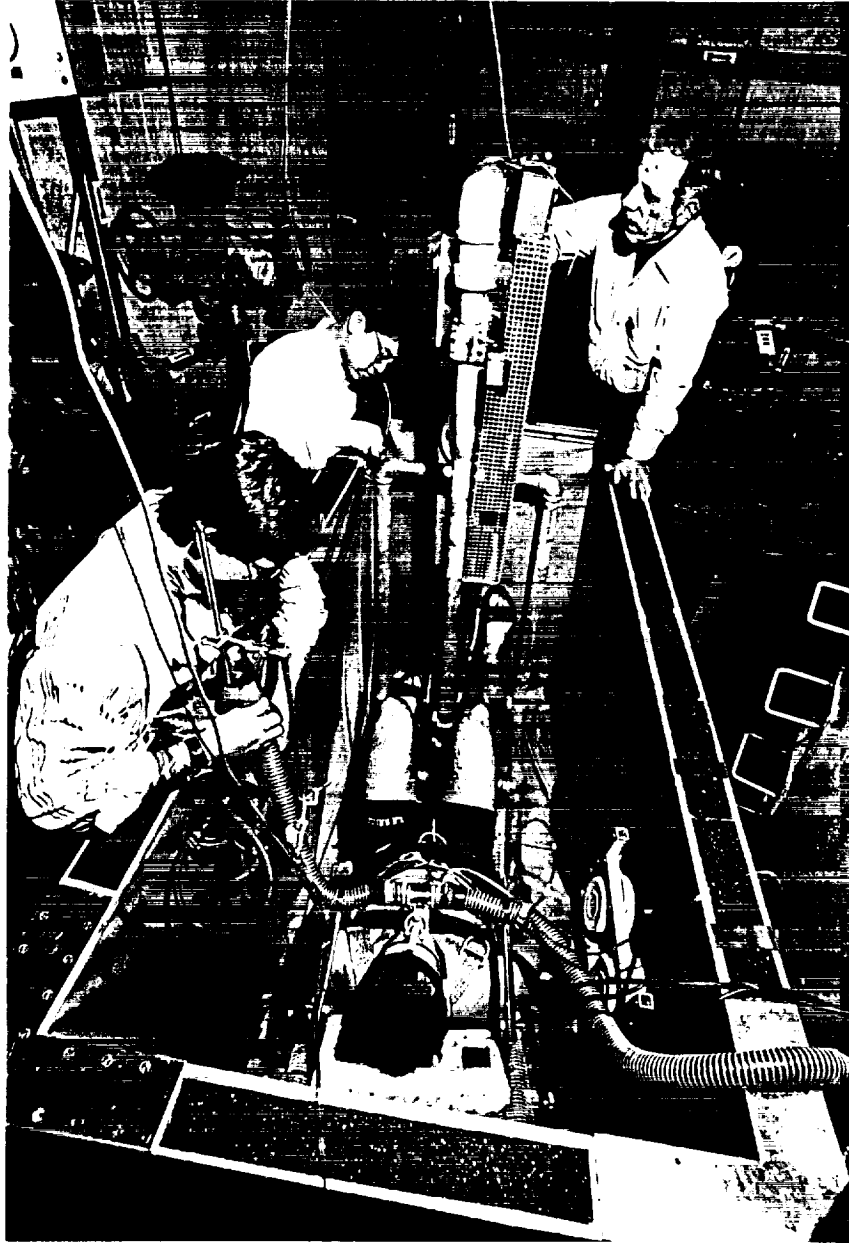


Fig. III-1

Effect of Pedaling Rate on $\dot{V}O_2$ Consumption Under Dry and Wet Conditions (Mean Values in 8 Subjects)

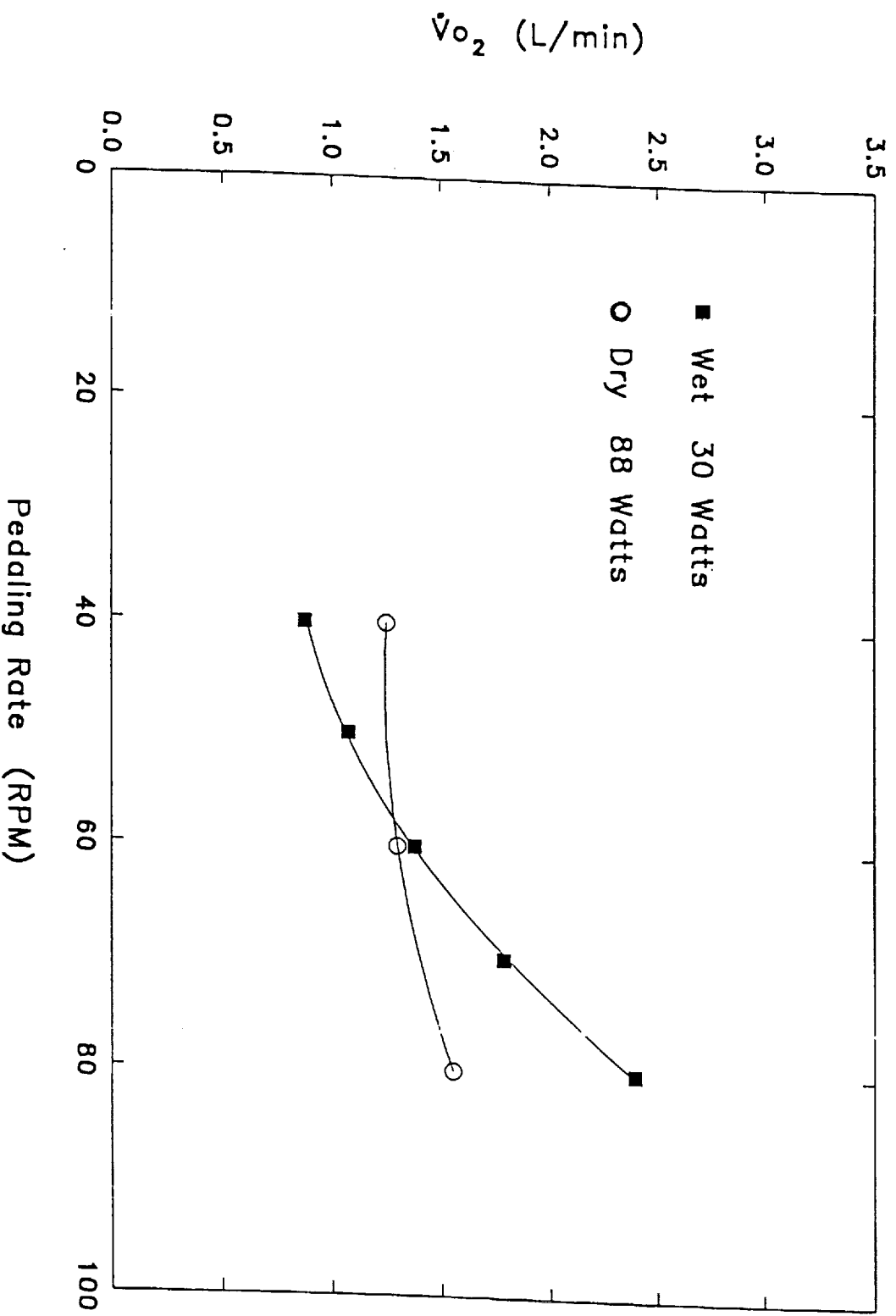


Fig. III-2

Relationships of O₂ Uptake to Ergometer Workload Under Wet and Dry Conditions

N=8, 60 RPM

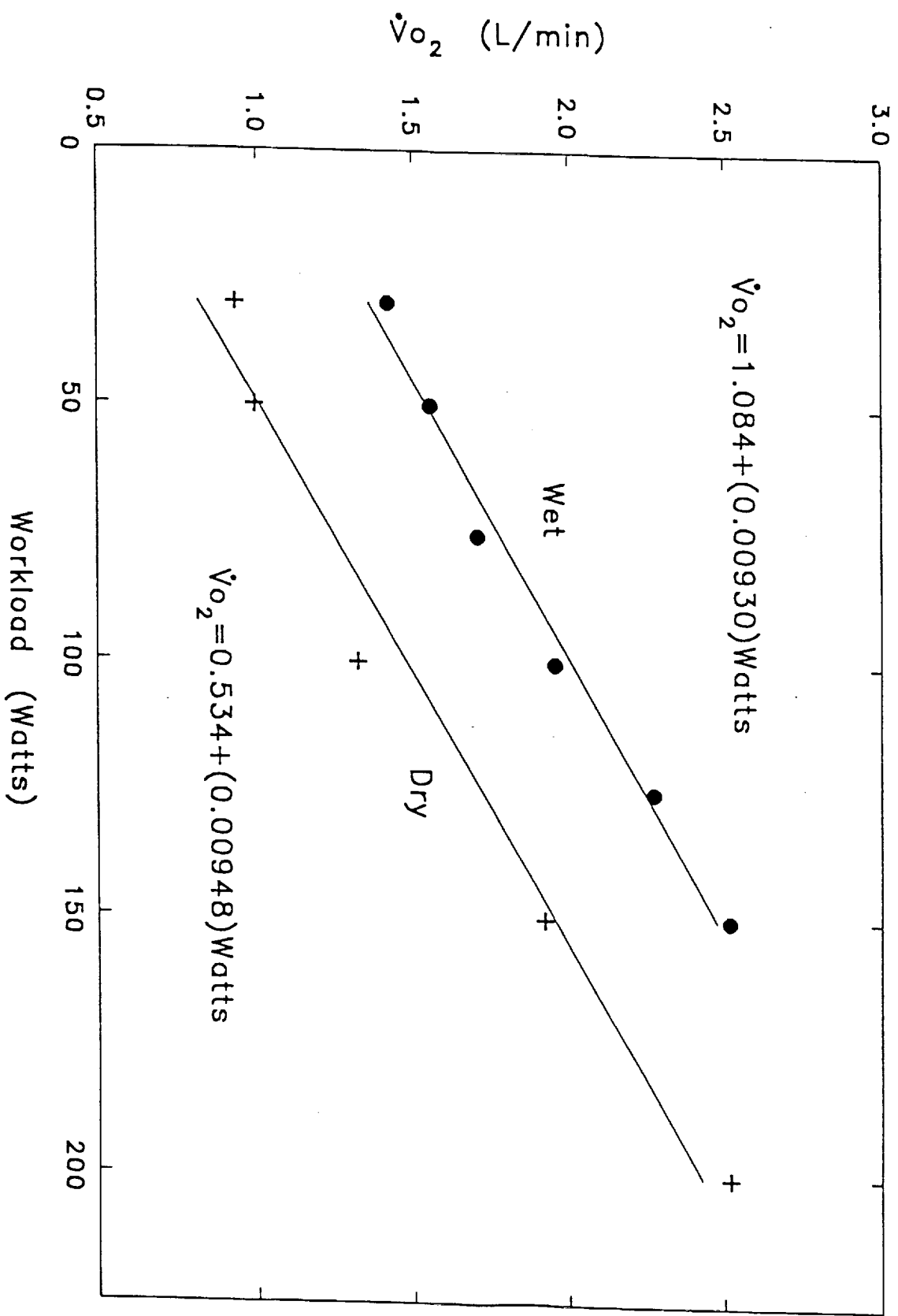


Fig. III-3

**EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON
 NORMALIZED CEREBRAL CIRCULATORY RESPONSES TO EXERCISE**
 (Mean Values \pm SEM, N = 6)

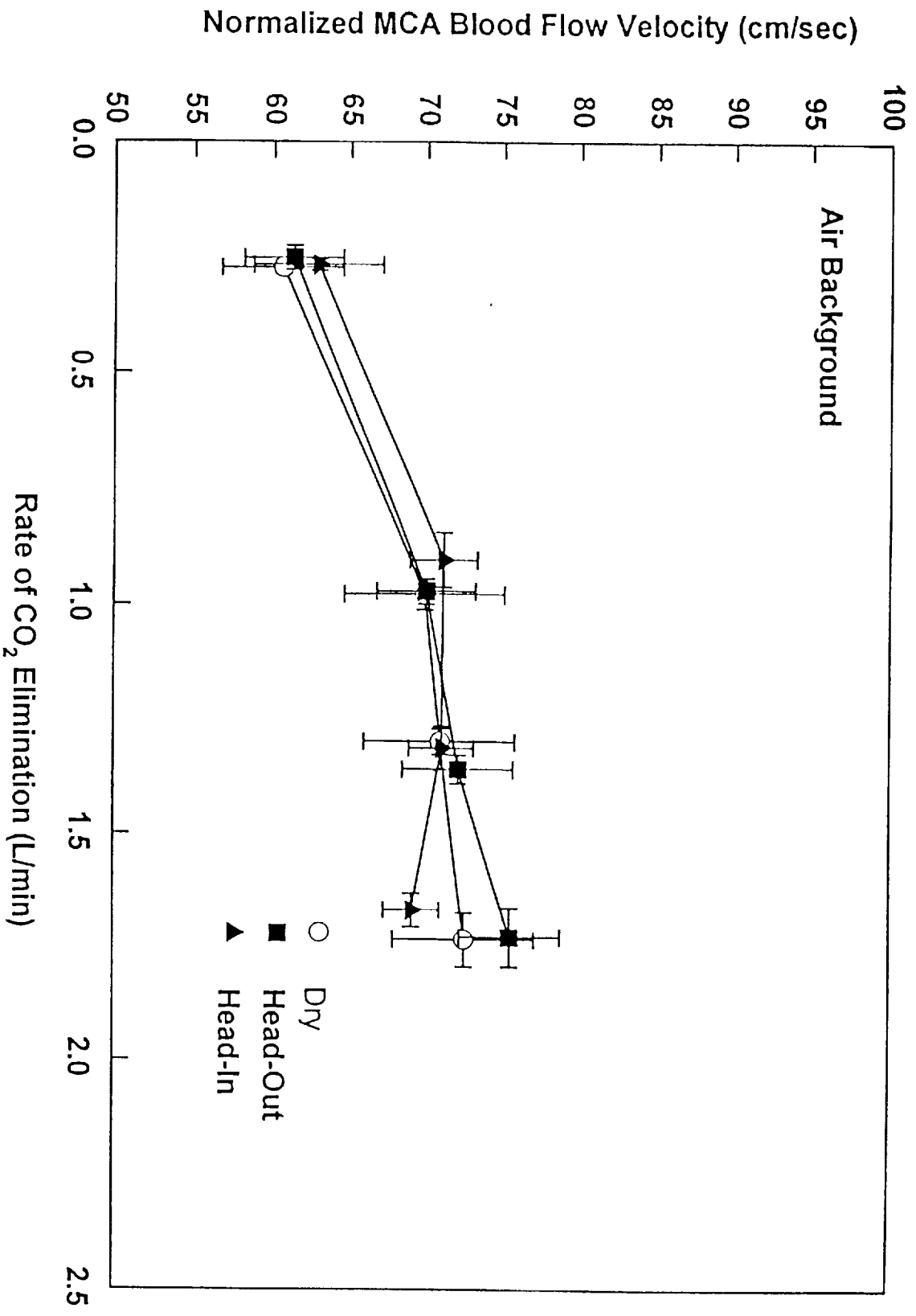


Fig. IV-1

EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON VENTILATORY RESPONSES TO EXERCISE

(Mean Values \pm SEM, N = 8)

