# SEVENTH SYMPOSIUM ON UNDERWATER PHYSIOLOGY (PROGRAMS, ABSTRACTS AND MINI-PAPERS)

**Author(s):** Bachrach, A.
Chairman of the Symposium Governing Board

**Performing Organization Name and Address:**
Federation of American Societies for Experimental Biology
9650 Rockville Pike, Bethesda, MD 20014

**Controlling Office Name and Address:**
Office of Naval Research
800 North Quincy Street
Arlington, VA 22217

**Monitoring Agency Name and Address:**

**Distribution Statement (of this Report):**
Distribution is unlimited.

**Supplementary Notes:**

**Key Words (Continues on reverse side if necessary and identify by block number):**
- Hyperbaric Physiology
- Respiratory Physiology
- Cardiopulmonary Physiology
- Neurophysiology
- Decompression
- Decompression Sickness
- Inert Gas Narcosis
- Body Heat Loss
- Oxygen Toxicity
- Psychomotor Performance
- Hydrostatic Pressure Effects

**Abstract (Continues on reverse side if necessary and identify by block number):**
The Program featured state-of-the-art reviews by eminent authorities, followed by shorter research papers selected by the Symposium Governing Board from submitted mini-papers. In response to the Call for Papers, more than 100 contributions were received, of which 46 were selected for oral presentations in symposia and 37 were programmed as poster presentations. Symposia included the following topics:
Oxygen Toxicity
Oxygen Sufficiency and Utilization within the Cell
Metabolism and Thermal Physiology
Molecular and Cellular Effects of Hydrostatic Pressure
High Pressure Nervous Syndrome
Cardio-Respiratory Responses to Exercise
Inert Gas Exchange and Decompression
Health Hazards

Attendance for the Symposium was gratifying, with a total of 298 registrants representing 25 countries. The majority (65%) were from countries other than the U.S.A.
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- Oxygen Toxicity
- Oxygen Sufficiency and Utilization Within the Cell
- Metabolism and Thermal Physiology
- Molecular and Cellular Effects of Hydrostatic Pressure
- High Pressure Nervous Syndrome
- Cardio-Respiratory Responses to Exercise
- Inert Gas Exchange and Decompression
- Health Hazards

There appeared to be a broad consensus that the return to presentations of intensive current status reviews produced some unusually fine papers, and that the 7th Symposium was a professionally rewarding experience.

A copy of the Program, Abstracts and Mini Papers booklet is enclosed which will serve as the final technical report for the symposium.

Arthur J. Bachrach, Ph.D.
Symposium Chairman
7TH SYMPOSIUM ON UNDERWATER PHYSIOLOGY

UNDERSEA MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING
EUROPEAN UNDERSEA BIOMEDICAL SOCIETY ANNUAL MEETING

A Satellite of the XXVIII International Congress of Physiological Sciences

July 5-10, 1980
Athens Hilton
Athens, Greece

PROGRAM, ABSTRACTS, AND MINI-PAPERS
7th SYMPOSIUM ON UNDERWATER PHYSIOLOGY
GOVERNING BOARD

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*In Memoriam

Sponsors of the 7th Symposium:
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Charles W. Shilling, Executive Secretary

Address for both the Symposium Secretariat and Undersea Medical Society:
9650 Rockville Pike, Bethesda, Maryland, 20014, U.S.A.
PROGRAM, ABSTRACTS AND MINI-PAPERS

THE UNDERSEA MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING

THE 7TH SYMPOSIUM ON UNDERWATER PHYSIOLOGY

THE EUROPEAN UNDERSEA BIOMEDICAL SOCIETY ANNUAL MEETING

JULY 5 - 10, 1980
The Athens Hilton Hotel
Athens, Greece
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GENERAL INFORMATION

REGISTRATION AND INFORMATION
Athenian Lobby, Athens Hilton

Hours:
Saturday, 5 July .............................. 1200 - 1800
Sunday, 6 July ............................... 0800 - 1700
Monday, and Tuesday, 7-8 July .......... 0830 - 1700
Wednesday, 9 July .......................... 0830 - 1300
Thursday, 10 July ............................ 0830 - 1700

For information of any kind, consult the Symposium Registration/Information Desk.

