ABSTRACTS BIOMEDICAL RESEARCH AND UNDERWATER BREATHING APPARATUS EVALUATION DIVES 10 TO 1600 FEET APRIL 1-2, 1974 CONFERENCE

L. W. Raymond, et al

Navy Experimental Diving Unit
Washington, D. C.

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Results and tentative conclusions of biomedical research and underwater breathing apparatus evaluation performed in a series of dives ranging from 10 to 1600 feet are presented in abstract form.

**Underwater Breathing Apparatus**

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ABSTRACTS
BIOMEDICAL RESEARCH AND UNDERWATER BREATHING APPARATUS EVALUATION DIVES 10 TO 1600 FEET
APRIL 1-2, 1974 CONFERENCE

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ABSTRACT

Results and tentative conclusions of biomedical research and underwater breathing apparatus evaluation performed in a series of dives ranging from 10 to 1600 feet are presented in abstract form.
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INTRODUCTION

In 1972 and 1973, under the direction of the U.S. Navy Supervisor of Diving, CDR J. J. Coleman, USN, the Navy Experimental Diving Unit and the Naval Medical Research Institute joined in cooperative research studies during a dive series ranging in depth from 10 to 1600 ft. The dives from 10 to 1000 ft were performed at the Experimental Diving Unit, then the final dive to 1600 ft was conducted at the Taylor Diving and Salvage Co., New Orleans, Louisiana.

The results and tentative conclusions of this research were presented for general discussion at a Working Conference at the Naval Medical Research Institute on April 1-2, 1974. This report is a collection of the abstracts of the subjects presented.

This research was a team effort of EDU and NMRI divers, diving officers, engineers, and technicians, plus both research and clinical medical personnel. The Bureau of Medicine and Surgery helped support the studies. The Naval Hospital Bethesda lent physicians and provided laboratory support. The Taylor Diving and Salvage Company operated their excellent hyperbaric chamber complex for the 1600 ft dive.
Spaur et al showed last year that man can work effectively and live comfortably for periods of at least 7 days in helium-oxygen at 49.5 atm. abs. (ATA). This pressure is equivalent to 1600 ft (488 m) sea water. Two of six men were studied during compression, habitation and decompression from 49.5 ATA in hyperbaric chambers with a Po2 of 0.30-0.35 ATA, over 32 days. To avoid thermal discomfort, ambient temperature was maintained at 30-32°C (86-89.6°F), relative humidity was 68-74%, and there was no measurable atmospheric velocity (V 10 m/min, or 33 ft/min). Comfort and thermal balance were adequate during mild activity when the men wore swim trunks and cotton jersey shirts. When they were seated without shirts, rectal temperature fell during 2-hr observation periods at depths below 800 ft, while skin temperature and oxygen consumption remained normal, and scrotal temperature was increased. The fall in rectal temperature during rest appears to be due more to skin convection than to respiratory heat losses, and the coefficient of cutaneous convection could be expressed in the form of Varene's equation, in

\[ h_c = 1.09 P^{0.78} \]

which \( h_c \) is in watts/m² °C, and \( P \) is pressure in ATA. Individual differences in \( h_c \) and in core-to-skin heat conductance appeared to be related to body type. The weight loss seen in all 6 subjects (Clin. Res. 21, 4/74) appeared to be due more to fluid shifts than to caloric deficiencies, since the divers regained weight during the latter days of the dive while consuming fewer calories (2211 kcal/day) than they had during the period of weight loss, at similar levels of activity.
It is now clear that man can work at pressures equivalent to ocean depths of 1600 ft. Exposure to such pressure (50 ATA) is accompanied by weight loss, as also occurs in the weightless state of outer space. Since the mechanism of weight loss in hyperbaric helium-oxygen atmospheres is unknown, we studied 5 men who lived in such an atmosphere in a pressure chamber for 32 days. During the first half of the study, weight loss averaged 4.5 kg despite free access to food and fluids which yielded a caloric intake of 2400 kcal/day. Weight loss was partially reversed in the second half of the dive, during which caloric intake was 2200 kcal/day. The weight loss appears to be due to an initial diuresis, caused either by peripheral vasoconstriction or by changes in intrathoracic pressure due to dense-gas breathing. Urinary nitrogen losses due to high hydrostatic pressure were not excessive. During weight loss, the urinary Na/K ratio and osmolality and serum K fell, suggesting that aldosterone secretion was increased due to a reduced intravascular volume, but that antidiuretic hormone activity did not rise. Weight loss in hyperbaric He-O2 environments thus appears to be due to fluid imbalance related to the Gauer-Henry reflex (atrial distension inhibiting vasopressin secretion) rather than to caloric deficiency or thermal stresses, which have formerly been invoked.
SEX AND THE SATURATION DIVER