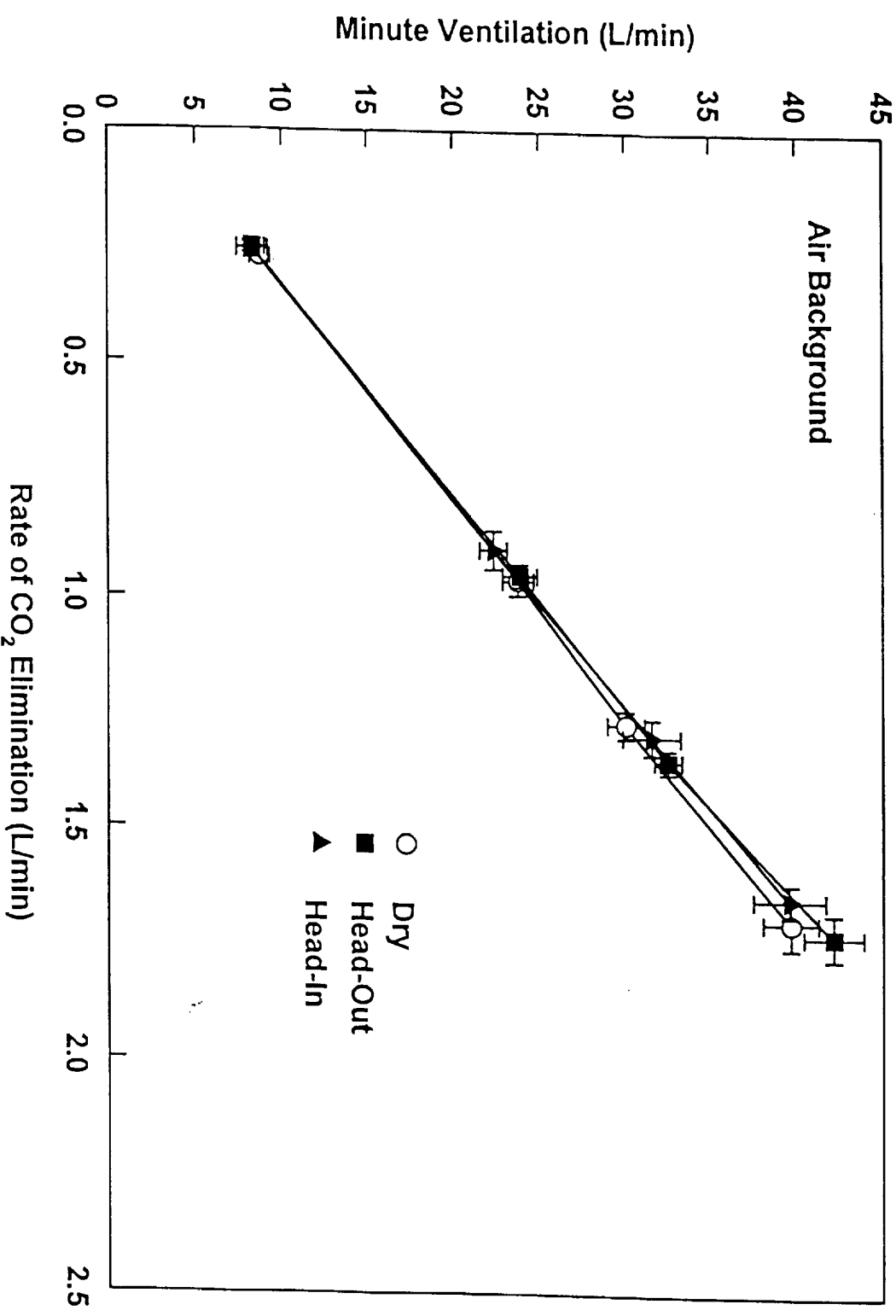


Fig. IV-2

EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON END-TIDAL CO₂ RESPONSES TO EXERCISE

(Mean Values \pm SEM, N = 8)

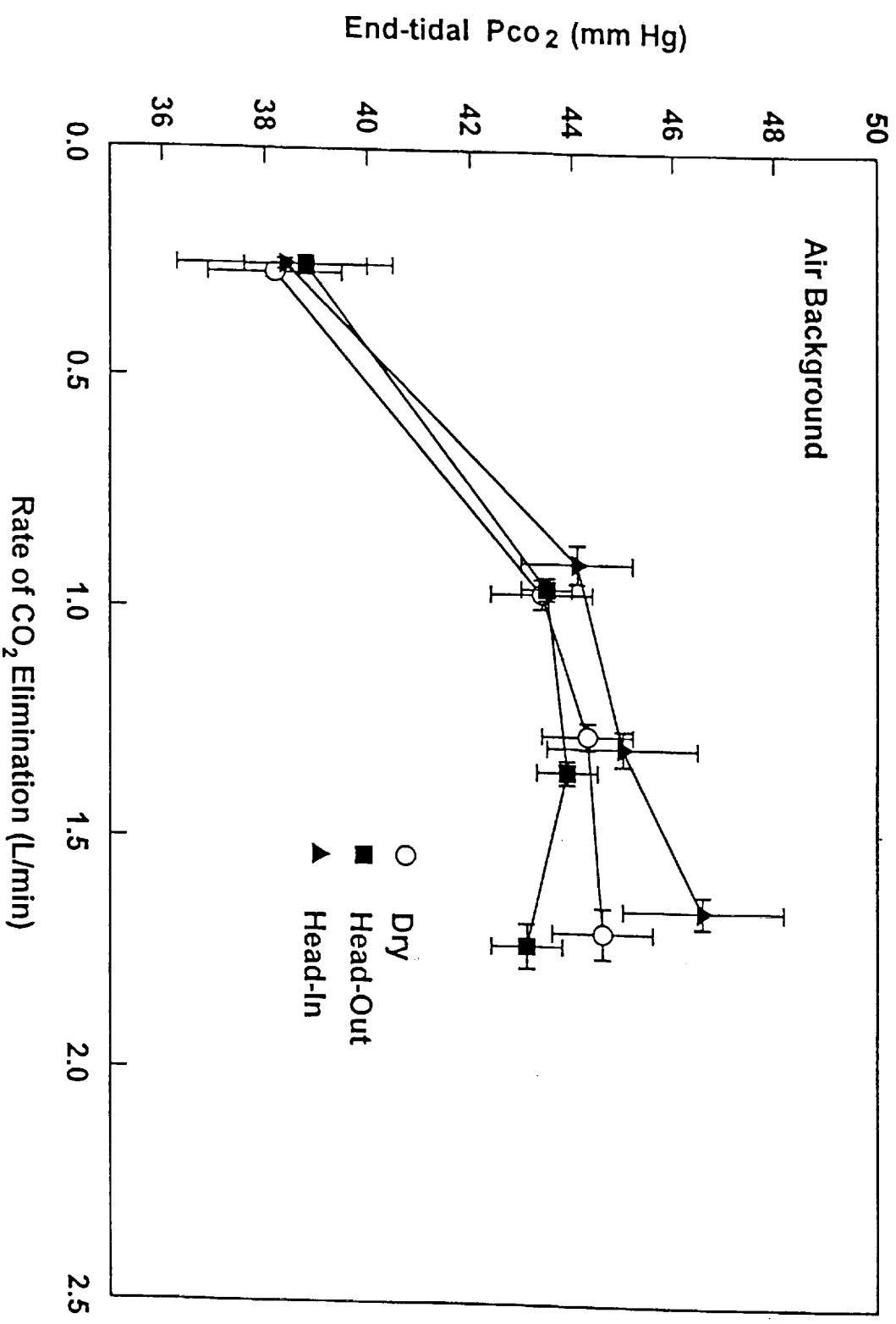
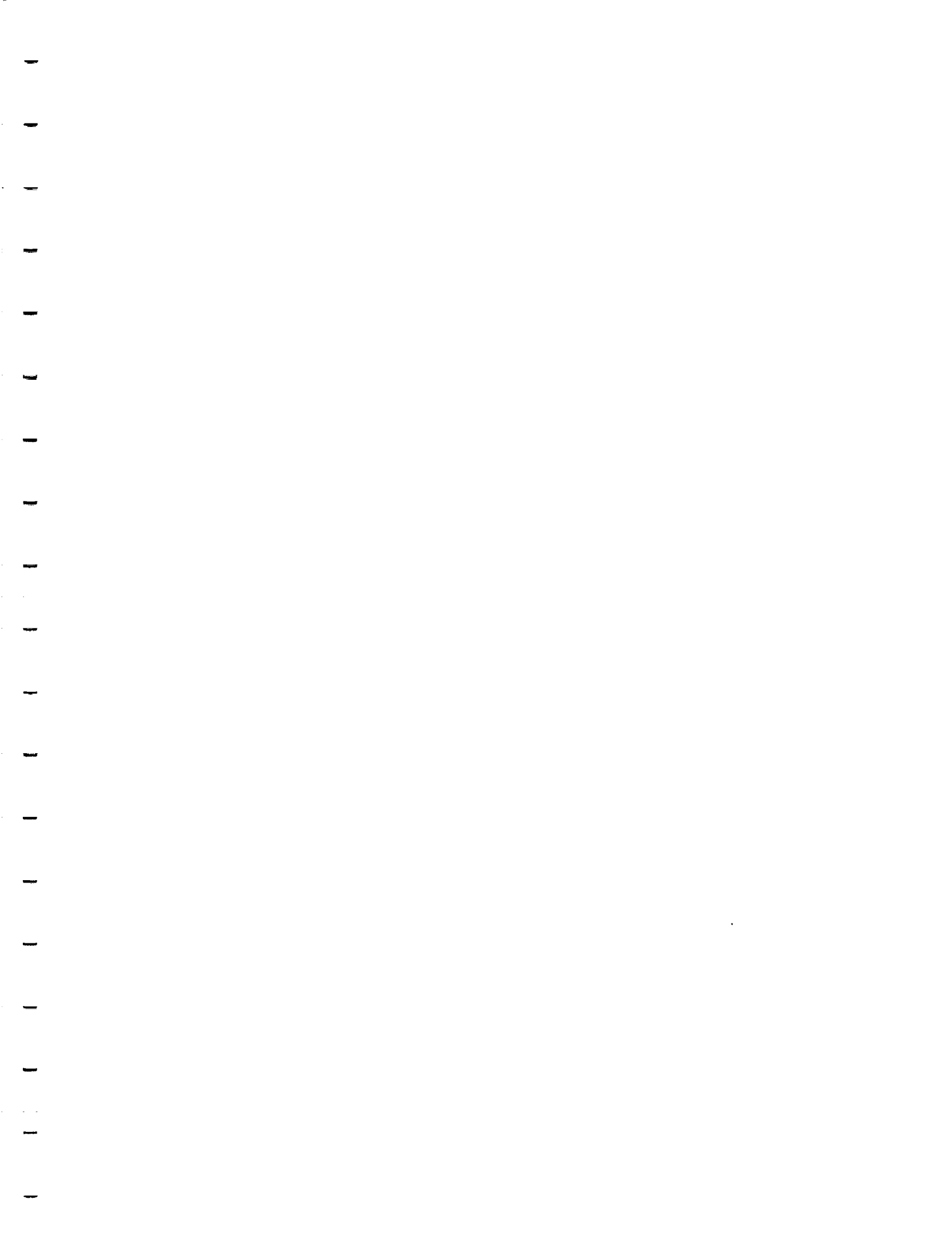


Fig. IV-3



EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON CEREBRAL CIRCULATORY RESPONSES TO HYPERCAPNIA (Mean Values \pm SEM, N = 8)

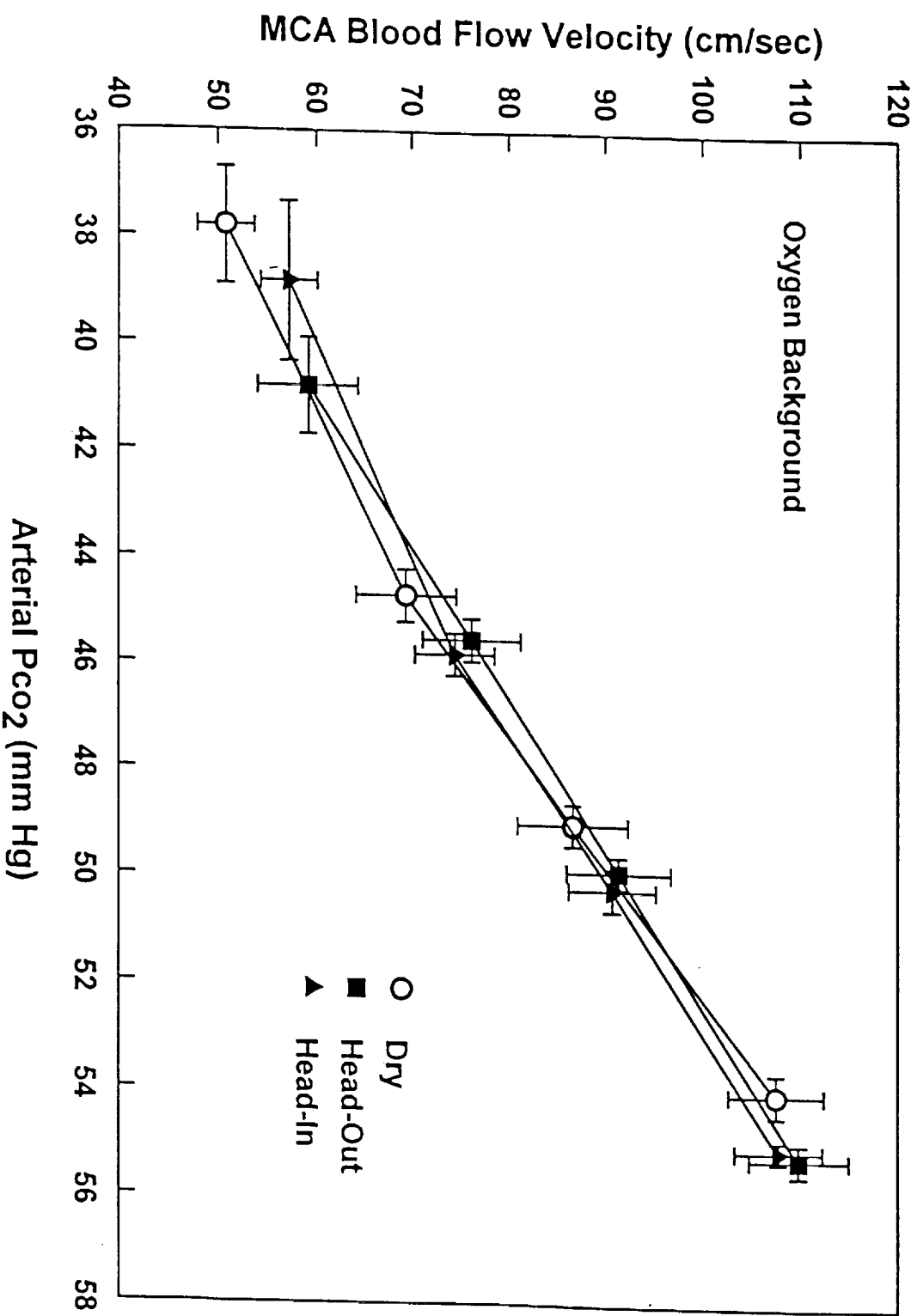


Fig. V-1

EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON VENTILATORY RESPONSES TO HYPERCAPNIA

(Mean Values \pm SEM, N = 8)

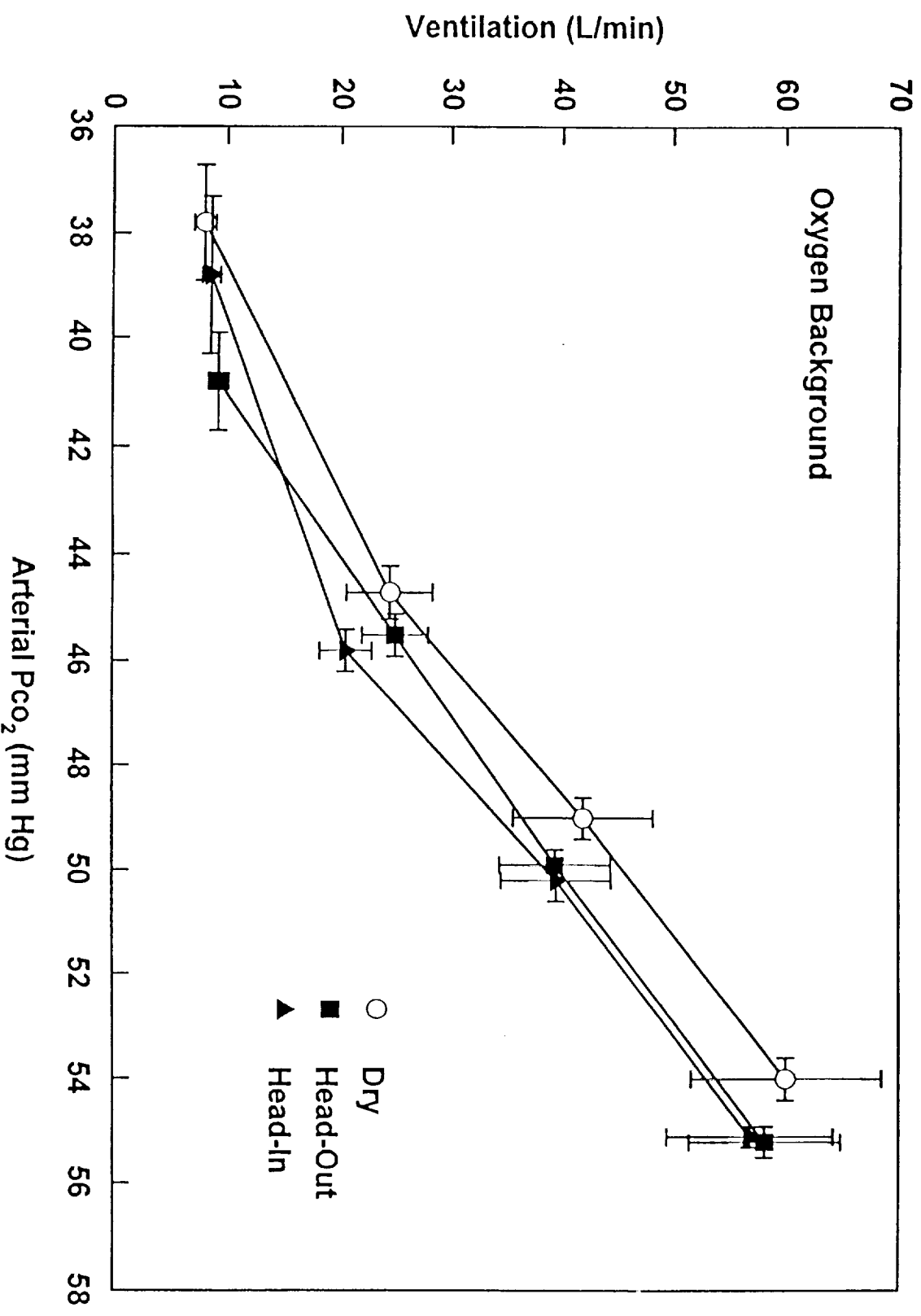


Fig. V-2

EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON HEART RATE RESPONSES TO HYPERCAPNIA (Mean Values \pm SEM, N = 8)