Notices about Symposium events will be posted on bulletin boards near the Information Desk.

SECRETARIAT
Symposium and UMS staff will be available at the Information Desk in the Athenian Lobby throughout the hours shown above.

MESSAGES
Those who wish to leave messages for registrants during the above hours should ask the hotel operator (Athens Hilton telephone number: 720-201) for the 7th Symposium Registration/Information Desk, Athenian Lobby. Messages will be posted on the bulletin board adjacent to the Information Desk.

BANQUET AND LUNCHEON TICKETS
Available at the Registration/Information Desk, Athenian Lobby.

Tickets are 500 Drachmas per person and must be purchased by 1200 Hours on Monday, 7 July.

VISITOR INFORMATION
Information on Athens attractions, museums and tours is available at the Symposium Registration/Information Desk, Athenian Lobby.

CURRENCY EXCHANGE
Exchange of foreign currencies may be made at the Ionian and Popular Bank of Greece, located off the main lobby of the Athens Hilton Hotel.

AIRLINE RESERVATIONS
Several of the major airlines have offices in the Athens Hilton. DO NOT FORGET TO RECONFIRM YOUR RETURN FLIGHT.

HOTEL DINING AND LOUNGE FACILITIES
The Athens Hilton facilities include the Trattoria, an Italian specialties restaurant; the Taverna Ta Nissia, a tavern following the Greek style; a Roof Top Supper Club overlooking the Acropolis; the Pan Piano Bar; and the Byzantine Coffee Shop which is open 24 hours daily. The Coffee Shop is extremely busy and, accordingly, the service can be rather slow so allow sufficient time in your schedule if you intend to breakfast in the hotel.

SYMPOSIUM PROCEEDINGS
The PROCEEDINGS of the 7th Symposium will be published shortly after the meeting. If you wish to be included on the mailing list to receive order forms for the PROCEEDINGS when available, please leave your name and address at the Registration/Information Desk.

CONTINUING MEDICAL EDUCATION CREDITS
The program of the 7th Symposium, including the Undersea Medical Society and European Undersea Medical Society sessions, has been certified for one CME hour credit for each hour of scientific sessions attended. Certification forms are available at the Symposium Registration/Information Desk, Athenian Lobby.
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<td>1200-1800 Symposium Regis. &amp; Info.</td>
<td>1500-1645 Sess. 4- Oxygen I</td>
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<td>1500-1900 Sess. 16- HPNS</td>
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<td>1500-1900 Sess. 17- Posters: Metab. &amp; Thermal Phys.</td>
<td>1830-1930 EUBS Annual General Meeting</td>
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<td>If you have not purchased tickets for &quot;An Evening in Piraeus&quot; or the UMS Lunch, do so today.</td>
<td>1715-1900 Sess. 12- Posters: Molec. &amp; Cell Effect of Hydrostatic Pressure</td>
<td>1715-1900 Sess. 12- Posters: Molec. &amp; Cell Effect of Hydrostatic Pressure</td>
<td>Afternoon &amp; evening free for individual plans.</td>
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<td>2030-7th Symp. Opening Reception</td>
<td>1930- An Evening in Piraeus</td>
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<td>Afternoon &amp; evening free for individual plans.</td>
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PROGRAM

SATURDAY, 5 JULY

REGISTRATION AND INFORMATION - Athenian Foyer
1200 to 1800 Hours

SUNDAY, 6 JULY

REGISTRATION AND INFORMATION - Athenian Foyer
0800 to 1700 Hours

UNDERSEA MEDICAL SOCIETY ANNUAL
SCIENTIFIC MEETING

WELCOME AND OPENING REMARKS
0815 - Terpsichore Ballroom
JEFFERSON C. DAVIS, President, Undersea Medical Society

SESSION I

DECOMPRESSION — Terpsichore Ballroom
Co-Chairmen: H. V. HEMPLEMAN and B. G. D'AUGST