Johnsonbaugh, R. E., L. W. Raymond, J. Sode, and S. B. Engel

Previous studies of the effect of surgical and emotional stress on man have shown a decrease in functioning of the hypothalamic-hypophyseal-testicular axis as measured by changes in serum testosterone, leutinizing hormone (LH) and follicle stimulating hormone (FSH). Prior reports at this conference have shown a stress response to chronic chamber pressures equivalent to 300, 1000, and 1600 ft sea water, as measured by adrenocortical activation. It would be of importance to know whether the reproductive axis was altered in any significant manner during these simulated deep sea water dives. We measured serum testosterone, LH and FSH at 10 ATA, serum testosterone and LH at 31 ATA and serum FSH at 50 ATA in teams of 5-6 U. S. Navy divers exposed to He-0₂ atmospheres for 10 to 32 days at pressures equivalent to 300, 1000, and 1600 ft of sea water.

All values obtained were within the normal range. On only one day of the 10 ATA dive was serum testosterone and LH significantly lower and this followed a twenty-four hour period in which the divers were kept working exhaustively. The remainder of the values did not differ significantly from pre- or post-dive values nor was there any significant change between intra-dive values.

These data indicate that under deep diving conditions with He-0₂ atmosphere the hypothalamic-hypophyseal-testicular axis is not adversely affected.
BLOOD GlUCOSE IN DEEP-SEA DIVERS

Raymond, L., J. Sode, R. Johnsonbaugh, R. Bower and W. Spaur

Unlike the glucose intolerance observed in man following simulated weightlessness or exposure to hypobaric hyperoxia, divers who inhabit hyperbaric, hyperoxic He-0₂ environments are said to have hypoglycemia. We therefore measured serum glucose (FBS), insulin (IRI) and growth hormone (GH) following the usual 8-12 hr overnight fast, in normal men chronically exposed to He0₂ at pressures equivalent to 75, 300, 1000 and 1600 ft sea water. As in prior studies, FBS in He-0₂ was often lower than pre-exposure values in air (p= .20, .01, .02 and .01, respectively) but in vitro studies indicated that a part of the differences of FBS in He-0₂ was due to artifacts incurred during decompression of blood samples. FBS was normal during all the dives, the lowest value being 75±4 mg%(SEM). IRI was normally coupled to FBS, except during decompression from 1000 ft, where transient dissociation was suggested by a rising IRI in the face of falling FBS. GH concentrations were normal in all dives, except for the first morning at 1600 ft where GH = 10.6 ± 4.2 ng/ml, in the presence of marked neurological dysfunction. We thus found no evidence of "hyperbaric hypoglycemia" other than the in vitro lowering during decompression of blood samples. Therefore, neuroglucopenia cannot be invoked to explain the "High Pressure Nervous Syndrome" of divers at great depths.
SATURATION DIVING - STRESS OR SANCTUARY?

Sode, J., L. W. Raymond, R. E. Johnsonbaugh, G. E. Sloan, D. Hellman

Man has now worked for periods of a week in hyperbaric chambers at pressures up to 50 atm. abs. These chamber exposures test the performance of man and his life support systems prior to open-sea dives to equivalent depths. Uncertainty exists as to whether the chamber dives impose significant stress. Although land-based and carefully monitored, they nevertheless carry the risk of equipment malfunction, human error, and compression/decompression sickness. Communications are greatly impaired between divers due to helium speech problems at great pressures. In addition, exhausting work, thermal stresses, and confinement may be part of the experimental protocol. We have measured 24-hour urinary 17-hydroxycorticosteroids (17-OHCS), free cortisol (F), epinephrine (E) and norepinephrine (NE) in teams of U. S. Navy divers exposed to He-0₂ atmospheres for 10 to 32 days, at pressures equivalent to 300, 1000 and 1600 ft sea water. Adrenocortical and sympathto-adrenomedullary activity was stimulated by these exposures. Mean values (+ S.D) were as follows:

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<th>17-OHCS</th>
<th>F</th>
<th>E</th>
<th>NE</th>
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<tr>
<td>mg/gm Cr</td>
<td>mcg/gm Cr</td>
<td>mcg/gm Cr</td>
<td>mcg/gm Cr</td>
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<tr>
<td>Saturation Divers</td>
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<td></td>
<td></td>
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<tr>
<td>300 ft</td>
<td>4.4± 0.4</td>
<td>107± 20</td>
<td>------</td>
</tr>
<tr>
<td>1000 ft</td>
<td>5.0± 0.8</td>
<td>299± 81</td>
<td>3.8± 1.0</td>
</tr>
<tr>
<td>1600 ft</td>
<td>4.2± 0.5</td>
<td>193± 25</td>
<td>4.7± 1.7</td>
</tr>
<tr>
<td>Free-living normals</td>
<td>3.0± 0.6</td>
<td>62± 17</td>
<td>5.9± 2.0</td>
</tr>
<tr>
<td>Hospitalized patients</td>
<td>4.0± 1.6</td>
<td>89± 43</td>
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These data indicate that even under optimal circumstances in the absence of untoward sea states, hostile marine life, overt thermal stress or other hazards of open-sea dives, living and working under deep hyperbaric chamber conditions is associated with a substantial degree of adrenocortical activation. The consequences of this activation upon man's adaptation to these environments requires further investigation.
NUTRITION IN HYPERBARIC He-O2 ENVIRONMENTS

V. Frattali

Body weight loss is a common occurrence in saturation diving that appears to be related to the depth and duration of a dive in both laboratory chamber experiments and undersea excursions. Apart from those instances involving prolonged exposure to a cold stress, weight loss is elicited even under those circumstances where the chamber or habitat environment is maintained in a comfortable thermal zone. More often than not, daily caloric consumption is not below 2500 kcal and may exceed 4000 kcal. In order to assess the nutritional implications of this physiological reaction to a hyperbaric environment, a series of clinical and biochemical tests were performed on blood and urine samples collected from the six divers involved in the Navy 1600 ft dive conducted in 1973. Specimens were collected prior to, during, and after the dive; a diet log was maintained by each diver.

The maximum average weight loss was approximately 4.5 kg during a period in the dive when average daily caloric consumption was of the order of 2200 to 2400 kcal. Commensurate with the weight loss was the observation that a negative fluid balance appeared to persist throughout most of the dive. Increases in concentration of serum protein and albumin and a decrease in serum potassium concentration are consistent with the induction of a state of hypohydration in the six divers. Further, observed fluctuations in the excretory rates for thiamin, riboflavin, niacin and N1-methylnicotinamide mimic changes that occur in humans who voluntarily subject themselves to a semi-starvation dietary regimen for a period not exceeding 10 days.

Although the results from this saturation dive do not permit a critical assessment of cause-effect relationships for changes in nutritional state, hypotheses relating to the insensible thermal drain of a hyperbaric environment and to fluid imbalance related to the Gauer-Henry reflex are at least plausible.
A number of enzymes have been examined during hyperbaric exposure, including LDH, GOT, GPT, and CPK. These were selected since the most information is available on their response to tissue damage. For instance, LDH is present in all tissues and many types of damage cause rises in this enzyme. GPT is similarly a soluble enzyme and increases primarily in liver damage. GOT occurs both as a soluble enzyme and as part of a cellular organelle, the mitochondrion. Consequently, relatively mild tissue damage which can be visualized as damage to cell membranes allows both GOT and GPT to "leak" from the cell. More severe damage resulting in cell necrosis causes greater elevations of GOT and GPT, and it has been demonstrated that both the soluble enzyme and that associated with mitochondrion appear in serum during severe tissue damage. Finally, CPK is present in skeletal muscle, cardiac muscle, and brain, but nearly totally absent from other body tissues. CPK is elevated in a number of pathological syndromes, such as early muscular dystrophy, but it is also increased by hypoxia and exertion.

From in vitro experiments, the increase in serum CPK appears to be most associated with the maintenance of intracellular ATP levels. Hence, Sweetin and Thompson have shown that decreased glucose supply, presumably resulting in decreased availability of ATP, results in leakage, not only of ions such as potassium, but also enzymes like LDH and CPK. Decreased availability of metabolic energy may be a more logical explanation for changes we have observed in hyperbaric environments, thus allowing leakage of small amounts of CPK into serum. It may also explain why large changes in CPK and other enzymes are observed only in severe episodes of decompression sickness. The less severe elevations of CPK referred to earlier are generally not associated with concomitant increases in other serum enzymes.

During the bottom time of the dive at 1600 ft, the mean CPK level was approximately twice that observed either during the baseline period or decompression. Other enzymes such as SGOT did not change at any time during the dive.