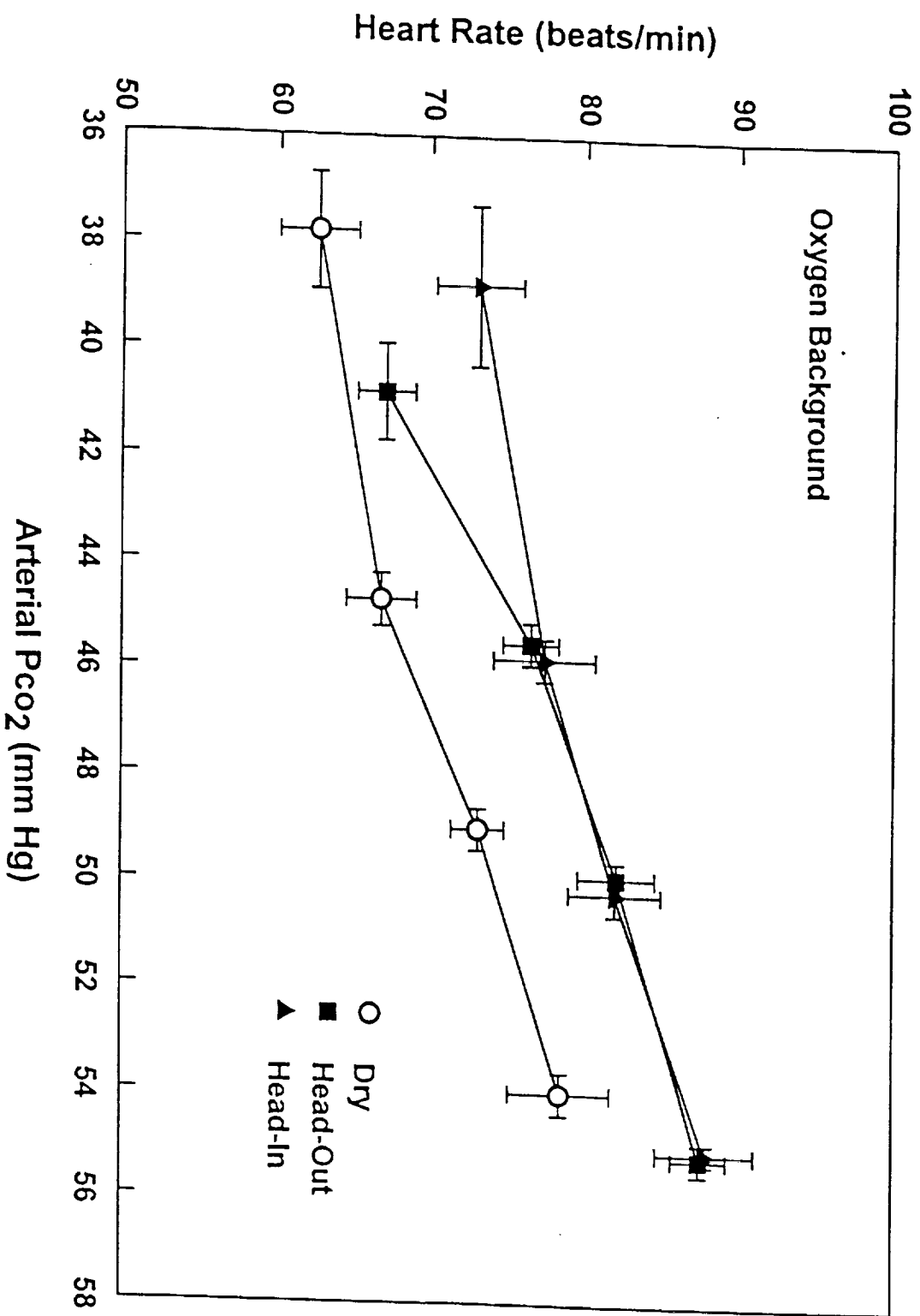


Fig. V-3

EFFECTS OF HEAD-OUT AND TOTAL IMMERSION ON MEAN BLOOD PRESSURE RESPONSES TO HYPERCAPNIA

(Mean Values \pm SEM, N = 8)

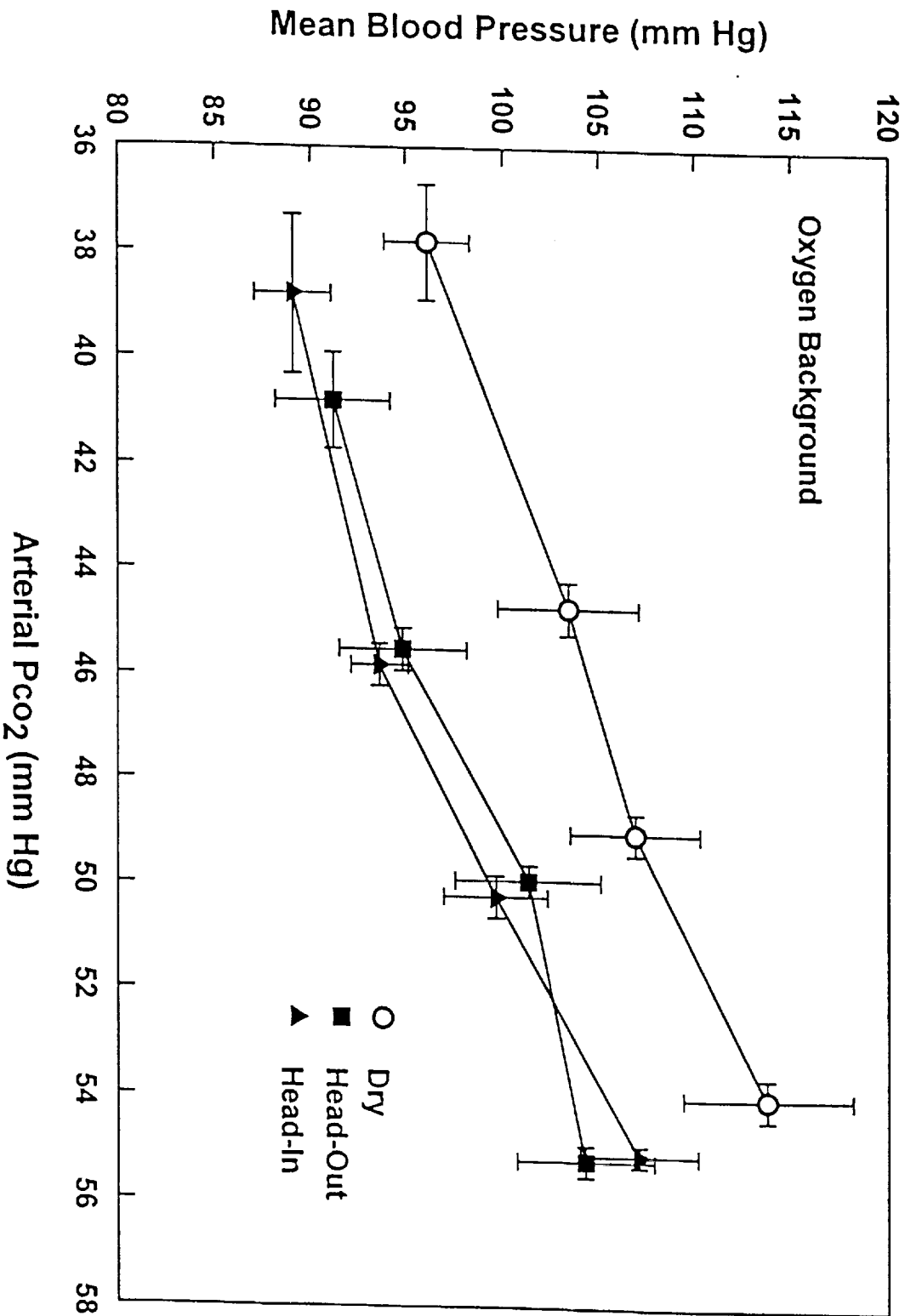
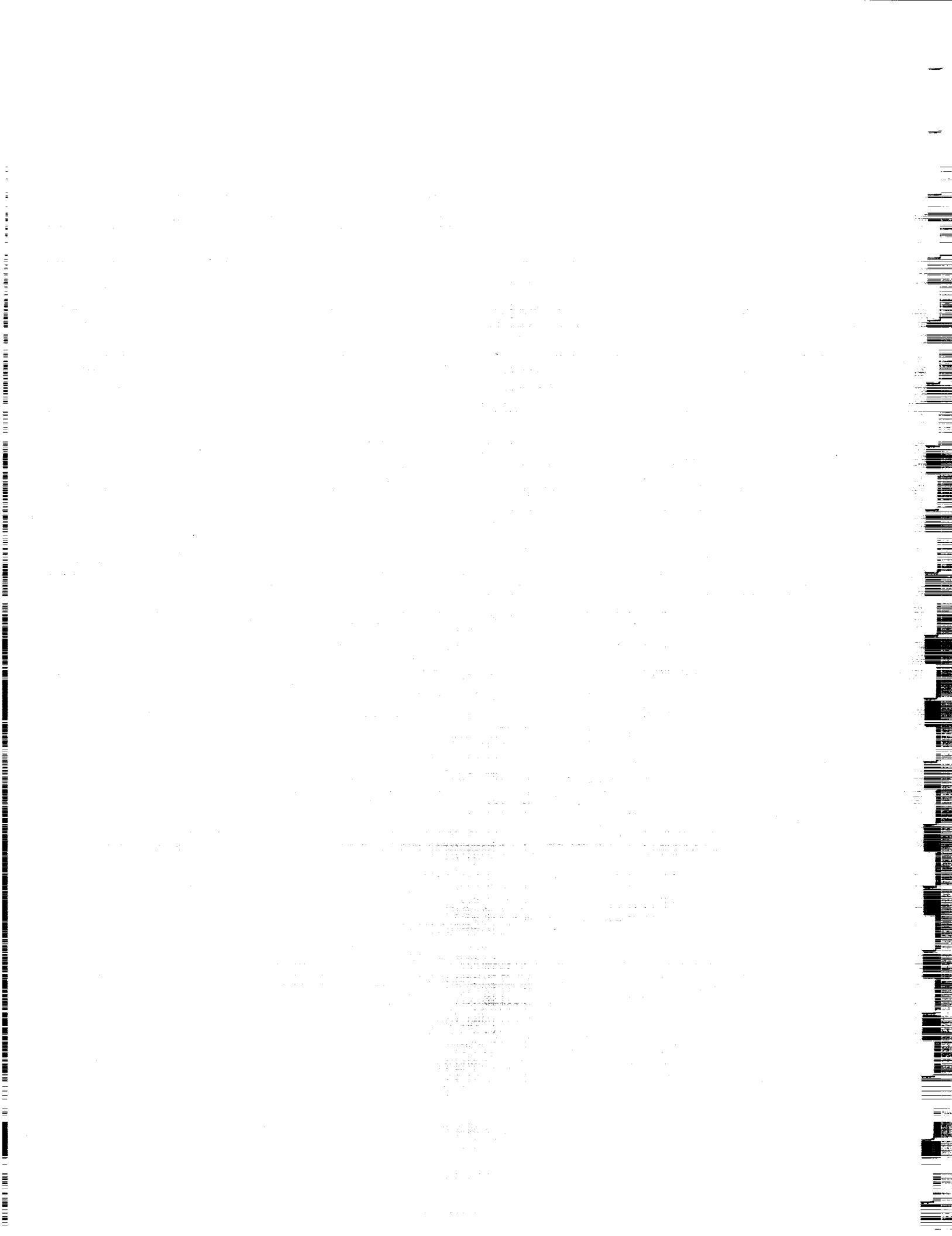
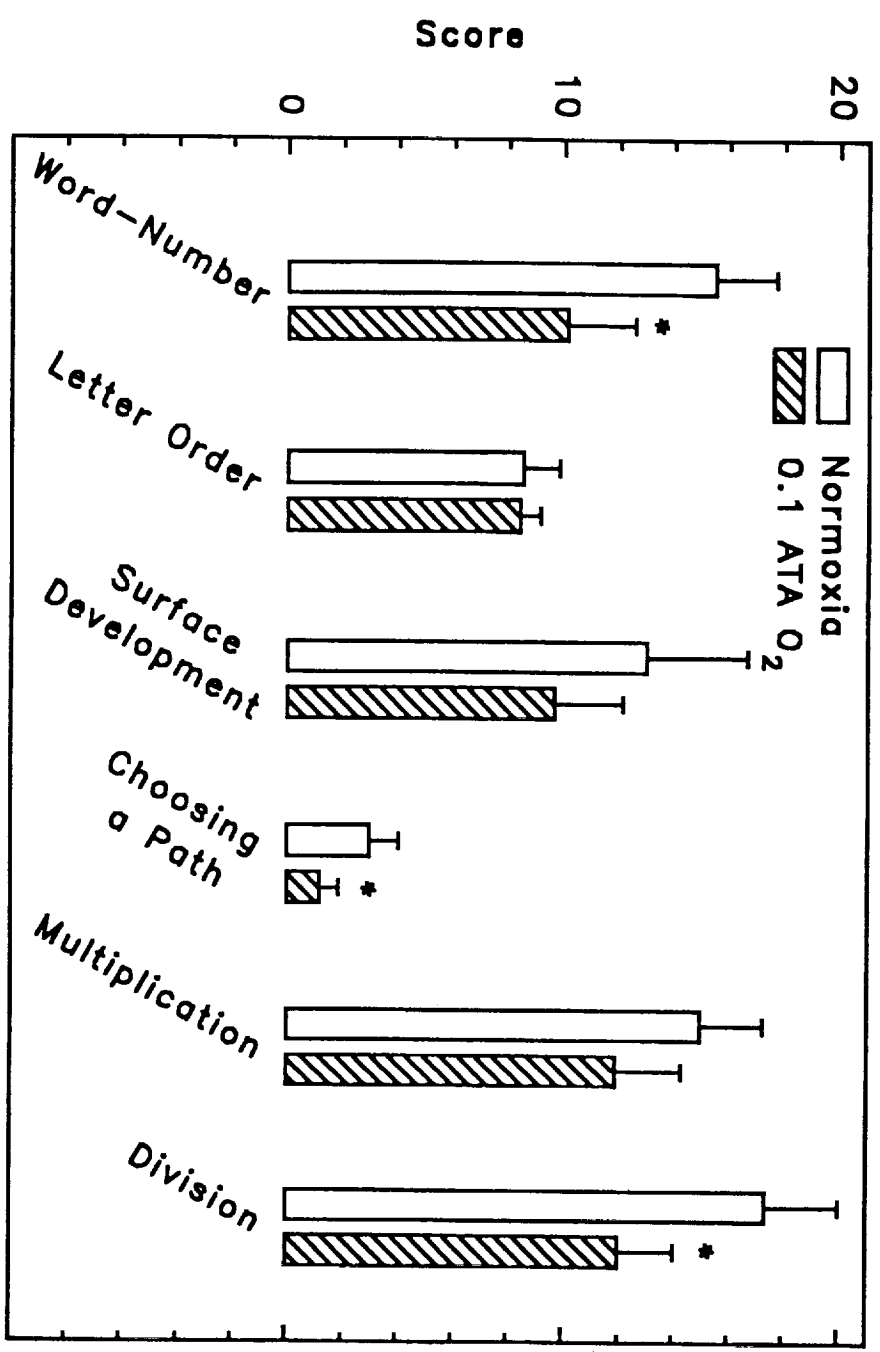