0830 Evaluation of different decompression schedules by agarose gel bubble. Y. MANO, M. SHIBAYAMA and H. MAEDA

0845 The development and testing of high altitude diving tables using extrapolated U.S. Navy critical tissue pressure criteria. R. L. BELL, A. C. THOMPSON and R. E. BORGWARDT


0915 The perfusion/diffusion dilemma: resolution and clarification by isobaric gas switching. B. G. D'AUGST, C. YOUNG, R. WHITE, and R. DUNFORD

0930 Pitfalls in the diagnosis of dysbaric osteonecrosis and the significance of suspected lesions. J. K. DAVIDSON, W. P. TROWBRIDGE and D. N. WALDER


SESSION 2

POSTER PRESENTATIONS — Nectar/Ambrosia Room
0830-1200 (Coffee with the authors 1000-1030.)

Board #

1 Treatment of cardiovascular dysfunction resulting from cerebral air embolism. D. E. EVANS, A. I. KOBRINE, E. T. FLYNN and M. E. BRADLEY

2 Neurophysiological and biochemical studies in He-N2-O2 atmosphere at 11 ATA. I. STOILLOVA, V. KOLEV, I. DOSSEVA, I. VENKOV, T. TENCHева, A. DISHELOV and A. VARBANOVA

3 Visceral malformations, resorptions, and birthweight among fetal rats exposed to air at increased atmospheric pressure. M. E. BOLTON and A. L. ALAMO

4 Brainstem evoked potential changes associated with variations in middle-ear pressure. B. M. CLOPTON and J. M. MILLER

5 Analysis of medical reasons for withdrawing medical certification of fitness in commercial divers in the U.K. W. A. CROSIE

6 Modelling, measurements, and moments of inert gas exchange. P. K. WEATHERSBY and L. D. HOMER

7 The effects of cold stress on venous gas bubble production in man following a no-decompression dive. R. DUNFORD and J. HAYWARD

8 Size distribution of intravascular bubbles induced by decompression. B. D. BUTLER, B. A. HILLS and T. E. SUTTON

9 Thermal effects of recompressed bubbles. R. G. BUCKLES, M. E. COX and J. B. ECKENHOFF

10 Results of validation testing of flying-after-diving schedules. B. E. BASSETT

11 An analysis of the effects that hyperbaric oxygen has upon pressure reduction tolerances in rats and humans. D. E. YOUNT and D. A. LALLY

12 Physicochemical properties of the nonionic surfactants surrounding gas cavitation nuclei (microbubbles). J. S. D'ARRIGO

SESSION 3

HYDROSTATIC PRESSURE — Terpsichore Ballroom
Co-Chairmen: J. C. ROSTAIN and P. B. BENNETT

1030 Acute injection of phenytoin and long latency evoked potentials in guinea pigs under high pressure helium. P. G. KAUFMANN, J. C. FARMER, JR. and F. G. HEMPEL

1045 Evaluated microvibration on cut with compression effect to 51 ATA (He-N2-O2). K. SEKI, H. NAKAYAMA and M. MATSUDA

1100 H.P.N.S. in human during 38 hours compression to 450m with N. injections. J. C. ROSTAIN, B. GARDETTE, M. C. GARDETTE-CHAUFFOUR and R. NAGUET

1115 Diazepam under hyperbaric conditions in rats. L. GRAN, R. COGGIN and P. B. BENNETT

1130 Changes in red cell membrane enzymes in man during simulated dives of up to 55 bar in helium-oxygen. J. A. PACIOREK and R. F. CARLYLE

1145 The effect of hydrostatic pressure on enzymes involved in the oxygen metabolism. E. MORILD and J. E. ØLMHEIM
SESSION 4

OXYGEN I — Terpsichore Ballroom
Co-Chairmen: Y. G. ZORBAS and M. D. FAIMAN

1500 The effect of hyperbaric oxygen inhalation upon the ultrastructure of the lung alveoli. T. K. AKERS

1515 Alterations in oxidative metabolism during recovery from pulmonary oxygen toxicity. W. D. CURRIE, P. C. PRATT and A. P. SANDERS