To conclude, other workers have shown in vitro that decreased oxygen supply or decreased glucose levels can allow soluble enzymes to "leak" through cell walls. This peculiar susceptibility of CPK may be due to the relatively higher energy requirements of muscle tissue. In the absence of any data to link the minor elevations of serum CPK to specific
pathological processes, such as decompression sickness, it seems attractive to postulate that these increases result from a decreased availability of metabolic energy during hyperbaric exposure.
Endogenous carbon monoxide (CO) from crew metabolism accumulates in the gas phase and as COHb in diver blood during saturation diving. Distribution of total CO between blood and gas can be predicted from the total gas volume, total blood hemoglobin, and the gas oxygen tension. Fasting blood COHb was measured in six divers at intervals through the pressurization phase, bottom time, and during decompression in a simulated saturation dive to 50 ATA in helium-oxygen mixtures with constant oxygen content of 0.32 ATA.

Twenty-four hours after entering the chamber all divers, habitual smokers and non-smokers, had reached the same COHb corresponding to that of a normal non-smoker. The COHb increased at a linear rate of 0.28% saturation per day throughout the periods of pressurization and bottom time. These data combined with the calculated changes in CO within the chamber gas, indicate an accumulation of CO which is approximately double the expected rate of endogenous CO formation in air at 1 ATA.

Blood COHb decreased during decompression. The change was slow during the first week of decompression but increased as the pressure was reduced through a combination of venting chamber gas and addition of oxygen to maintain constant O₂ at 0.32 ATA. The blood COHb reached a value approximately equal to that for normal non-smokers breathing air at 1 ATA before decompression was completed. Data obtained during decompression were used to estimate endogenous CO production of the six divers. The value calculated, 10.7 uM/Kg day, is similar to that for normal men in air, 8.5 uM/Kg day. The calculated accumulation rate of CO during pressurization and bottom time is about 183% of the calculated rate of endogenous formation during decompression. These data may be interpreted as a minor increase in heme turnover in this period combined with small additions of CO in the helium and oxygen gases used for pressurization.
HYPERBARIC GASES AND THE O2 TRANSPORT SYSTEM OF RED BLOOD CELLS

L. A. Kiesow

Hyperbaric inert gases increase the affinity of red cell hemoglobin for oxygen. The quantitative extent of this effect depends on the partial pressure of the inert gas, its chemical nature, and the biochemical state of the hemoglobin. Molecular mechanisms by which hyperbaric gases can produce the observed changes in hemoglobin-oxygen affinity will be discussed. Particular attention will be given to the effects of helium on the oxygen transport systems of the red cells.
POSTURAL EQUILIBRIUM AND VESTIBULAR RESPONSE 
AT 49.5 ATA

W. R. Braithwaite - Electronystagmography
T. E. Berghage - Statometer and Balance Rail Performance

In response to the relatively high incidence of vestibular symptoms reported during deep experimental saturation dives, the U. S. Navy included in its 1600 ft chamber dive protocol measures of postural equilibrium and vestibular function. Six subjects were pressurized in six days to 49.5 ATA. After spending seven days at this pressure, they were decompressed in 19 days to the surface. The tests administered prior to, during, and following the dive included electronystagmography with mental concentration, optokinetic stimulation, cold caloric stimulation, and positional testing along with three balance rail tests and two statometer tests. All of the measures of spontaneous and induced nystagmus were within normal limits while the tests of standing steadiness all showed statistically significant deviations from surface values. Performance on both the balance rail and the statometer showed a striking deterioration associated with increased pressure and some adaptation with time at depth. The decrements that were observed are very complex in nature with statistically significant interactions among all of the experimental variables. Experimental variables in the study included ambient pressure, individual differences, time at 1600 ft, dive phase (compression versus decompression) and in the case of the statometer, response frequency.
ALTERATIONS IN LUNG VOLUMES AND VENTILATORY DYNAMICS DURING A DIVE TO 49.5 ATA


Flow-volume loops, forced vital capacity (FVC), maximum voluntary ventilation (MVV) and lung volumes were measured in six U. S. Navy divers during a 1600 ft helium-oxygen dive conducted by the U. S. Navy Experimental Diving Unit in April-May 1973. The Wedge Spirometer was used with a 2-channel Brush Recorder and a Brush X-Y Recorder (model 1000). The frequency response of the X-Y Recorder was 4 cps in the range in which the measurements were made, therefore, peak flows were slightly underestimated. Maneuvers were done in the dry and head-out immersed (wet). There was a 300 cc increase in FVC during the 32 day dive. The dry peak expiratory flow changed from 12.4 L/sec on the surface (air) to 6.7 L/sec at 1600 ft (He02) to 16.4 L/sec (He02) at 20 ft during decompression. During decompression, the head-out immersed peak expiratory flows were 1-2 L/sec greater than in the dry. The difference between wet and dry flow rates decreased progressively at 75%, 50%, and 25% VC. The MVV changed from 175 L/min on the surface (air) to 100 L/sec at 1600 ft with the decompression values being significantly greater than the compression values ending at 20 ft with 225 L/min. Wet MVV tended to be greater than dry at comparable depths. The expiratory reserve volume increased linearly from the surface to 1600 ft with return to baseline values during decompression (r = .93). The absolute increase from the surface to 1600 ft was 0.8 L.
CARDIORESPIRATORY RESPONSES OF IMMERSED EXERCISING DIVERS AT
31.30 AND 49.48 ATA