Fig. V-4



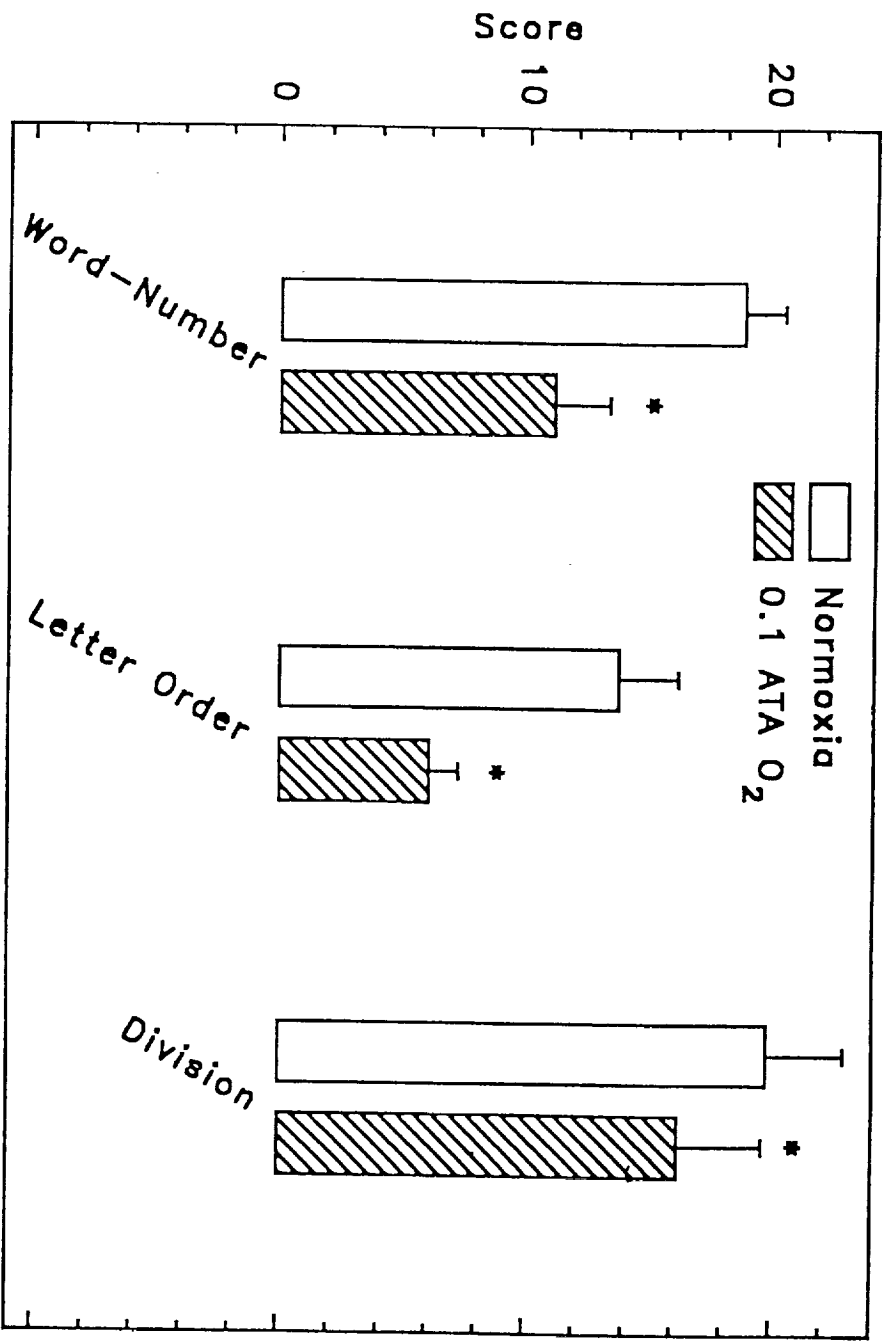
Performance on Six Mental Function Tests in Inexperienced Subjects during Exposure to Hypoxia (Mean \pm SEM, N=8)



* Difference from control value statistically significant, $p \leq 0.05$.

Fig. VI-1

Performance on Three Mental Function Tests during Exposure to Hypoxia (Mean \pm SEM, N=9)



* Difference from control value statistically significant, $p < 0.05$.

Fig. VI-2

ACUTE ADAPTATION TO HYPOXIA (0.10 ATA O₂)

RESTORATION OF NORMOCAPNIA IMPROVES MENTAL PERFORMANCE

(Mean \pm SEM, N=8, * P<0.05)

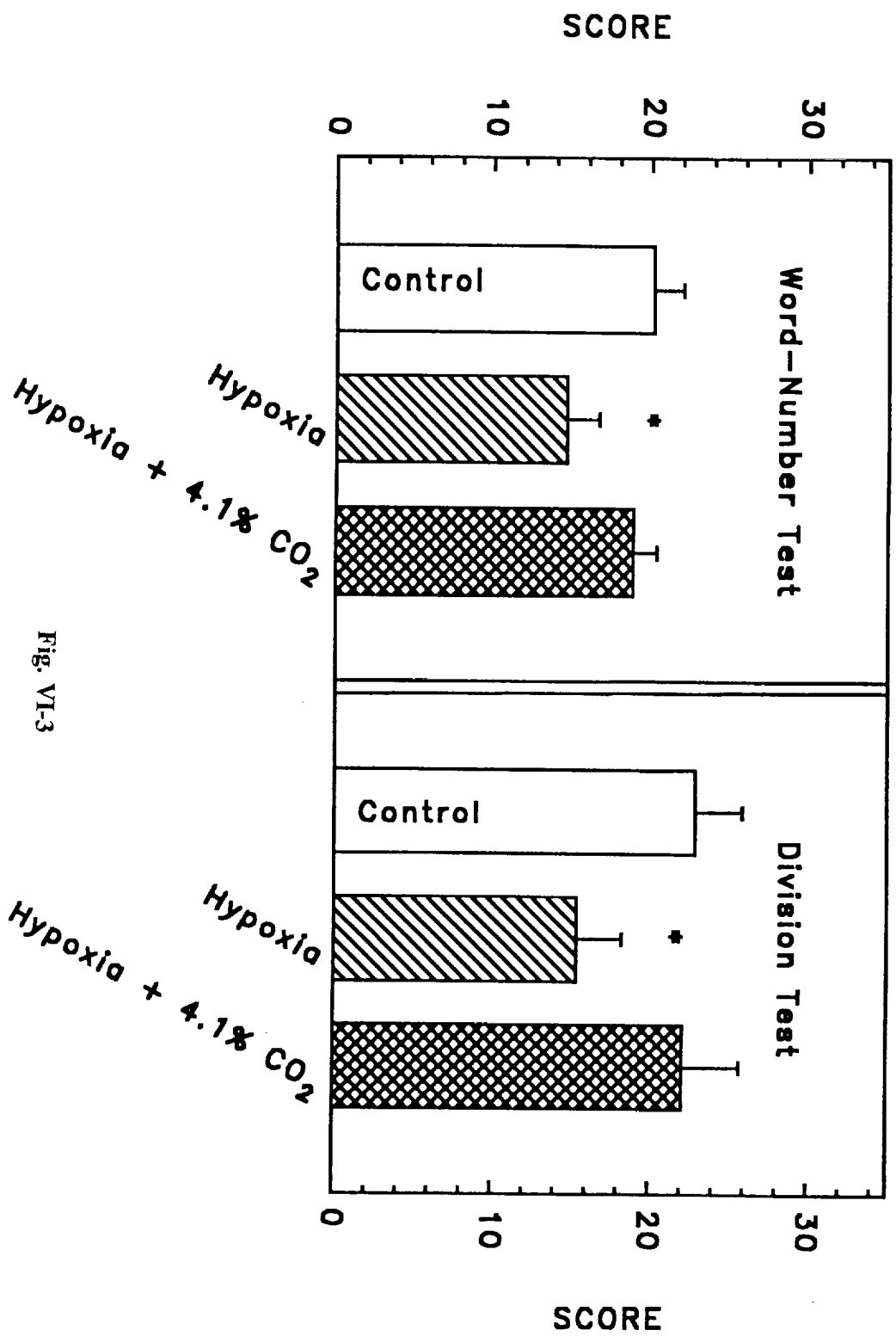
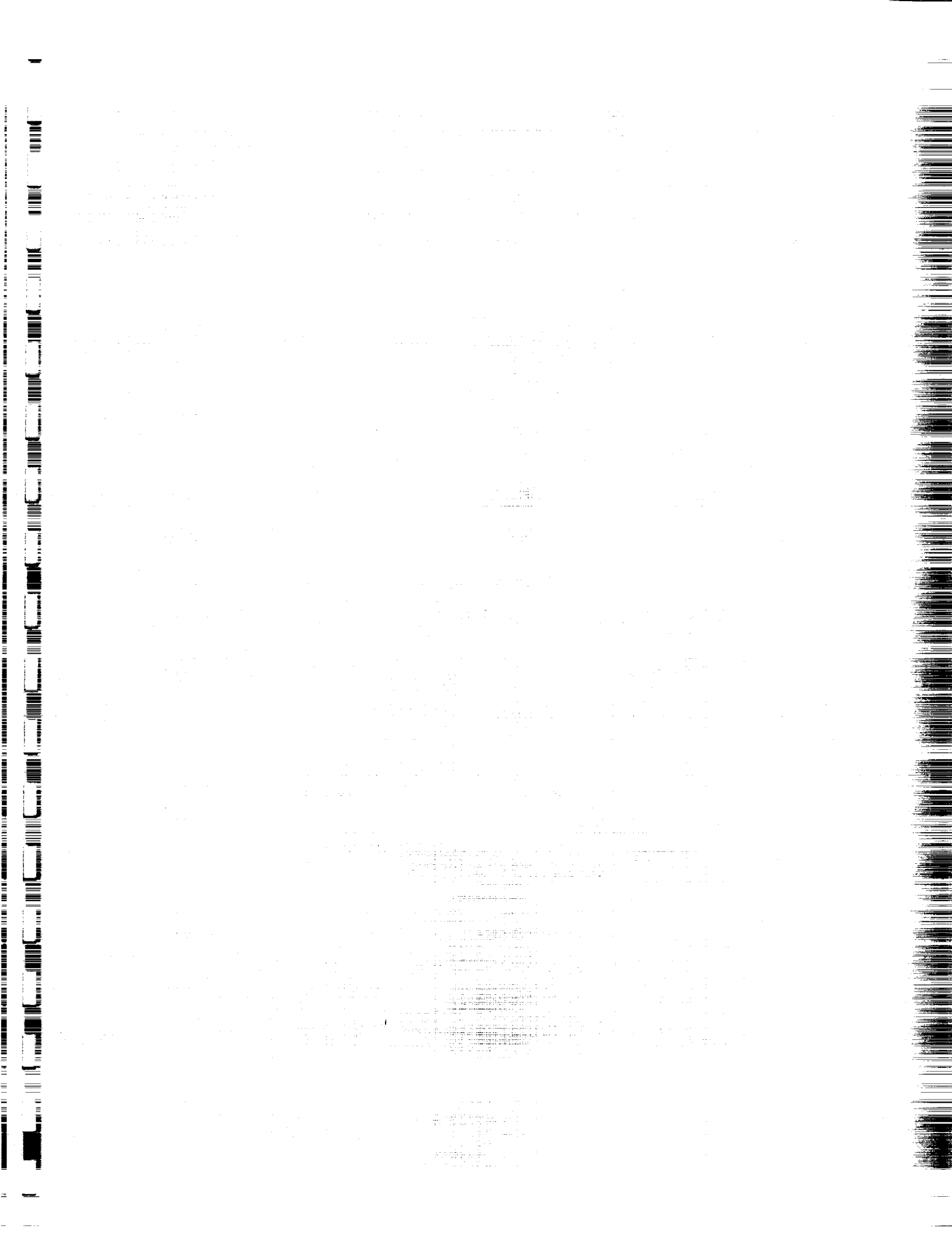


Fig. VI-3



ARTERIAL OXYHEMOGLOBIN SATURATION AND HEART RATE DURING HYPOXIA AND HYPOXIA WITH RESTORED NORMOCAPNIA

(MEAN \pm SEM,  TEST ADMINISTERED)

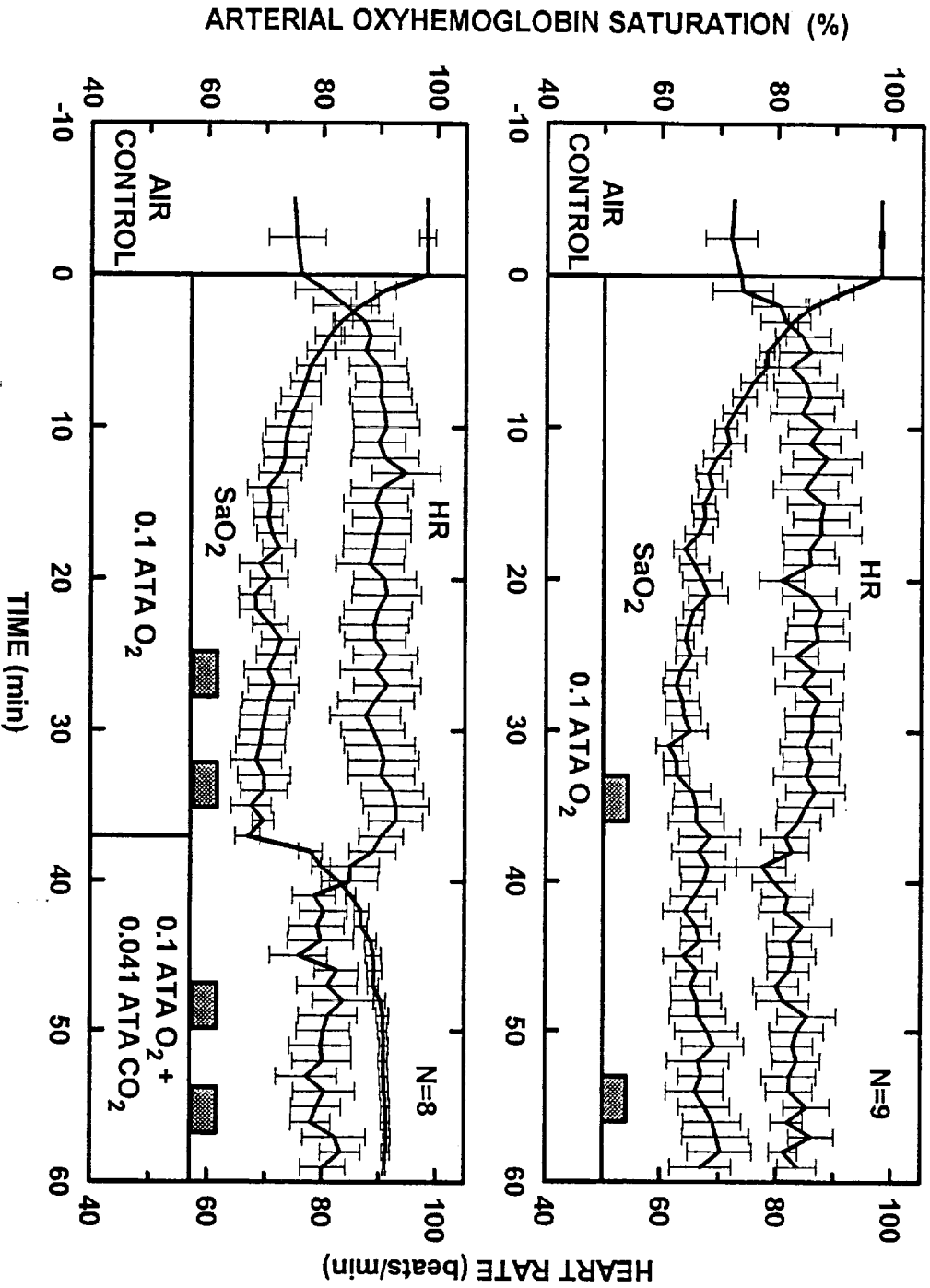


Fig. VII-1

END-TIDAL PO₂ DURING EXPOSURE TO HYPOXIA AND HYPOXIA WITH RESTORED NORMOCAPNIA

(MEAN ± SEM, ■ TEST ADMINISTERED)