1530 On the influence of exogenous and endogenous substrate accumulation on drug-induced variations in glutamic acid decarboxylase activity prior to oxygen high pressure exposure. B. E. SEGERBO

1545 Oxygen convulsions in mice. Influence of nitrogen admixture. N. BARTELSON, B. CRIBORN and A. MUREN

1600 Hop-induced cerebral vasoconstriction. Its contribution to CNS-toxicity kinetics. B. BLEIBERG, A. LANIR and D. GERSHON

1615 Tolerance of mice to pulmonary oxygen toxicity. A. LANIR, D. KEREM and D. GERSHON

1630 CNS and pulmonary oxygen toxicity during intermittent exposure to hyperbaric oxygen and air. D. KEREM, C. BITTERMAN and B. BLEIBERG

SESSION 5

POSTER PRESENTATIONS — Nectar/Ambrosia Room
1500-1900 (Coffee with the authors 1645-1715)

Board #

1 Stress and mental performance under water. P. G. A. M. JORNA

2 Noninvasive continuous monitoring of diver pulmonary performance. M. J. ACKERMAN

3 Hydrostatic pressure: Its effects on cellular membrane ion transport. W. R. GALEY, P. S. VAN NIECE and C. V. BEATO

4 The effects of prone immersion on lung function. I. DASKALOVIC, A. HASHIMOTO, E. H. LANPHIER and W. G. REDDAN

5 Thoracic shape, lung volume and diaphragmatic contraction during immersion. V. D. MINH and G. F. DOLAN

6 Blood metabolites in resting and exercising rats at various partial pressures of nitrogen and oxygen. R. de G. HAN-SON, R. M. GRAY, P. SMYTHE and K. G. M. M. ALBERTI

7 Emergency thermal protection for saturation diving. G. H. EGSTROM and A. DICARO

8 Heat stress during dives in warm water. I. HOLMER and G. KIHLSTROM

9 Effect of body temperature and composition on recovery from hypothermia. J. B. MORRISON, J. S. HAYWARD and M. L. CONN


11 An analysis of emergency heating requirements for personnel transfer capsules. E. H. WISSLER

SESSION 6

OXYGEN II — Terpsichore Ballroom
Co-Chairmen: E. KINDWALL and D. ELLIOTT

1715 Induction of cytochrome P-450 by hypoxia and hyperoxia in vivo and in vitro. H. A. ROWE, S. F. GOTTLIEB and I. S. LONGMUIR

1730 Hydrogen oxygen exposure of rabbits at 30 ATA with multi-day survival. H. E. ÖRHAGEN, C. G. LUND- GREN and A. MUREN

1745 Effect of normobaric and hyperbaric oxygen on cyanide intoxication. T. TAKANO, Y. MIYAZAKI, I. NASHIMOTO and K. KOBAYASHI

1800 Hyperbaric gasoline: Tissue oxygen characteristics in chronic, soft tissue wounds. P. J. SHEFFIELD

1815 Adrenergic and cardiopulmonary responses to exercise with air and helium-oxygen at 1 ATA. E. T. FLYNN, D. E. EVANS, K. M. GREENE, D. C. LEGRYS and R. P. LAYTON

1830 Differential performance behavior after a 40-hour compression to 450 MSW. C. LEMAIRE

1845 Influence of exercise on ventilatory capacity at depth. A. PASCE and C. LUNDGREN

7TH SYMPOSIUM OPENING RECEPTION
2030 Hours - Pool Area
HOSTED BY THE GREEK GOVERNMENT

MONDAY, 7 JULY

REGISTRATION AND INFORMATION - Athenian Foyer
0830 to 1700 Hours

7TH SYMPOSIUM ON UNDERWATER PHYSIOLOGY

WELCOMING REMARKS
0830 Hours - Terpsichore Ballroom

A. J. BACHRACH, Symposium Chairman
C. J. LAMBERTSEN, University of Pennsylvania Medical Center
S. G. ALIVISATOS, University of Athens
S. MARKETOS, Secretary General, Ministry of Social Services
SESSION 7

OXYGEN TOXICITY — Terpsichore Ballroom
Chairman: H. SALTZMAN; Co-Chairman: M. W. RADOMSKI
Rapporteur: A. B. FISHER