Spaur, William H., W. R. Braithwaite, L. D. Thalmann, M. M. Knott,
J. H. Crothers, L. W. Raymond

Cardiorespiratory responses of immersed exercising divers were studied during hyperbaric chamber complex experimental dives to 31.30 ATA (1000 ft) and 49.48 ATA (1600 ft). The in-water studies were performed using the MK 10 Mod 4 closed-circuit underwater breathing apparatus which maintained the oxygen partial pressure \( (P_{O_2}) \) between 0.4 and 0.5 ATA and the carbon dioxide partial pressure \( (P_{CO_2}) \) less than 4 mm Hg. Divers performed work on an electrically braked bicycle ergometer. At 31.30 ATA the 3 divers were able to sustain 6-min work periods at work load settings of 5 and 7.5 kg-m/sec and 2-min work periods at 10 kg-m/sec. At the 7.5 kg-m/sec setting, mean heart rate was 152, pH 7.29, arterial lactate 55 mg% and arterial \( P_{CO_2} \) 52 mm Hg. At the time the divers' work capacity was exceeded on the 10 kg-m/sec setting, the mean heart rate was 157, pH 7.27, arterial lactate 70 mg% and arterial \( P_{CO_2} \) 55 mm Hg. For the 49.48 ATA dive, the MK 10 Mod 4 was modified by enlarging the gas passages and replacing the corrugated hoses with 3.18 cm smooth bore hoses. The 3 divers studied were only able to comfortably sustain 6-min work periods at the light work load setting of 2.5 kg-m/sec. At the 5 kg-m/sec setting, the divers experienced extreme respiratory distress which they described as an inability to inspire sufficient gas. The mean heart rate was 136, respiratory rate 29, pH 7.39 and arterial lactate 23 mg% at the termination of the maximum 5 kg-m/sec work rate tolerated. The arterial \( P_{CO_2} \) of the 3 divers was 22, 42, and 45 mm Hg. The findings indicate that the work capacity of the immersed diver at 49.48 ATA is significantly less than at 31.30 ATA and that the mechanism limiting the divers' exercise capability in the water at 49.48 ATA was different from the ventilatory impairment with carbon dioxide retention found in the 31.30 ATA studies.
EQUIPMENT PHYSIOLOGICAL TESTING OF THE MK 12 PROTOTYPE
SURFACE-SUPPORTED HARD-HAT SYSTEM IN THE HELIUM-OXYGEN
RECIRCULATING MODE

Spaur, William H., W. R. Braithwaite, W. B. Wright, E. D. Thalmann
L. W. Raymond

A prototype helium-oxygen recirculating helmet has been
tested to its planned normal operational depth in a hyperbaric
chamber complex. Five immersed divers were studied at 10 ft,
5 at 200 ft, and 4 at 300 ft. Each test sequence consisted
of 10 min rest followed by four 6-min work periods of
increasing intensity. Each work period was separated by a 5-
min rest period. Work was performed on an electrically braked
bicycle ergometer at settings of 5, 7.5, 10, and 12.5 kg-m/sec.
The work involved in moving the legs and pedals against water
resistance was not considered. Heart rate, inspired (helmet)
Pco2, arterial Pco2, pH, lactate and pyruvate were measured. The
divers were able to complete work loads at the 10 or 12.5 kg-m/sec
setting at all depths. Heart rates during the initial rest
period and during mild work were progressively higher as dive
depth increased. Heart rates increased linearly with increased
work loads and were 163, 174, and 176 per min at the heaviest
work rate at 10, 200, and 300 ft respectively. At the heaviest
work rate, arterial lactate measured 97, 100, and 63 mg% at the
respective depths. Inspired Pco2 was 3.4 mm Hg at 10 ft,
2.9 mm Hg at 200 ft, and 6.8 mm Hg at 300 ft during the
heaviest work rate. Arterial Pco2 decreased with increasing work
loads at 10 ft. At 200 ft, the arterial Pco2 peaked at 46
mm Hg and then diminished slightly at the heavier work loads. At
300 ft, the arterial Pco2 increased with increasing work load
reaching 43 mm Hg from an initial hypocapnia. The results
indicate that the prototype helmet system adequately supported
divers performing heavy work to the test depth of 300 ft.
EQUIPMENT PHYSIOLOGICAL TESTING OF THE MK 12 PROTOTYPE SURFACE-SUPPORTED HARD-HAT SYSTEM IN THE OPEN-CIRCUIT AIR MODE