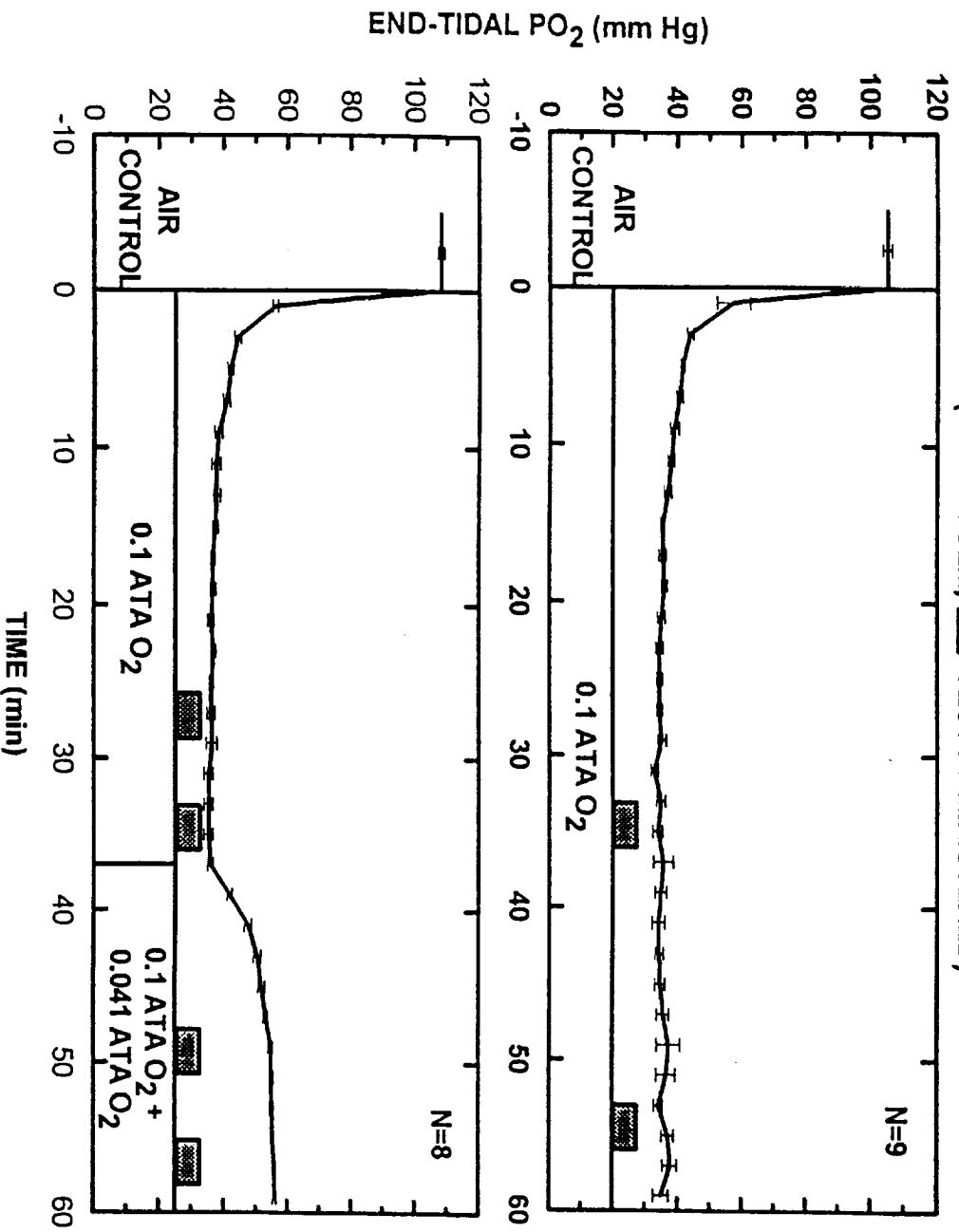


Fig. VII-2

ESTIMATED ARTERIAL PCO₂ DURING EXPOSURE TO HYPOXIA AND HYPOXIA WITH RESTORED NORMOCAPNIA

(MEAN ± SEM, ■ TEST ADMINISTERED)

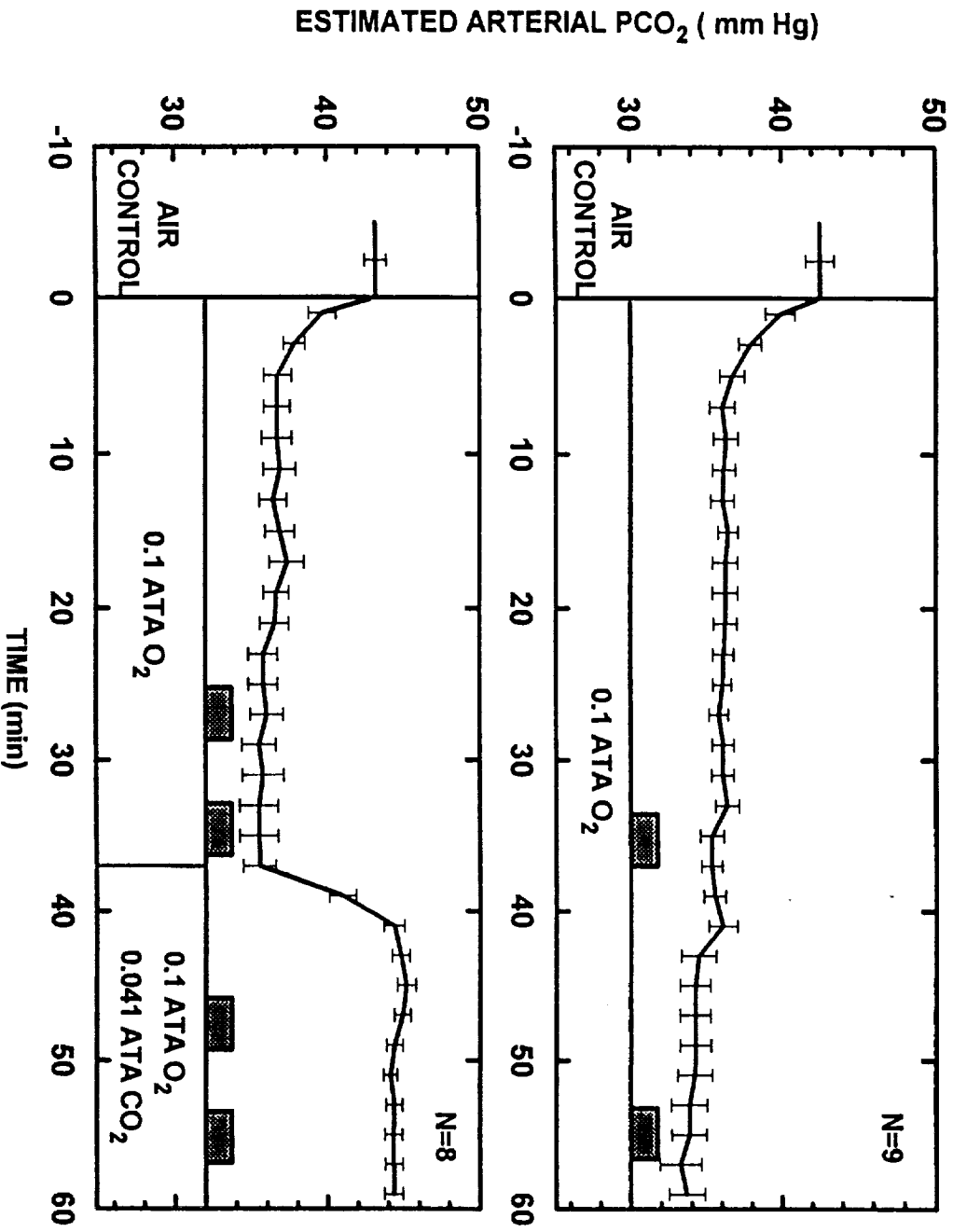


Fig. VII-3

VENTILATION DURING EXPOSURE TO HYPOXIA AND HYPOXIA WITH RESTORED NORMOCAPNIA (MEAN ± SEM, ■ TEST ADMINISTERED)

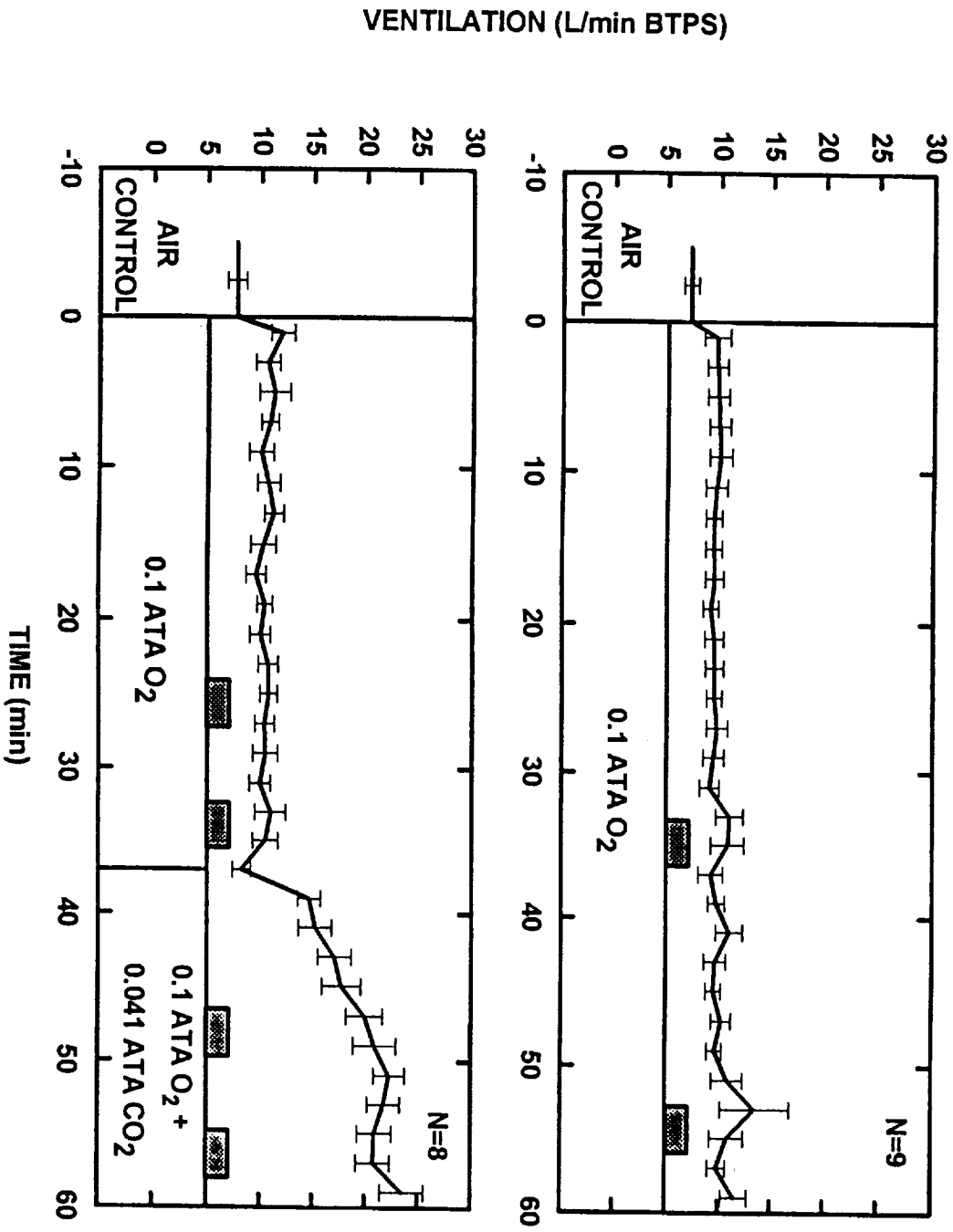
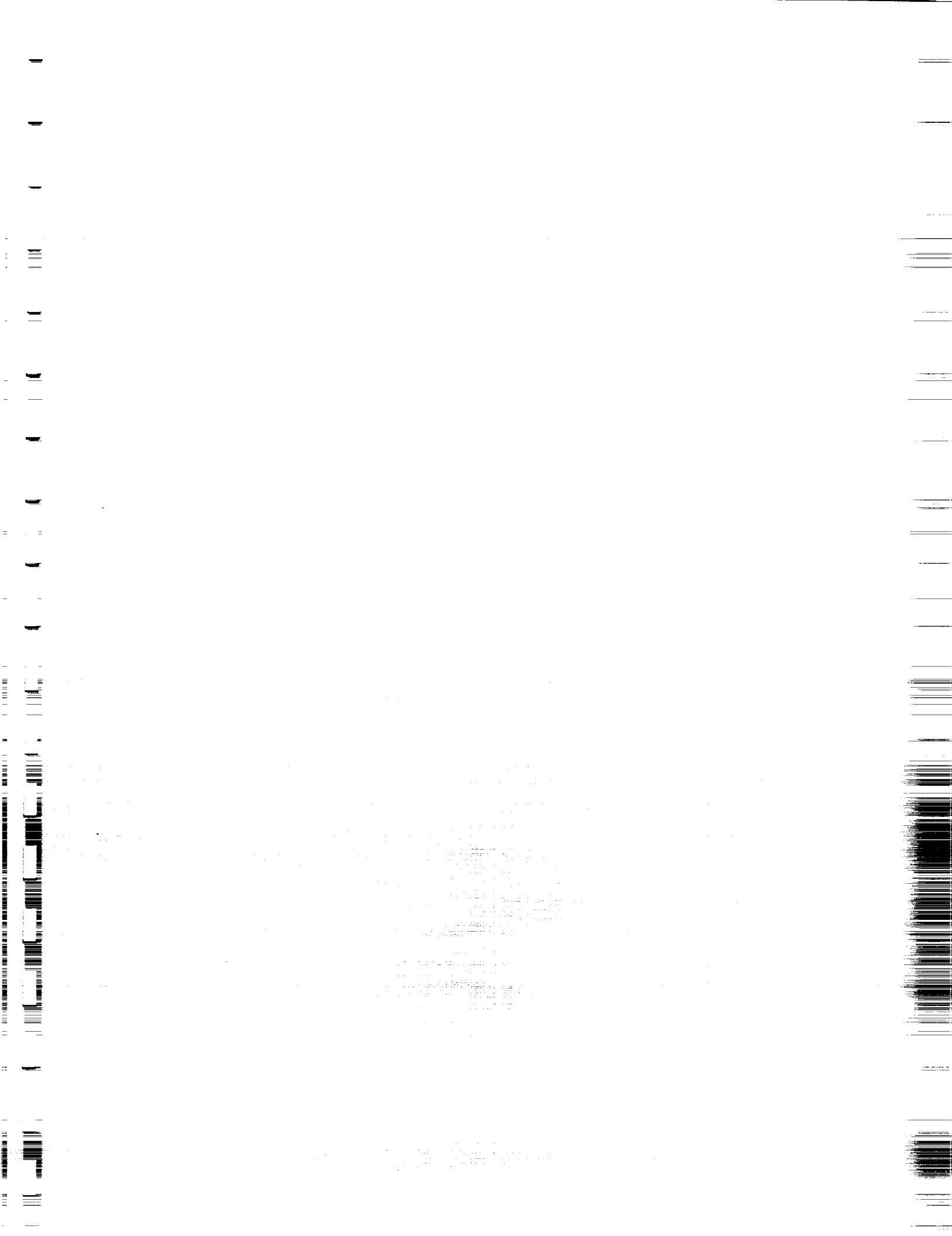