0900 Review: Current concepts of oxygen toxicity. J. CLARK
0930 Mechanism(s) of central oxygen toxicity: A re-evaluation. M. D. FAIMAN, R. J. NOLAN, D. E. DODD, J. M. WAECHTER, R. C. DIRKS, K. HAYA and J. A. ZEMPEL
0950 The central role of ammonia in OHP induced convulsions. E. W. BANISTER and A. K. SINGH
1010 Coffee and Poster Presentations
1040 Changes in cell volume following hyperbaric exposure: A manifestation of oxygen toxicity. J. POOLEY and D. N. WALDER
1100 Lung ATP turnover during oxidant stress. A. B. FISHER
1120 Protection from pulmonary oxygen toxicity by treatment with low doses of bacterial endotoxin. L. FRANK, M. J. CHIANG and D. MASSARO
1140 Evolution of pulmonary diffusing capacity after deep saturation dive with high O2 level during decompression. R. H. HYACINTHE and B. BROUSSOLLE

SPECIAL FILM
1200 Hours - Terpsichore Ballroom
The Duke 650 Meter Dive. P. B. BENNETT

POSTER PRESENTATIONS — Nectar/Ambrosia Room
0900-1200

SESSION 8

PSYCHOMOTOR PERFORMANCE AND HIGH PRESSURE NERVOUS SYNDROME
Board #

2 A theory of inert gas narcosis. B. FOWLER
4 Genetics of variability in susceptibility to HPNS Type I seizures in mice. R. D. MCCALL and D. FRIERSON, JR.
6 Modification of electrophysiological sleep under the hyperbaric environment (31ATA, He-N2-O2, 34 days, 3 divers). K. SEKI, H. NAKAYAMA and M. MATSUDA

SESSION 9

CARDIO-RESPIRATORY EFFECTS
Board #

7 Inertance as a factor in uneven ventilation in diving. J. R. CLARKE, M. A. FISHER and M. J. JAEGGER
8 The arrhythmogenic potency of hydrostatic pressure on cardiac conduction. T. J. DOUBT and P. M. HOGAN

10 Pulmonary function in divers. M. CIMSIT and V. FLOOK
11 Regulation and frequency of heart rate during op. n-seg saturation diving. S. M. GOSOVIC and A. I. RADOVIC
12 Influence of the inspiratory effort and swallowing on the cardiovascular response to simulated diving and breathing. T. F. HUANG and C. T. PENG
15 The effect of water temperature on vital capacity during head-out immersion. D. I. KURSS, C. E. G. LUNDGREN and A. J. PASCHE

SESSION 10

OXYGEN SUFFICIENCY AND UTILIZATION WITHIN THE CELL — Terpsichore Ballroom
Chairman: A. KOVACH; Co-Chairman: J. C. DAVIS
Rapporteur: L. A. KIESOW

1500 Review: Current concepts of oxygen sufficiency and utilization within the cell. F. F. JOBSIS
1530 Use of aortic body and carotid body chemoreceptors as internal probes to monitor tissue oxygenation. S. LAHIRI
1550 Heterogeneity of capillary distribution and capillary circulation in mammalian skeletal muscles. E. M. RENKIN, S. D. GRAY, L. R. DODD and B. D. LIA
1610 Retinal oximetry with hypercapnia and hyperbaric oxygen. F. G. HEMPEL, S. R. BURNS and H. A. SALTZMAN
1630 A mechanism for the beneficial effect of hyperbaric oxygen on staphylococcal osteomyelitis. J. T. MADER and G. L. BROWN
1650 Coffee and Poster Presentations

SESSION 11

METABOLISM AND THERMAL PHYSIOLOGY — Terpsichore Ballroom
Chairman: K. BONDI; Co-Chairman: M. MATSUDA
Rapporteur: G. EGSTROM