Spaur, William H., W. R. Braithwaite, W. B. Wright, E. D. Thalmann

A prototype air open-circuit helmet system has been tested to its planned normal operational depth in a hyperbaric chamber complex. Five different immersed divers were studied at depths of 10, 100, and 200 ft. Each test sequence consisted of 10 min rest followed by four 6-min work periods of increasing severity. Each work period was separated by a 5-min rest period. Work was performed on a bicycle ergometer at settings ranging from 5 to 15 kg-m/sec. The work involved in moving the legs and pedals against water resistance was not considered. Heart rate, inspired (helmet) \( \text{Pco}_2 \), arterial \( \text{Pco}_2 \), pH, lactate and pyruvate were measured. The divers were able to sustain heavy work loads at all depths. Inspired \( \text{Pco}_2 \) at the heaviest work loads was 10 mm Hg at 10 ft and 4 mm Hg at 100 ft. Arterial \( \text{Pco}_2 \) during the heaviest work rate was 36 mm Hg at 10 ft and 45 mm Hg at 100 ft. At 200 ft, the inspired \( \text{Pco}_2 \) and the arterial \( \text{Pco}_2 \) progressively increased as work load was increased. At work loads of 7.5 and 10 Kg-m/sec, when the inspired \( \text{Pco}_2 \) reached 15 mm Hg, the arterial \( \text{Pco}_2 \) exceeded 50 mm Hg. During the heaviest work rate the inspired \( \text{Pco}_2 \) reached 19 mm Hg and the arterial \( \text{Pco}_2 \) was 59 mm Hg. The results indicate that the air open-circuit prototype helmet system adequately supported divers performing heavy work to depths of 100 ft. However, at 200 ft, heavy exercise caused unacceptable inspired \( \text{Pco}_2 \) and hypercarbia.
Reflex cardiac action of four men was studied at 28.8, 22.9, 14.2 and 3.7 ATA by observing heart rate during breath holding during face immersion in 15°C and 35°C water, and during the Valsalva maneuver. Heart rates during any of these maneuvers under pressure did not significantly differ from control rates at one atmosphere. However, certain trends were evident. Pooling of the breath-holding and face immersion data indicated that the usual slowing of the heart during these maneuvers was potentiated by increased atmospheric pressures up to 14.7 ATA and then returned to near control levels at 28.8 ATA. The marked bradycardia seen after strain release during the Valsalva maneuver was also diminished at the greater pressures. The afferent pathways of these maneuvers are dissimilar and one may conclude, since both their responses seem to be blunted at the higher pressures, that the effector pathway (the parasympathetic) or the effector organ — the heart, has some altered function.

Data from four subjects exposed to pressures up to 30 atmospheres absolute do not indicate that cardiogenic reflex action approaches hazardous levels and that, indeed, heart reflex action may be somewhat reduced under certain hyperbaric conditions due to altered nervous function.
Among the various measures used to evaluate central nervous system functioning during the U.S. Navy's deep chamber dive to 1600 FSW, were sixteen performance tests from the SINDBAD testing system. Previous studies have shown the 16 tests used to be independent measures of separate human abilities. Six tests of perceptual skill, five tests of cognitive ability, and five motor performance tests were administered to six subjects at the surface and at three depths; 1000, 1300, and 1600 FSW. Performance on all but one of the perceptual tests showed either slight improvement or no change at all. Performance deteriorated on four of the five cognitive tests. The one cognitive test that did not show a decrement is heavily influenced by learning and it is possible that the effects of practice masked the influences of the environment. Of the five tests that require motor skill, four showed slight decrements and one showed improvement. The motor test that improved was a multilimb coordination task that required relatively smooth gross motor movements of both arms. The SINDBAD test results, when coupled with the subjective reports of the divers, tend to indicate that divers working at depths as deep as 1600 ft will experience difficulty. Tasks involving fine motor dexterity and fast intellectual response seem to present the greatest problem. Perceptual-observational type tasks, gross motor tasks, and deliberate intellectual tasks seem to be unaffected.
MICROTREMOR