Fig. VII-4



RELATIONSHIP OF ARTERIAL Pco₂ TO RATE OF CO₂ ELIMINATION DURING OXYGEN-EXERCISE EXPOSURE AT 2.0 ATA IN MAN

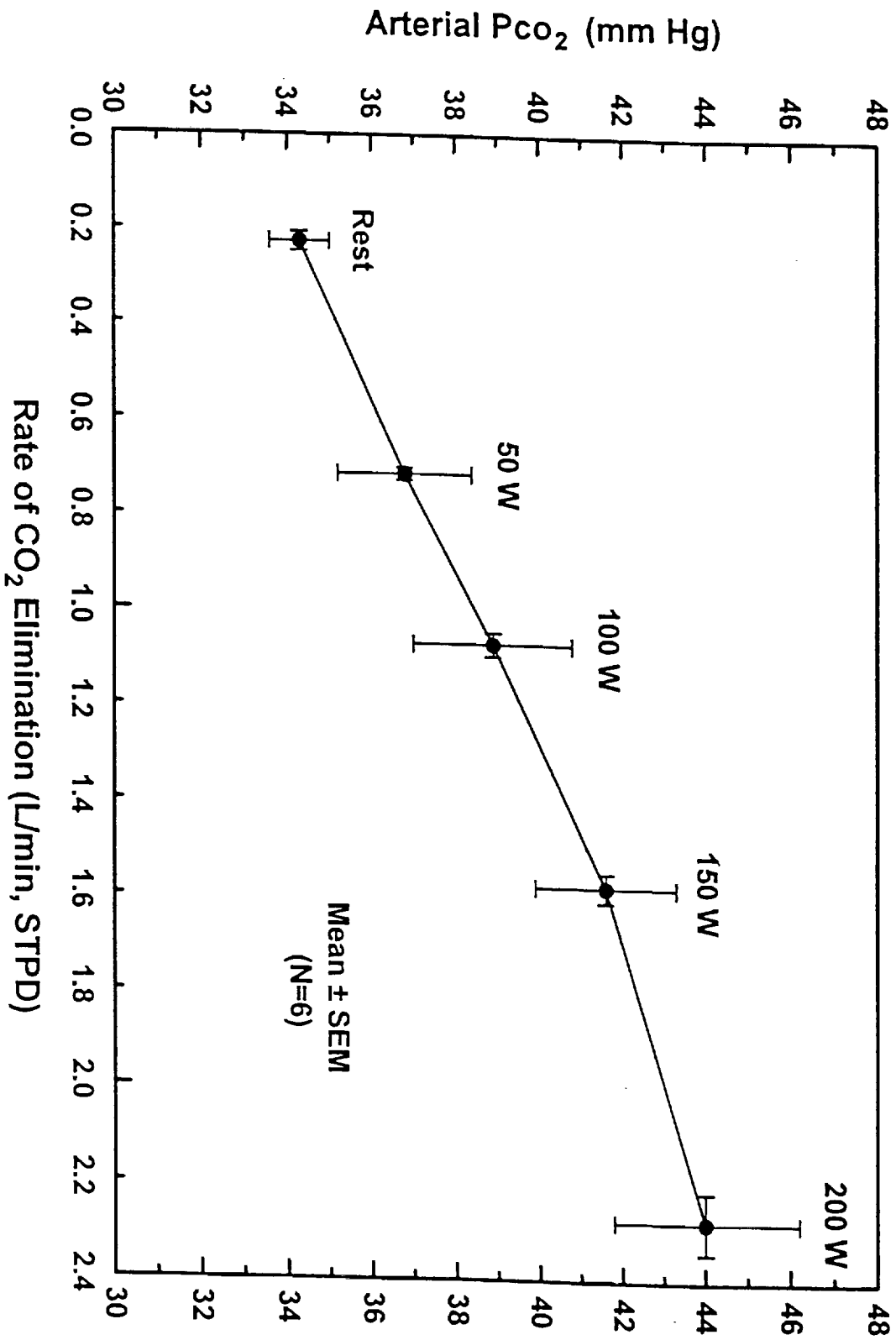


Fig. VIII-1

**PERFORMANCE MEASUREMENT SYSTEM (PMS)
HUMAN PERCEPTUAL, COGNITIVE AND
PSYCHOMOTOR FUNCTIONS**

**PC-DOS VERSION
MARK III-90**

**OPERATING INSTRUCTIONS
AND
MANUAL OF TESTS**

R. Gelfand and C. J. Lambertsen

**EBRDC Report 8.1.91
Environmental Biomedical Research Data Center
Institute For Environmental Medicine
University of Pennsylvania Medical Center
Philadelphia, PA 19104-6068**

TABLE OF CONTENTS

PART I

BACKGROUND AND OPERATING INSTRUCTIONS

INTRODUCTION	3-12
Origin of PMS at the Institute For Environmental Medicine	
Evolution of the PMS	
The PMS Test Library	
Historical review of computer applications to psychometrics	
Current status in computerized human psychometric testing.	
Technical Description of the PMS	
References	
HARDWARE REQUIREMENTS AND INSTALLATION	12-14
The computer	
Default (primary) and secondary video and parallel ports	
Analog-to-digital converter	
Projector	
Power supply/junction box	
Interconnecting PMS hardware	
HARDWARE DIAGNOSTIC SOFTWARE	14-16
The secondary (subject) video (CGAT and CGAC)	
The subject display/response console (PANEL and PANEL2)	
The projector (PROJECT)	
The position control devices-Joysticks (JOYT)	
Calibration of the Joysticks	
TEST SOFTWARE	17-18
Installation	
Early & subsequent versions of Test software for DOS computers	
Running PMS Tests	
CREATING AND RUNNING TEST SEQUENCES: THE EXEC UTILITY	18-25
Installation	
Introduction	
Overview	
Detailed instructions	

PART II

PERFORMANCE MEASUREMENT SYSTEM MANUAL OF PSYCHOLOGICAL TESTS
Dorothy E. Fletcher Dec. 7, 1983

INTRODUCTION

Origin of PMS at the Institute For Environmental Medicine.

Conventional methods of administering a variety of mental function and performance tests is by use of special purpose devices (pencil and paper, electromechanical and electronic apparatus), each specific to an individual test. From about 1960 to 1970, investigators at the University of Pennsylvania's Institute For Environmental Medicine utilized such standard approaches for cognitive, sensory and psychomotor assessment in determining dose-response relationships for narcotic agents and in determining effects of high pressures of inert gases (at-rest and in exercise), both relevant to diving (1-5). Such methods were not convenient for repetitive serial testing, were not applicable to underwater administration, and test scores were not immediately available however, so when an important project arose in which such tests were required in a submerged setting, new methods had to be found.

Early developments of integrated apparatus to overcome the limitations of conventional methods of test item administration and scoring during changing states of stress had also been recognized by investigators elsewhere, leading to development of systems with acronyms PEMCON (6) and SINDBAD (7,8). While these systems provided for serial testing, as hard-wired electromechanical devices they were limited in flexibility and in capability for expansion or modification of test programs.

Evolution of the PMS.

Fortunately, details of the U.S.Navy sponsored SINDBAD were made available to the Institute at the time of a major investigation in 1971. Further development was then undertaken. This was completed for use in another major study in 1974 as a software-based system employing the Digital Equipment Corp. PDP-12 minicomputer for control of test item administration, receipt of subject responses, scoring and computation of descriptive statistics (9, 10).

Further development involved the Subject Console (described below) which was simplified from its original form and fitted with helium-resistant light-emitting diode displays to replace incandescent illuminators. An electronic circuit board was designed and integrated into the console to reduce the number of wires connecting the console to its controller from 64 to 8. Its ability to function underwater, and in high pressure and high altitude environments was preserved.

Later, the system was adapted to the PDP-11 minicomputer, then to an early (Vectorgraphic Co.) microcomputer and more recently to personal computers (IBM PC and compatibles). All tests which formerly required the random access projector for test item presentation can now also be implemented by video graphics.

The PMS Test Library.

The Performance Measurement System Test Library was selected at its inception to permit assessment of a range of the human abilities which relate to performance under varied levels of physiological and psychological stress, or pharmacological effect (11-15). It includes tests of sensory, perceptual, cognitive, memory, psychomotor, system equalization, and motivational abilities and states. This current test library of 30 tests (Table 1), with the Performance Measurement System Hardware has been employed at the U.S. Navy Experimental Diving Unit (16) and at the Norwegian Underwater Technology Center (17-19), as well as at the Institute For Environmental Medicine (10,20).

Historical Review of Computer Application to Psychometrics.

Psychological research has utilized digital computers for many years in a variety of applications. The predominant time course and nature of this utilization closely parallels the progress in computer technology. Early in the sixth decade of this century, physically large mainframe computers of limited capacity (by today's standards) were the norm, and the precursor of the laboratory-type computer was nascent. However, investigators in Psychology were already using computers to a limited extent in such endeavors as on-line experiment control, simulation of mathematical models, artificial intelligence, and data analysis and interpretation (21-24). A primary development in that decade was for the use of computers in personality, aptitude and interest assessment batteries (24,25).

As the decade came to an end, on-line automated psychological evaluation had progressed to human psychometric testing as well as personality/interest assessment. Because of the severe restrictions on the range of tests which time-shared mainframe computers imposed, these initial efforts were based on computer control of special purpose electro-mechanical devices (24-27).

The advent of the laboratory minicomputer opened new opportunities in computer-implemented psychometric testing. As the power of these computers increased and their cost fell, and as manpower costs rose, computerized automation of psychometrics was viewed as an increasingly attractive alternative. Thus, a transition from special-purpose systems to psychometric automation on this new generation of computing machines began in the seventh decade (25). The computerization of psychometrics at this stage was incomplete due to limitations in computer graphics capability and speech synthesizer capabilities. The computers were still limited to controlling traditional graphics display media and audio devices such as random access slide projectors, film loops, and audio tape players.

Table 1
Performance Measurement System Tests
and
Abilities, Skills, and States Tested

Group	Test no.	Test name	Ability, skill, state assumed tested
A	1	Hidden Patterns Test	Flexibility of Closure
	2	Nearer Point Test	Length Estimation
	3	Number Comparison Test	Perceptual Speed
	4	Card Rotations Test	Spatial Ability
B	5	Key Insertion Test	Finger Dexterity
	6	Wrench and Cylinder Test	Manual Dexterity
	7	Stylus Test	Tapping; Aiming
C	8	Visual Reaction Time Test	Reaction Speed
D	9	Time Reproduction Test	Time Estimation
E	10	Word-Number Test	Associative Memory
F	11	Letter Sets Test	Inductive Reasoning
	12	Surface Development Test	Visualization
	13	Choosing a Path Test	Spatial Scanning
G	14	Multiplication Test	Numerical Ability
	15	Addition Test	"
	16	Subtraction Test	"
	17	Division Test	"
H	18	Choice Reaction Time Test	Response Orientation
I	19	Visual Digit Span Test	Span Memory
J	20	Compensatory Control Test	Manual Tracking
	21	Compensatory Coordination Test	Multiple Limb Coord.
	22	Pursuit Control Test	Manual Tracking
	23	Pursuit coordination Test	Multiple Limb Coord.
	24	Static Control Test	Steadiness
K	25	Operations Test	General Reasoning: Numerical Ability
L	26	Multiple Affect Adjective	Anxiety
	27	Check	Depression
	28	List	Hostility
M	29	Sensation Seeking and	Sensation Seeking
	30	Anxiety States Test	Anxiety

As the seventh decade progressed, computer size and price continued to fall. By the end of the decade, table-top microcomputers were becoming common, and the concept of the personal computer had been introduced. Microprocessor based machines, albeit limited in memory capacity, were replacing laboratory systems in some applications, including on-line control and data acquisition/processing in psychometric testing. Computer graphics were still not significantly improved over the older machines, and microprocessor based instrumentation for psychometrics remained dependant on external devices. However many personality and interest assessment systems were fully automated in the seventh and early eighth decades.

As the eighth decade moved on, microcomputers became markedly more powerful (vast increases in speed, memory, and graphics capability) while physical size remained substantially constant and prices fell dramatically. Inexpensive speech synthesizers with impressive capabilities became available. The technology now exists with which to develop entirely computer-based general purpose test systems.

Current Status in Computerized Human Psychometric Testing.

At this time, computerization of mood, personality and interest scales has reached a sophisticated level with programs for on-line administration, analysis, interpretation and report generation commercially available for a number of personal type computers. The development of computer-automated psychometric testing, however, is still in early stages of development (25).

Examination of the scientific literature on psychometric development and utilization shows that current emphasis is on the development of new tests, particularly those utilizing the new and powerful computer graphics capabilities, and on the establishment of proper tolerances and methods for such basic actions as measuring reaction time. There are relatively few descriptions of systems with more complex test batteries for special-purpose applications, and there are not as yet published reports of efforts to develop a broadly useful computer-automated psychometric test system. There is activity relevant to such development, however, in the form of research to establish a system which will allow psychologists to formulate and execute tests in a modular mode which is convenient and easy for them to use without any great understanding of machine-language or even higher-level computer language programming techniques (28-30).

One research area which has received special attention from those interested in computer-automated human psychometric testing is the assessment of impact of environmental pollutants. Several systems with batteries of tests suitable for this application have been developed and described (25). Other areas with activity in computer-automated psychometrics include the screening of pilots and air traffic controllers, assessing the impact on humans of various agents or conditions over time, assessing the impact of extended work periods and fatigue, and evaluating regional cortical dysfunction in patients with psychiatric disorders (25,27). Commercially, one system is available

for conducting psychometrics in remote locations (31). This system has origins in military development programs, and its emphasis has been on portability.

Systems with broader computerized human performance testing capability are one designed for quantitation of neurologic function to aid clinical neurologists (32), and another under development by a U.S. tri-service group to evaluate cognitive performance for chemical defense purposes (33,34). This latter program represents one of the most complex hardware systems described, calling for utilization of the speech synthesizer and a computer network bringing to a central unit the results from many distributed test stations. An expanding test battery is available for this system.

Technical Description of the PMS.

The Investigator and the Subject generally are at different "stations" in use of the PMS. The subject Station consists of the subject Display-Response console, a set of position response sticks ("joysticks"), a random access projector with special computer interface, and a video display, or the video display alone. An eight wire cable connects the Subject Console to a junction box adjacent to the computer at the Investigator station. An eight wire cable is also required to connect the joysticks to the junction box. A coaxial cable connects the video display to the computer. Finally, a 15 wire cable connects the junction box to the computer. If the station is remote from the investigator, audio and possibly video communication facilities will usually be required.

The investigator station consists of an IBM or compatible microcomputer with a hard drive of at least 20 Mb. The computer must have dual video display adapters: the default display to which the computer "boots up" drives the video at the Investigator Station, while the secondary video operates the display at the Subject Station. In addition the computer must have two parallel ports (one for a printer, the other to operate the PMS), an analog-to-digital converter, and the Performance Measurement System Software. Further, there is unit which combines the PMS power supply and a junction box where the system's interconnecting cables come together.

The Subject Display-Response Console is described in detail below.

The Display-Response Console. The display-response console (Fig. 1) is a square one foot on a side, and several inches thick, supported at a comfortable viewing angle. It has recessed illuminated "cells" as follows. At the top are five contiguous variable 7-segment light emitting diode displays, variable zero to nine. Below are three rows of cells with five fixed characters in each, with the digits 1-5 in the upper row, 6-0 in the center row, and the symbols for division, subtraction, a "star", addition, and multiplication in the bottom row. The cells in the bottom row are shaped alternately round and square, while those in the two rows above are all round. All cells are independantly illuminated under computer control. For some tests (Test Groups B,C,D,I,K), this panel is used both for test item display, and for subject response. The panel is used for subject response in all tests except those employing the "joysticks".

Subjects respond to test items by placing a hand-held device containing a magnet in an appropriate cell. The three magnetic devices currently in use are a cylinder (stylus), a key insertion device, and a two piece wrench and cylinder device. The latter two are for psychomotor tests, while the stylus is used in perceptual and cognitive tests.

SUBJECT DISPLAY-RESPONSE CONSOLE

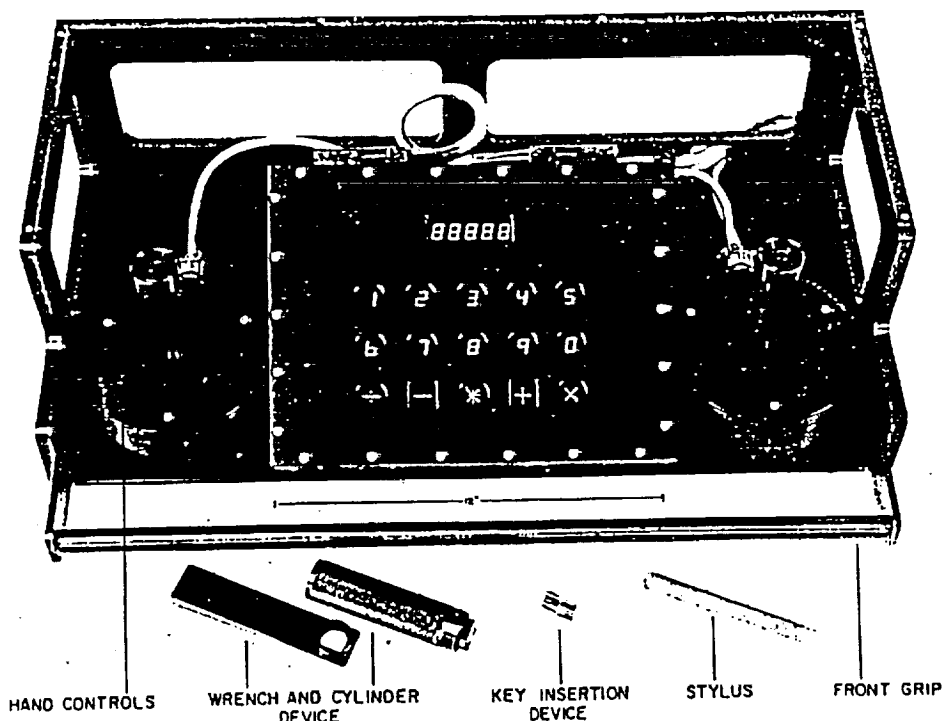


Fig.1. Display and Response components of the Performance Measurement System.