1720 Review: Current concepts of metabolism and thermal physiology. P. WEBB
1750 An analysis of heat stress under hyperbaric conditions. K. R. BONDI
1810 Contribution of metabolic and respiratory heat to core temperature gain after cold water immersion. M. L. CONN, P. A. HAYES and J. B. MORRISON
1830 The metabolic and thermal status of divers during simulated dives to 55 bar. M. P. GARRARD, P. A. HAYES, R. F. CARLYLE and M. J. STOCK
SESSION 12

MOLECULAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE

Board #

1 A study of the specific action of "per se" hydrostatic pressure on fish considered as a physiological model. L. BARTHELEMY, A. BELAUD and A. SALIOU
2 Osmotic fragility of erythrocytes: Effects of hydrostatic pressure and pentanol. A. C. HALL and A. G. MACDONALD
3 A mathematical analysis of high pressure and anaesthetic effects. M. I. HALSEY, A. F. MOTT, C. C. SPICER and B. WARDLEY-SMITH
4 Contrasting actions of hydrostatic pressure and helium pressure on growth of saccharomyces cerevisiae. S. R. THOM and R. E. MARQUIS
5 Effects of different normoxic hyperbaric exposures on glucose, lactate and glycogen brain concentrations. T. OBRENOVITCH and F. BRUE

SESSION 13

INERT GAS EXCHANGE AND DECOMPRESSION

Board #

7 Study on definition of maximum permissible gas flow in lungs during decompression. J. PARC and J. LE CHUITON
8 Evaluation of decompression tables by a model describing bubble dynamics in vivo. S. MEISEL, Y. TALMON and D. KEREM
9 Computer simulation of diffusive gas mixing in the lung at 10 ATA. H. D. VAN LIEW
10 Some recent experiments on bubble formation in supersaturated gelatin. D. E. YOUNT, C. M. YEUNG and T. D. KUNKLE

SESSION 14

HEALTH HAZARDS

Board #

11 Microbiological studies on acute otitis externa in saturation divers. S. R. ALCOCK
12 An epidemiological study of fatal diving accidents in two commercial diving populations. M. E. BRADLEY
13 Drug therapy of decompression sickness. B. BROUS-SOLLE
14 Decompression sickness in commercial diving population. M. R. CROSS and L. A. BOOTH
15 An evaluation of cardiopulmonary resuscitation techniques for use in a diving bell. R. MYERS and M. E. BRADLEY

SESSION 15

MOLECULAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE — Terpsichore Ballroom

Chairman: L. BARTHELEMY; Co-Chairman: M. I. HALSEY
Rapporteur: A. G. MACDONALD

0915 Review: Current concepts of molecular and cellular effects of hydrostatic pressure. A. G. MACDONALD
0945 Effects of hyperbaric conditions on the multiplication of Echo 11 Herpes Simplex Virus (Type 1 and Type 2) in tissue culture. C. CHASTEL, L. BARTHELEMY, A. BELAUD and A. MICHAUD
1030 Coffee
1100 Effects of high hydrostatic pressures on Na+ transports across isolated gill epithelium of sea water acclimated eels Anguilla anguilla. A. J. R. PEQUEUX
1120 A quantitative description of pressure-induced alterations in ionic channels of the squid giant axon. B. B. SHRIVASTAV, J. L. PARMENTIER and P. B. BENNETT
1140 Transient versus steady state effects of high hydrostatic pressure. K. T. WANN, A. G. MACDONALD, A. A. HARPER and M. L. J. ASHFORD
1200 The effects of high pressures of inert gases on cholinergic receptor binding and function. J. F. SAUTER, L. BRASWELL, P. WANKOWICZ and K. W. MILLER

SESSION 16

HIGH PRESSURE NERVOUS SYNDROME

Chairman: R. NAQUET; Co-Chairman: J. VOROSMARTI
Rapporteur: D. MILLAR

1500 Review: Current concepts of high pressure nervous syndrome. J. HALLENBECK
1530 The effects of general anaesthetics on post-synaptic responses. H. J. LITTLE and W. D. M. PATON
1610 Prevention of HPNS: The possible use of structural looseners of anaesthetics. B. WARDLEY-SMITH and M. J. HALSEY
1630 Rapid compression with trimix (He-N2O3). P. B. BENNETT, R. COGGIN, J. ROBY and J. N. MILLER
1650 Coffee and Poster Presentations
1720 The effect of high pressure on cooperative lipid-protein interactions. H.-J. GALLA and J. R. TRUDELL
1740 Currents in a voltage-clamped vertebrate neuron at hyperbaric pressure. J. J. KENDIG
(Session 16 Continued)

1800  Differential effects of pressure on the mammalian central nervous system. P. G. KAUFMANN, P. B. BENNETT and J. C. FARMER, JR.