Bachrach, Arthur J. and Arthur Findling

Microtremor samples were collected on 6 subjects at surface, 400 ft, 1000 ft, 1300 ft and 1600 ft using the NMRI force transducer. Two forces were used at all depths, 50 gm and 500 gm, calibrated by the diver subjects. The samples which were analyzed show a general decrease in the low-frequency area from the surface sample down to 400 ft and a further decrease at the 1000 ft level. The 1300 ft and 1600 ft samples exhibit an increase from the 1000 ft samples. All subjects show an increase in amplitude* at all depths with the greatest at the 1600 ft level, but this is not reflected in the frequency analysis. Differences between the 50 gram weight requirements and the 500 gram weight indicate the heavier requirement is associated with increased tremor, owing probably to more demanding neuromuscular control.

Problems in collecting data at the 1000 ft and 1300 ft samples are reflected in the noise in the 12-13 Hz area and put in doubt the validity of the samples collected at these two depths. There were no indications of a tremor "signature" among subjects.

*The assistance of LCDR T. E. Berghaye in the analysis of these data is gratefully acknowledged.
THE EEG IN SLEEPING DIVERS

Wilcox, Robert H. and Frank Russo

The extension of electroencephalographic (EEG) studies to night sleep during deep dives has not been automatic, but as a measure of brain function and a possible early indicator of reduced waking efficiency, it has certain advantages. Sleep is hardly a homogeneous process, but rather consists of several distinct "stages" defined by EEG criteria which normally occur in a reliable pattern during the night. Moreover, the very nature of the sleep process makes it relatively resistant to voluntary control, thereby insuring that most disruptions of normal patterns are free from fluctuations in subject motivation.

The Navy 1600 ft dive at New Orleans sleep recordings were undertaken to test an hypothesis developed during two 1,000 ft dives earlier in the year, at Duke University and the Experimental Diving Unit in Washington, D.C. In all three of the divers monitored during these two projects, reductions were observed at or near 1,000 ft in what is known as Stage 3 and Stage 4 sleep, characterized by large amounts of high amplitude, low frequency waves in the EEG. Stage 4 sleep in particular seems to be especially necessary for the organism, as it occurs relatively early in the sleep period and is the first type of sleep to be made up after sleep deprivation; it can also be considered our deepest sleep in terms of slowness of electrical activity in the brain and also of reduced physiological parameters such as heart rate, respiration rate, and body temperature. By taking sleep recordings at both 1,000 ft and 1,600 ft at New Orleans, we could check for similar reductions at a greater depth level and, on the same dive, replicate our 1,000 ft investigations.

During the Navy 1600 ft dive scorable sleep records were obtained on two divers for one night each at 40 ft, 1000 ft, and 1600 ft. Both of these subjects showed no Stage 4 sleep at all at 1000 ft. Percentages of delta sleep (Stage 3 and 4 combined) were also least for both divers at 1000 ft, though this trend was not statistically significant. However, percentages of Stage 4, Stage 3, and Stages 3 plus 4 sleep at 1600 ft all showed no significant differences from the shallow controls. Sleep profiles in general appeared most stable at the shallow depth, less stable at 1600 ft, and most erratic at 1000 ft. No differences in REM or in the other stages of sleep were seen. Subjective sleep logs proved largely inconclusive, in that both divers consistently characterized their sleep as "light, with many awakenings" due primarily to the difficult sleep conditions.
Pooling of the New Orleans data with that of the previous two dives shows a significant depression of Stage 4 and Stages 3 plus 4 sleep at 1000 ft compared to shallow controls, but little difference at 1600 ft. This effect has persisted despite equivalences in total sleep time, and REM sleep time and periodicity, and was not confounded by daytime naps. Furthermore, it cannot be explained by adaptation to depth or compression, as the 1000 ft effects at New Orleans were obtained during decompression after the more normal 1600 ft recordings. Thus, a U-function of Stage 4 and delta sleep to depth is described, and a 1000 ft barrier with unique properties is indicated.
# Hyperbaric Effects on Sleep at Various Simulated Depths