The magnetic devices act on miniature magnetic reed switches positioned beneath each of the lower 15 panel cells. As the magnet reaches about half-way into the cell the reed switch closes, signaling to the computer either a number, a symbol, or a timed event.

The "joysticks" mounted on either side of the panel for tracking tests are isometric (force sensitive) devices with a single push button on each.

Test administration locations need not be limited to having the subject in direct view of the investigator. The remote subject (in another room, in an environmental chamber, underwater) can be in view by means of closed-circuit television.

References.

1. Dickson, J.G. Jr., C.J. Lambertsen, and J.G. Cassils. Quantitation of performance decrements in narcotized man. In: Underwater Physiology IV. C.J. Lambertsen (ed). New York: Academic Press, pp. 449-455, 1971.
2. Bradley, M.E., and J.G. Dickson. The effects of nitrous oxide narcosis on the physiologic and psychologic performance of man at rest and during exercise. In: Underwater Physiology V. C.J. Lambertsen (ed). Bethesda: FASEB, pp. 617-626, 1976.
3. Lambertsen C.J. and W.B. Wright. Multiday exposures of men to high nitrogen pressure and increased airway resistance at natural inspired oxygen tension. (Report of Collaborative Studies-Predictive Studies II). Aerospace Med. 44:821-869.
4. Lambertsen C.J., R. Gelfand, R.E. Peterson, R. Strauss, W.B. Wright, J.G. Dickson, Jr., C. Puglia, and R.W. Hamilton, Jr. Human tolerance to He, Ne, and N₂ at respiratory gas densities equivalent to He-O₂ breathing at depths to 1200, 2000, 3000, 4000 and 5000 feet of sea water. Aviat. Space Environ. Med. 48:843-855, 1977.
5. Hamilton, R.W., Jr. Psychomotor performance in normoxic neon and helium at 37 atmospheres. In: Underwater Physiology V. C.J. Lambertsen (Ed). Bethesda: FASEB, pp. 651-664, 1976.
6. Parker, J.F., Jr., Reilly, R.E., Dillon, R.F., Andrews, T.G. and E.A. Fleishman. Development of a battery of tests for measurement of primary perceptual-motor performance. Arlington, VA: Biotechnology, Inc., 1965.
7. Reilly, R.E., and B.J. Cameron. An integrated measurement system for the study of human performance in the underwater environment. Falls Church, VA: Biotechnology, Inc., 1968.

8. Bain, E.C., III, and T.E. Berghage. Evaluation of SINDBAD tests. U.S. Navy Experimental Diving Unit, Research Report 4-74, 1974.
9. Fletcher, D.E. Rationale and status of the development of a system for investigation of behavior under stress. Report of Workshop II on the Development of Standardized Assessment of Underwater Performance. Bethesda, MD: Undersea Medical Society, Inc., 1977.
10. Fletcher, D.E., C.J. Lambertsen, R. Gelfand, J.M. Clark, and R.E. Peterson. Perceptual, memory, cognitive and psychomotor functions. In: Work Capability and Physiological Effects in He-O₂ Excursions to Pressures of 400-800-1200 and 1600 Feet of Sea Water-Predictive Studies IV. C.J.Lambertsen, R. Gelfand, and J.M. Clark (eds): Institute For Environmental Medicine Report 78-1, Ch. E-10. Philadelphia:University of Pennsylvania, 1978.
11. Cattell, R.B. Abilities: Their structure, growth, and action. Boston. Houghton Mifflin Co., 1971.
12. Guildford, J.P., and R. Hoepfner. The analysis of intelligence. New York: McGraw-Hill Book Co., Inc., 1971.
13. French, J.W. The description of aptitude and achievement tests in terms of rotated factors. Chicago: The University of Chicago Press, 1951.
14. French, J.W., R.B. Ekstrom and L.A. Price. Manual for kit of reference tests for cognitive factors. Princeton: Educational Testing Service, 1963. (Revised 1969).
15. Fleischman, E.A. Development of a behavior taxonomy for describing human tasks: A correlational-experimental approach. J. Appl. Physiol., 51, 1-10, 1967.
16. Curley, M.D., and F.K. Butler, Jr. Visual reaction time performance preceding CNS oxygen Toxicity. Undersea Biomed. Res., 14, 301-310, 1987.
17. Vaernes, R., D. Hammerburg, B. Ellertsen, R. Peterson, and S.J. Tonjum. Central nervous system reactions during heliox and trimix dives to 51 ATA. DeepEx 81. Undersea Biomed. Res. 10(3): 169-192, 1983.
18. Vaernes, R.J., S. Tonjum, A.G. Lindrup and E. Myrseth. Central nervous system reactions during two heliox dives to 36 ATA. Undersea Biomed. Res. 11(1)-Supplement: 39, 1984.

19. Vaernes, R., A. Pasche, K. Segadal, A. Lindrup, and L. Eines. Working in water for 4 hours at 350 msw on heliox: an analysis of diver performance as a function of HPNS, body temperature, lockout time, and number of lockouts. Undersea Biomed. Res. 11(1)-Supplement:23,24, 1984.
20. Fletcher, D.E., R. Gelfand, C.J. Lambertsen, J. Clark, and J. Pisarello. Effects on human abilities of continuous O₂ exposure at 3.0 ATA for 3.5 hours. Undersea Biomed. Res. 11(1)-Supplement: 34, 1984.
21. Green, B. The use of high speed digital computers in studies of form perception. In: Form Discrimination as Related to Military Problems (NRL Publication 561). J.W. Wulfbeck and J.H. Taylor (eds). Washington, D.C.: National Academy of Sciences, 1957.
22. Newell, A., J.C. Shaw, and H.A. Simon. The elements of a theory of human problem solving. Psychology Review. 65:151-166, 1958.
23. Wrigley, C. Electronic computers and psychological research. American Psychologist. 12:501-508, 1957.
24. Fowler, R.D. Landmarks in computer-assisted psychological assessment. Journal of Consulting and Clinical Psychology. 53(6): 748-759, 1985.
25. Bartrum, D. and R. Bayliss. Automated testing.: Past, present and future. J. Occupational Psychology. 57:221-237, 1984.
26. Space, L.G. The computer as psychometrician. Behavior Research Methods and Interpretation. 13:595-606, 1981.
27. Elwood, D.J. Automation of psychological testing. American Psychologist. 24:287-289, 1969.
28. Chute, D.L. MacLaboratory for psychology: General experimental psychology with Apple's Macintosh. Behavior Research Methods, Instruments, and Computers. 18(2):205-209, 1986.
29. Foree, D.D., D.A. Eckerman and S.L. Elliott. M.T.S.: An adaptable microcomputer-based testing system. Behavior Research Methods, Instruments, and Computers. 16(2): 223-229, 1984.
30. Maxwell, K.J. and R.W. Schvaneveldt. The PSYCHLAB programming system: A tool for developing experimental control programs. Behavior Research Methods & Instrumentation. 15(1): 49-56, 1983.
31. Otto, D., S. Baumann, and G. Robinson. Application of a portable microprocessor based system for electrophysiological field testing of neurotoxicity. Neurobehavioral Toxicology and Teratology. 7: 409-414, 1985.

32. Thorne, D.R., S.G. Genser, H.C. Sing, and F.W.Hegge. The Walter Reed Performance Assessment Battery. Neurobehavioral Toxicology and Teratology. 7: 415-418, 1985.
33. Hegge, F.W., D.L. Reeves, D.P. Poole, and D.R. Thorne. Unified tri-service cognitive performance assessment battery (UTC-PAB): Hardware/software design and specification. JWGD3 MILPERF Report.85-2, 1985.
34. Englund, C.E., D.L. Reeves, C.A Shingledecker, D.R. Thorne, K.P.Wilson, and F.W. Hegge. Unified tri-service Cognitive performance assessment battery (UTC-PAB): Design and specification of the battery. JWGD Report. 86-1,1986.

HARDWARE REQUIREMENTS AND INSTALLATION.

The Computer. The PMS requires an IBM PC/XT/AT or fully compatible computer (ordinarily not supplied by ECOSYSTEMS), with 640K memory, at least one floppy disk drive, a hard drive with at least 20 MB capacity and two empty slots. To operate with PMS, the computer requires in addition to the usual configuration of a video card and monitor and parallel port LPT-1 for the printer, a second video card and monitor, a second parallel port configured as LPT-2, and an analog-to-digital converter.

Default (primary) and secondary video and parallel ports. The PMS requires two video cards and two monitors. The primary video (the video which is activated when the computer "boots up") is for use by the investigator. This default video must be in the MDA mode, to avoid memory conflict with the secondary video, for use by the subject, which must be in the CGA mode. The PMS software automatically configures the secondary video when it is called for use in Test Item presentation.

Ecosystems provides the second video card (CompuAdd model CPG-400), which combines CGA graphics with a monochrome composite video output and a parallel port configured as LPT-2. The use of composite video provides for a simple coaxial cable connection between the computer and the subject's display monitor with essentially no practical limit on distance between them. The parallel port LPT-2 provides communication between the PMS hardware and the computer. If your computer already has two parallel ports enabled as LPT-1 and LPT -2, then the parallel port on the CPG-400 must be disabled before installation (see CPG-400 manual).

To install the CPG-400, check its configuration (see manual); then simply plug it into an empty slot in the computer. Then attach the adapter which converts the DB-9 connector to an RCA-type jack.

Analog-to-digital converter. An analog-to-digital converter is employed by the PMS system to convert analog voltages from the four axes of the two "Joysticks" to digital form. The converter employed and supplied by Ecosystems is Industrial Computer Source model ML-8, which is installed "as is" in a second empty slot in the computer.

Follow the ML-8 manual installation instructions, and use the ML-8 software on the diskette supplied to determine if there is an address conflict, and if there is, how to correct it. The 36 pin analog-to-digital converter connector provides for all signals to and from the joysticks.

Projector. The projector supplied by Ecosystems has an interface which provides random access under computer control. Communication to and from the computer is via LPT-2.

Power Supply/Junction Box. The power supplies necessary for operation of the PMS are combined into a single unit with a junction box which contains connectors for interconnection of the PMS hardware.

An adjustable regulated 9 volt DC supply provides power to the printed circuit board inside the subject console. This voltage is reduced to 6.6 volts for the circuitry by an on-board regulator. To minimize power dissipation in this regulator, the output of the 9 volt supply is reduced to an amount just sufficient to permit the on-board regulator to function when the maximum number of LED segments are "lit". This condition is activated by use of the PANEL diagnostic utility described in a later section. The only other power required by PMS is twelve volts at 60 Hz provided to the joysticks from line voltage by a step down transformer.

Interconnecting PMS hardware. Connect the following to the Power Supply/Junction (Interconnection) Box. Take care that cables are connected to their proper destinations.

- The Subject Display Response Console
- The Joysticks
- The Projector
- The single cable from the Splice Box

Connect the following to the computer:

The CCTV monochrome video monitor with composite input to the RCA type jack on the CPG 400 video card with the coaxial cable (BNC connector at one end, RCA phono plug other end).

The two cables from the splice box. The smaller connector to the parallel port LPT-2. The larger connector to the ML-8 analog-to-digital converter. If the joysticks are not part of the system, there may be a single cable direct from the junction box to the computer LPT-2 port.

You can use the diagnostic software as described below to check for proper operation of the subject display/response console, the secondary (subject) video, the projector, and the joystick response devices after you install the software (see TEST SOFTWARE section).

HARDWARE DIAGNOSTIC SOFTWARE.

The secondary (subject) video (CGAT and CGAC). Be sure that the installed default video system is an MDA video card, or a multi-mode video card set to the MDA mode. If it is in a color mode, there will most likely be a video memory conflict with the secondary video. Follow instructions for your own installed system. You may need to acquire an MDA card if your current video can not be set to MDA.

Once the computer has booted with the default video in MDA, use the CGAT utility to test the secondary video.

type CGAT <return>

A sequence of messages will appear on the default monitor and a corresponding sequence of test displays will appear on the secondary monitor. If the computer were to "lock up" this would signal a video memory conflict due to the default video not in MDA mode. The final image requires the presence of a particular .pix file; if it is not present, you will get an error message. This is of no consequence and can be ignored. If the displays appeared as specified, then all PMS Tests which use video for Test Item presentation will operate.

The composite CCTV monitor needs to be checked to determine if the horizontal and vertical axes have equal deflection sensitivities. This can be accomplished using the CGAC utility:

type CGAC <return>

The display which now appears has outer and inner boxes, and horizontal and vertical "ticks". The inner box and the ticks are for use as aids in adjusting vertical size and/or linearity as necessary for equal spacing of the ticks across the screen and for equality to spacing of horizontal tick marks.

The subject display/response console (PANEL and PANEL2). To affirm proper operation of the Subject Console, be sure it is properly connected, and that the PMS Power Supply unit is ON.

type PANEL <return>

The default monitor gives instructions which provide for testing the individual panel LED displays and the corresponding magnetic reed response switches. An additional utility program repeatedly cycles the LED displays. To call this utility,

type PANEL2 <return>

The LED displays should now sequentially and cyclically be turned on and off.

The projector (PROJECT). To test for proper operation of the projector, be sure it is connected to the junction box and power is ON.
type PROJECT <return>

The Projector is exercised automatically, as indicated on the default monitor.

The position control devices - "joysticks" (JOYT). To check operation of the pushbuttons and X-Y axis responses of these devices, use the utility JOYT.

type JOYT <return>

The subject monitor displays a box with a jittering dot within. The jitter is normal. The left or right stick is activated by pressing its corresponding top-mounted button. While a button is held down, the box flashes. The default monitor indicates which stick is active and the dot's relative position within the box. This utility is also employed to calibrate the joystick sensitivities as described in the next section.

Calibration of the joysticks. The joysticks used in the PMS are isometric (force-operated) devices which have been calibrated for deflection sensitivities of 48 oz. from center to each border. Procedures for checking calibration and for recalibration are provided below.

The following tools are required.

1. Spring scale with 1/2 oz. resolution and range to 48 oz. minimum
2. Precision 48 oz. weight
3. Philllips and small slotted head screwdrivers.

Prior to performing the procedure below, check the calibration of the spring scale with the 48 oz. weight.

To check calibration, position the subject video display within easy view of the display/response console. With the system in operation, run the JOYT utility. A box will appear on the video display with a dot at or near the center.

In response to the prompt: CENTER JOYSTICK POSITION? (Y/N), type Y. The software will then compensate for any zero position offset of the dot within the box.

Attach the spring scale to the center of a joystick "knob". The knob is the larger diameter cylinder near the end of the "stick" which encloses the pushbutton. Align the spring perpendicular to the stick and apply a force of 48 oz. along an X or Y axis. The centered dot should then move to the border defined by the box on the screen. Rotate the spring scale 180 degrees and check deflection from center to the opposite border. Any assymetry could be due to nonlinearity of the video monitor, as well as to assymetry within the joysticks, which are of high quality. Check/adjust the monitor linearity with the CGAC utility if the need for this is indicated.

Repeat the above in the other axis of the joystick, and then for the other joystick. It is advisable to repeat this check prior to starting a Test Series.

Should the above procedure indicate that recalibration is necessary, proceed as follows:

To perform calibration, turn power off, and remove 5 machine screws holding each joystick housing to the base plate. Position the controller to be calibrated horizontally (on its side) with its housing near the edge of the horizontal surface (Fig. 2).

Hang the 48 oz. weight from the center of the joystick knob as described above (Fig. 2). Rotate the controller until force is exerted on an X or Y axis.

Use the trim potentiometers in the bottom of the housing to adjust deflection sensitivity (Fig. 2). Adjust the appropriate potentiometer as required until the dot is at the border. Rotate the joystick 180 degrees and check the deflection sensitivity from the center to the other border.

Repeat the above procedure in the other axis and for the other joystick.

Upon completion of these procedures, turn power off and replace the joysticks in their proper positions on the baseplate.