1820  Somatic evoked potentials in monkey during saturation dives (He-O2 and He-N2-O2). M. HUGON, K. SEKI, L. FAGNI and J. C. ROSTAIN

1840  Differentiation of the two components of the convulsion stage of the HPNS in vertebrates. R. W. BRAUER, R. W. BEAVER, H. W. GILLEN, W. M. MANSFIELD, JR. and R. D. MCCALL

POSTER PRESENTATIONS — Nectar/Ambrlosia Room
1500-1900

SESSION 17

METABOLISM AND THERMAL PHYSIOLOGY
Board #

7  Energy and body fluid balance during a 14-day dry saturation dive at 31 ATA (Seadragon IV). H. NAKAYAMA, S. K. HONG, J. CLAYBAUGH, N. MATSUI, Y. S. PARK, Y. OHTA, K. SHIRAKI and M. MATSUDA

A computer model designed to make rapid predictions of diver temperature changes. S. WILCOCK and V. FLOOK

SESSION 18

OXYGEN TOXICITY
Board #

Comparative effects of various protective agents upon acute cerebral hyperbaric oxygen toxicity in mice: Particular interest of some benzodiazepines. F. BRUE, F. JOANNY, A. CHAUMONT, J. CORRIOL and B. BROUSSOLLE

Effect of excessive oxygen upon the capability of the lungs to filter gas emboli. B. D. BUTLER and B. A. HILLS

SEM observations of oxygen toxicity in guinea pigs exposed to continuous 100%, 85%, or 75% oxygen at 1 ATM. A. E. McKEE and M. E. BRADLEY

The influence of inert gas concentration on pulmonary oxygen toxicity. M. R. POWELL and H. D. FUST

Brain GABA and cGMP as indices of metabolic lesions in CNS during acute oxygen toxicity. M. W. RADOMSKI and W. J. WATSON

Pulmonary prostaglandin metabolism during normobaric hyperoxia. C. L. SCHATTÉ and M. M. MATTHIAS

AN EVENING IN PIRAEUS
1930 Hours
Buses pick up registrants at the Athens Hilton, arriving at the National Yacht Club in Piraeus at 2000 for cocktails, dinner and entertainment. See General Information section for ticket information.

Buses depart National Yacht Club at 2300 Hours for return to the Hilton.

WEDNESDAY, 9 JULY

SESSION 19

CARDIO-RESPIRATORY RESPONSES TO EXERCISE — Terpsichore Ballroom
Chairman: C. E. LUNDGREN Co-Chairman: B. BROUSSOLLE
Rapporteurs: A. A. BOVE

0900  Review: Current concepts of cardio-respiratory responses to exercise. L. FAGRAEUS

0930  Exercise metabolism in humans on acute exposure to a 5.8 bar normoxic oxyhelium environment. R. de G. HANSON, R. M. GRAY, M. M. WINSBOROUGH, R. S. MCKENZIE AND K. G. M. ALBERTI

0950  Comparison of metabolic responses and growth hormone release during submaximal exercise in man breathing helium or air at normal barometric pressure. J. RAYNAUD, P. VARENE and J. DURAND

1010  Break

1040  Effects of exercise and hyperbaric air on ventilation and central inspiratory activity. C. M. HESSER and F. LIND

1100  Inspiratory dyspnea during exercise at 47 ATA. J. SALZANO, E. M. CAMPORESI, B. STOLP, H. SALZMAN, W. BELL and D. SHELTON

1120  Carbon dioxide retention with underwater work in the open ocean. J. DWYER, J. W. MACDONALD, B. W. STOLP and A. A. PILMANIS

1140  Cardiopulmonary functions and maximal aerobic power during a 14-day saturation dive at 31 ATA (Seadragon IV). Y. OHTA, H. ARITA, H. NAKAYAMA, S. TAMAYA, C. LUNDGREN, Y. C. LIN, R. M. SMITH, R. MORIN, L. E. FARHI and M. MATSUDA

UNDERSEA MEDICAL SOCIETY ANNUAL BUSINESS MEETING AND AWARDS LUNCHEON

1215 to 1500 Hours - Hesperides Room

The Suzanne Kronheim Memorial Lecture, presentation of awards, and business meeting. See General Information section for ticket information.