<table>
<thead>
<tr>
<th>DEPTH</th>
<th>SHALLOW 40-116 ft 1.2-3.5 ATA</th>
<th>DEEP 870-1,000 ft 26.4-30.3 ATA</th>
<th>DEEPEST 1,600 ft 48.5 ATA</th>
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<tbody>
<tr>
<td>SLEEP STAGE</td>
<td>W REM 1 2 3 4</td>
<td>W REM 1 2 3 4</td>
<td>W REM 1 2 3 4</td>
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<td>STUDY S</td>
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<td>DUKE G.B.</td>
<td>9.6 17.6 13.0 44.4 12.1 3.2</td>
<td>5.7 17.3 19.9 52.4 4.7 0.0</td>
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<tr>
<td>EDU P.M.</td>
<td>11.2 23.8 8.6 38.0 16.4 1.9</td>
<td>6.6 29.2 17.3 43.1 3.8 0.0</td>
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<tr>
<td>EDU J.J.R.</td>
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<td>17.2 11.4 20.8 35.6 9.4 5.6</td>
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<tr>
<td>NAVY-L.R. TAYLOR (N.O.) C.W.</td>
<td>8.3 20.5 11.7 35.9 9.0 14.7</td>
<td>9.1 22.2 16.5 50.2 2.0 0.0</td>
<td>8.9 23.4 18.5 37.6 4.3 7.5</td>
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<tr>
<td>NAVY-L.R. TAYLOR (N.O.) C.W.</td>
<td>13.2 18.7 11.7 36.7 8.2 11.5</td>
<td>4.9 32.8 8.5 42.6 11.2 0.0</td>
<td>13.9 28.4 21.5 22.6 4.6 9.1</td>
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<tr>
<td>MEAN%</td>
<td>9.7 19.1 10.3 40.0 12.8 8.2</td>
<td>8.7 22.6 16.6 44.8 6.2 1.1</td>
<td>11.4 25.9 20.0 30.1 4.4 8.3</td>
</tr>
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</table>
During the U.S. Navy's deep chamber saturation dive to 49.5 ATA (1600 FSW) several physiological parameters were monitored. Because subjects on previous deep dives have displayed abnormal neuromuscular responses, special attention was given on this dive to the monitoring of central nervous system (CNS) functioning. Measures of CNS functioning included electronystagmography, postural equilibrium, intentional tremor, perceptual motor skill, and electroencephalograms. The electroencephalograms were monitored at several depths during the compression phase of the dive, but only those obtained at 1000, 1300, and 1600 FSW were subjected to detailed computer analysis. At these three depths, six EEG channels were recorded on magnetic tape for each of the six divers. The frequency and amplitude of these recordings has been analyzed using a PDP-12/40 computer. The frequency analysis results show a slight rise in theta and beta and a reduction in alpha associated with increased pressure. The amplitude of the EEG signals showed a marked, statistically significant reduction with increased depth. The implications of these changes are not yet understood. At this point in time the results are merely an observed response pattern associated with increased helium-oxygen pressure.
A PROPHYLACTIC PROGRAM FOR THE PREVENTION OF OTITIS EXTERNA IN DIVERS

E. D. Thalmann

Otitis externa is probably the leading cause of lost diving time during saturation dives. Our experience at the Navy Experimental Diving Unit (NEDU) has been that on saturation dives lasting more than three days, the incidence of otitis externa has been 25% to 70% and once the symptoms of otitis externa do develop, the high temperature and humidity of the chamber environment make the disease especially refractory to the usual methods of treatment.

Over the past several years many prophylactic regimens have been tried at NEDU and none have proven particularly effective. Early in the planning of the 1000 foot dive in February 1973, it was decided to once again try to institute a prophylactic program for the prevention of otitis externa. The program consisted of irrigating both ear canals for 5 full minutes with a 2% acetic acid in aluminum acetate solution twice daily and once after each dive. This program has been tested on 4 saturation dives greater than 10 days in length and there has been no incidence of otitis externa. The irrigating solution appears to have no undesirable side effects.

A search of the literature revealed that acetic acid solutions in strength greater than 2% have been used successfully for the treatment of otitis externa while weaker solutions are less effective. Alcohol seems to be as effective a drying agent as aluminum acetate.
FREON CONTAMINATION AT 400 FEET DURING A SATURATION DIVE

Thalmann, Edward D., L. W. Jenkins

During a 1600 ft saturation dive done by the Navy Experimental Diving Unit, the chamber was inadvertently contaminated with Freon 113 at the 400 ft stop during descent. Gas chromatography was used to determine the extent of the contamination which was 30 ppm. The chambers were ventilated and the contamination was eliminated in a very short period of time. Although 30 ppm of Freon 113 is not considered a hazard at atmospheric pressure, the length and depth of the dive dictated that all the contaminant be eliminated from the chamber before the dive progressed. Once the contaminant was eliminated, the dive proceeded on schedule.