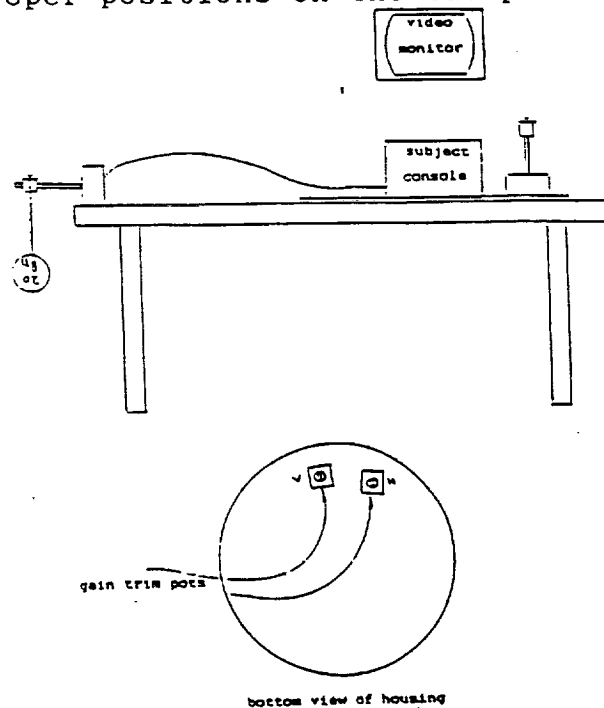


Fig.2. Setup for calibration of joysticks.

TEST SOFTWARE

Installation. The PMS distribution diskettes should be copied in their entirety onto your hard disk under a new subdirectory: \PMS for example. The distribution diskettes should then be put in a safe place.

Early and subsequent versions of Test software for DOS computers. In the initial conversion of the PMS Test software from CPM/Fortran to DOS/Qbasic, the limitation of being able to run only one test at a time was retained. Subsequently, development of a utility (EXEC) was initiated. This utility streamlines the administration of PMS Tests as will be described in the next Section of this manual.

Running PMS Tests. This information is superceded by the EXEC version of PMS software. It is provided here as an introduction to EXEC.

When you "call" a test by typing its group letter name, for example
B <return>

the first screen which appears on the investigator's monitor is a menu for Test Group B. The menu prompts for entry of information such as SITE of the experiment, SUBJECT identification, whether the Subject is Right or Left handed, TRIAL number, TEST identification, FORM of the Test, and ORDER to provide a seed number to the random number generator when it is used. The date and time of day need not be entered by the Investigator -- they are taken from the computer's clock and are placed in the printed output and the data file. It is therefore important at bootup to check and correct if necessary the DOS date and time.

This first screen is tailored to each Test Group, and so will vary slightly accordingly.

Fill in the information as prompted on each line, keying RETURN after each entry. Use the BACKSPACE key to correct erroneous entries, and the ARROW keys to move among lines.

When you key RETURN following the last entry on the screen, a new screen will appear, containing the standard Test Instructions for the particular test. These instructions are identical to those in the original PMS manual as prepared by D.E. Fletcher. They are presented for the convenience of the Investigator, and can be bypassed quickly by "hitting a key". Sometimes, a second page of instructions will appear.

To start the test, "hit any key". This starts a 5-second countdown on the Subject Console (exception: for the "J" Test, countdown appears on the Subject's video monitor) at the end of which the test starts. Two lines are then added to the Investigator's screen: The F1 key will terminate the test immediately, while the F2 key will extend the test to 900 seconds.

When the test is completed, all "nines" will light momentarily, then the entire Subject Console goes dark. The next investigator screen starts with a header containing information from the menu. It scrolls as it is filled with detailed test scoring and stops with a summary of test results.

The Investigator is then queried if a printout of test results is desired. If the response is Yes, a printout of the above is provided to a printer connected at port LPT-1.

Then, the Investigator is queried if the test results are to be stored onto a data file in the current subdirectory. If the response is Yes, all test data information is transferred to disk with an appropriate filename containing the subjects initials, Test identification and trial number with the suffix .dat.

CREATING AND RUNNING TEST SEQUENCES: THE EXEC UTILITY

INSTALLATION.

The PMS distribution diskettes should be copied in their entirety onto your hard disk under a new subdirectory: \PMSEEXEC for example. The distribution diskettes should then be put in a safe place.

To function under the control of EXEC, it has been necessary to modify the software of each PMS Test. These modifications do not in any way alter the way in which the Tests operate.

Each PMS Test tailored to function under control of EXEC can still function as a stand-alone Test as well, independent of the EXEC utility. Pre-EXEC versions of PMS Tests will not run under EXEC. Therefore, EXEC is supplied with a complete set of modified PMS Test software.

INTRODUCTION.

EXEC is a utility program which streamlines the administration of Performance Measurement System (PMS) Tests.

Prior to EXEC, an investigator who wanted to administer a series of PMS Tests to a subject had to manually call each test up and fill in a pre-test menu (test site, subject, experimental conditions, etc.) while the subject waited.

EXEC allows the investigator to create a sequence of tests and fill in their menus prior to their administration. It is easy to use. Its operation will be explained and illustrated below with the aid of reproductions of Print Screen "dumps" as they actually appear during configuration of an EXEC batch file.

OVERVIEW.

EXEC has two parts, as described below, which require investigator responses to on-screen prompts. This information is saved in a "batch file" which can be modified, used to create other batch files, and ultimately used to automate the administration of a test sequence to the subject, all within the EXEC program.

Part I. Entry/Modification of Test Sequence and Menu Fill-In. In this stage:

1. A source batch file is created and selected for editing.
2. A destination (runtime) batch file name is selected (where the results will be saved with this filename).
3. The Test sequence is entered.
4. Answers to menu questions are entered or modified. Answers can be left blank if information is not yet available.

When all menus have been completed, the batch file can be saved to the previously named destination file. This file can be retrieved and modified (by repeated calls to Stage I).

Part II. Using a Batch File to automate Test Administration to the Subject. In this stage:

1. A previously created batch file is called by the investigator.
2. Automated Test administration is initiated by the investigator. EXEC calls the Tests in the order specified above. If the investigator wishes to change or complete any menu items previously left blank prior to Test administration, then Part I should be invoked before Part II. Any unanswered menu questions remaining at the execution of Part II can be answered during run time of each Test.

DETAILED INSTRUCTIONS.

It is important to know at this point that under EXEC all Batch files are saved to the default drive/directory with the suffix .EXL. Test result files are saved with the suffix .RES. The suffixes .DAT and .PIX are reserved for data and video files necessary for the PMS .EXE files to function. Thus, at the end of a test or experiment session, the appropriate commands with the *.RES suffix can be used to transfer all test results to a subdirectory (\RES) for example) on the hard disk, to diskettes for long term storage, to print out the results, and to delete them.

ORIGINAL PAGE IS
OF POOR QUALITY

Starting EXEC. EXEC.EXE is the name of the executable file which comprises the Master Program. To start it, simply type exec after the DOS prompt.

The first screen which appears is as shown in Fig. 3. At this point you can Edit (create or modify) a batch file (of PMS Tests) by selecting option E, you can Run an existing batch file created previously by selecting R, or you can Quit (exit) EXEC and return to DOS by selecting Q.

If you select Edit, you can either assemble an entirely new group of PMS Tests to be sequentially administered, or you can modify a pre-existing batch file. If you select Run, a batch file will be executed (Tests will be administered).

Assembling the Group of Tests into a Batch File in Advance of Experiment. Fig. 4 shows the screen which appears when Edit is selected and the filename BCF has been assigned as both the Source Batch file and The Runtime Batch file. Since there is no file with this name in the default drive, their status is listed as "New".

To create a Source batch file, the first step is to select (by letter) from the PMS Test Library those Test Groups which contain the Tests to be administered, in the order of their administration. Up to 16 Tests can be run in sequence in the order selected here. As an example, three Test Groups have been selected (Fig. 5). After each Test Group identification letter, type <return>. After the final Test has been chosen, either depress the ESC key or keep typing <return> until the cursor passes 16. The next screen which appears is the menu for the first of the Tests selected as in Figure 6.

Filling-in the Menus. For each Test selected above, and in the order selected, the menu specific to that test will appear on the monitor (Figs 6-8). The process of filling in the menu answers will be illustrated by examples below.

-----SPECIAL FEATURE-----
USE OF "DITTO". In the upper right corner of each menu screen (e.g. Fig.4) the following instruction appears:

Use > to ditto

This is a page-to-page ditto (repeat), not the conventional "line-to-line" operator. It can be used to fill in corresponding lines on all succeeding menus, saving the investigator from repetitively entering the same information on successive ones. It is important to remember though that "ditto" functions only in the Edit mode, but not in the Run mode. It is thus advantageous to fill-in as much information in the Edit mode as is possible.

To use it, enter the information which is to be "dittoed". Then, instead of entering <return>, type "shift period" which sends the code for > representing the "ditto" operation.

Test Option Selection in Creating Menus.

Use > to copy an entry to all following menus.
Use <RETURN>, up & down arrows to move among questions.
Use left & right arrows, home, & end keys to move within a question.

NOTE. Not all options listed below appear on all menus.

Site. This can be used to identify location of the experiment: geographic, room, chamber, etc. Can not be left blank, use dummy character if necessary.

Subject. Fill with three characters (numbers or initials) to identify the subject. Can not be left blank, use dummy character if necessary.

Hand. Use R or L according to the subject's handedness. Default entry is R.

Trial. Use a 3-digit number, alphabetic, or alphanumeric. Default entry is 001.

Test. The alphabetic Test Group previously selected is shown here. It is necessary to enter the numeric (see IMPORTANT NOTE below) which corresponds to the specific Test within the Group which is to be used. In this example, it can be B1, B2, or B3 (Fig. 6), it is C1 (no other option) in Fig. 7, and it is F1 (No Other Option) in Fig. 8.

-----IMPORTANT NOTE-----

With the introduction of EXEC, numeric Test designations have been modified to provide for a two character alphanumeric identification of each Test for use within filenames. This also provides flexibility in future expansion of the Test Library. The original and corresponding new Test designations are given in Table II. Alphabetic Test Group identifications have not been changed. In the new scheme, within each Test Group individual Tests start with the number 1 and can go from 1 to 9. Thus, up to 234 specific Tests can be identified using only two characters.

Form. Some (not all) Tests have internal options, designated as "forms". The desired Test Form is entered here. The default value is 1.

Order. Some Tests or Test Forms incorporate a pseudo-random number generator to randomize some aspect of the Test Item administration. "Order" is a 4-digit number which initializes or seeds (selects the "starting point") of the number generator. To avoid repetition in the order of Test Item administration to an individual subject, you can select a different "order" before each experiment run. It may be desirable to repeat the "order" at corresponding stages of an experiment across a subject population so that all subjects are exposed to the same level of difficulty of Test Items. If a seed number is not

experiment across a subject population so that all subjects are exposed to the same level of difficulty of Test Items. If a seed number is not entered, and the default "R" is left in place, then the software selects a seed number at random.

Subject Display. Some Tests can use either a random-access projector (PROJ) or a video display (CGA) to present Test Items to the subject. The selection is made here; the default is CGA.

The investigator will be aware of differences between Test Item presentation by projector slides and video display. In many cases the differences are not dramatic. The investigator himself must judge which method to use.

Instruction Slide. For tests using PROJ or CGA, an INSTRUCTION SLIDE is displayed to the subject at the same time the INSTRUCTIONS appear on the investigator's video display. The default is for the instruction slide to be loaded into slot 79 in the slide carousel, or for the corresponding .pix image to be called up in the CGA version.

Starting Slide and # of slides. These two options allow the investigator to define the lower and upper bounds of a subgroup of all available test forms to be used in a test session. When the Test Form specifies sequential administration, Test Items are presented accordingly. When a random order is specified, the slides within the same subgroup are presented in a randomized fashion.

Experimental Conditions and Purpose of Test. Spaces are provided for a description of experiment circumstances. They can be left blank. This part of the menu is recalled at the conclusion of a Test run for retrospective annotation by the investigator.

Allow pauses during test ? (Yes, No). Selecting Y means that all the pauses or stops incorporated in the Tests remain operative. This option can be used when training subjects, or if for any reason the Investigator wishes control Test progression by "hitting any key" each time the Test program comes to a stop. Such stops typically occur when Test instructions appear on the Investigator's video, before the Test countdown is to begin, and at the end of each Test in the sequence of Tests

Selection of N causes all pauses within the Test to be ignored. As a consequence, once the concatenated Tests are started, they run one after the other in sequence without Investigator intervention. Individual tests are separated by the time required for the program to generate Test scores, compute descriptive statistics and then give the usual 5-second countdown before each Test. An exception to the above occurs with the J group of Tracking Tests. Here, the pauses for Investigator-initiated software calibration of the "joysticks", and for initiation of the 10-second practice session remain, even if Y is selected.

TABLE II

PMS TEST DESIGNATION: CONVERSION TO TWO CHARACTER IDENTIFICATION

<u>ORIGINAL</u> Group Number		<u>CURRENT</u> Group Number	
A	1	A	1
	2		2
	3		3
	4		4
B	5	B	1
	6		2
	7		3
C	8	C	1
D	9	D	1
E	10	E	1
F	11	F	1
	12		2
	13		3
G	14	G	1
	15		2
	16		3
	17		4
H	18		1
I	19	I	1
J	20	J	1
	21		2
	22		3
	23		4
	24		5
K	25	K	1
L	26	L	1
	27		2
	28		3
M	29	M	1
	30		2

Save results after test ? (Yes, Undecided, No). If Y is selected, Test results are automatically saved to the default drive as each Test in the sequence is completed. The filename automatically assigned from information in the menu is as follows:

ABCDEFGH.RES

where:-- ABC are subject identification
-- DE are Test identification, e.g. Test B1
-- FGH are trial number
-- suffix .RES is reserved for test result filename

The Undecided Option. This option (U) is made available for situations in which the decision whether or not to save results is to be made "on the spot". If this option is selected, then the batch program will stop when results are displayed on the investigator's video. Program execution resumes when a Y or N entry is made.

If N is selected, test results are not saved. This selection is made available for use during training and practice sessions if the investigator does not wish to archive those results.

Print results after test ? (Yes, Undecided, No). If Yes is entered, the Test results are printed immediately after each test is completed. The same considerations apply to the Undecided and No options as above.

Completion of Test Option Selection. As menu entries for each Test are completed, the menu appropriate to the Test next in order of selection is presented (Figs. 6, 7, 8).

After the final entry on the menu of the last Test selected has been completed, you are prompted :

Save runtime batch file XXXXXXX (Y/N)

If you enter Y, the batch file just created is saved to the default drive/directory with the "runtime" filename (and with suffix .EXL) selected at the initiation of EXEC. This batch file can be used as many times as needed for the same subject in serial runs on a given day, for the same subject on different days, for multiple subjects on a given day or over time, as appropriate.

Each time the batch file is used, its menus can be slightly modified to alter Subject, Trial, etc. The same batch file might be employed under varying circumstances e.g. differing experimental conditions or for different purposes. If desired, each specific runtime file used can be saved by naming them (for example) as BCF1, BCF2, and so on.

Running a Batch File. In preparation for an actual experiment run, enter EXEC and at the prompt (Fig. 1) select R. Next, when prompted, enter the file name as in Fig. 9. This will be the batch file saved as a "runtime file" as described above. The next screen which appears has four options (Fig. 10), which are self explanatory.

Printout of Test Results. Figure 11 show the ancillary information which is printed preceding the actual test results and scoring. Note that in addition to the information in the menus in the column on the left, the right hand column contains the Date and Time of Day (at computer bootup, or before running tests, be sure to check/update the computer clock's date and time). In addition, the time taken to review instructions, and the duration of the Test are also provided here.

More on Repetitive use of a Source File. When a Batch file is created with a Source file name such as BCF, you can use this file name as the Runtime file name as well. The Batch file will be saved as BCF.EXL. This can be a master file which is retained and updated each time it is used.

Each time Batch file BCF.EXL is used (run), it would be named as the Source file. The first time used, the corresponding Runtime file could be named as BCF1; the second time, BCF2, In this manner of operation, all Runtime.EXL files would be saved, along with the BCF Source file.

As an alternative, if each Runtime.EXL file were not to be saved, BCF1 could be repeatedly used as the Runtime filename. In this case, only Source file BCF and a single (most recently used) Runtime file BCF1 would be retained on disk.

For help: R. Gelfand
 (215) 476-7892

 A. Slater
 (215) 825-6000

ORIGINAL PAGE IS
OF POOR QUALITY