SUZANNE KRONHEIM MEMORIAL LECTURE
Mental activity related to the blood flow and metabolism of the brain. D. H. INGVAR, University Hospital, Lund, Sweden

PRESENTATION OF AWARDS
The Albert R. Behnke Award, The Stover-Link Award, and The Oceanic Society International Award

REMARKS BY THE INCOMING PRESIDENT, PAUL WEBB

FOLLOWING THE LUNCHEON, AFTERNOON AND EVENING FREE FOR INDIVIDUAL PLANS.
THURSDAY, 10 JULY

SESSION 20

INERT GAS EXCHANGE AND DECOMPRESSION — Terpsichore Ballroom

Chairman: H. V. HEMPLEMAN; Co-Chairman and Rapporteur: K. D. REIMANN

0900 Review: Current concepts of inert gas exchange and decompression. P. WEATHERSBY
0930 Species independent maximum no-bubble decompression from saturation dive. Y. C. LIN
0950 Determination of safe tissue tension values during the surface interval in surface decompression schedules for helium-oxygen dives. P. O. EDEL
1010 Break
1040 Assessment of decompression profiles and divers by doppler ultrasonic monitoring. R. Y. NISHI, K. E. KISMAN, B. C. EATOCK and G. MASUREL
1100 Monitoring bubble formation with an integrating pulse-echo ultrasonic method. S. DANIELS, J. M. DAVIES, W. D. M. PATON and E. B. SMITH
1120 Migration of lung surfactant to pulmonary air emboli. B. A. HILLS and B. D. BUTLER
1140 Prevention of decompression sickness by combined cyproheptadine-amphetamine treatment. C. CHRYSSANTHOU, L. RODRIGUEZ and P. BRANDEN

EUROPEAN UNDERSEA BIOMEDICAL SOCIETY

SESSION 21

HEALTH HAZARDS — Terpsichore Ballroom

Chairman: A. A. BOVE; Co-Chairman: C. CHRYSSANTHOU

Rapporteur: D. H. ELLIOTT

1500 Review: Current concepts of aural barotrauma. J. C. FARMER, JR.
1530 Mechanisms of aural barotrauma. J. MILLER, A. AXELSSON, D. McPHERSON and W. POTTER
1550 Water-borne microbial pathogens and diving environments. O. P. DAILY, S. W. JOSEPH, J. D. GILLMORE, R. J. SEIDLER, D. A. ALLEN and R. R. COLWELL
1610 Management of health hazards associated with the salvage of toxic chemicals using a saturation diving technique. A. MARRONI, J. GETHING and D. ZANNINI
1630 Break
1700 Review: Current concepts in bone necrosis research. D. N. WALDER
1730 Abnormal bone and cartilage collagen metabolism in experimentally induced dysbaric osteonecrosis. D. B. PARSONS, M. E. BRADLEY
1750 A detailed histological and radiological controlled study of selected bones from divers. C. R. WEATHERLY, W. M. PARK, M. HADDAWAY and I. CALDER
1810 The efficacy of spinal anesthesia at high pressure. H. F. NICODEMUS, H. McELROY and R. F. EVY
SESSION I

DECOMPRESSION

EVALUATION OF DIFFERENT DECOMPRESSION SCHEDULES BY A CLOSER GAGE. V. B. Bell, W. B. Gage, and W. H. Jaffe. Dept. of Public Health, Tokyo Medical and Dental University, Tokyo, Japan.

Decompression schedules after dive are actually different in countries than in the U.S. and Japan, and it is too difficult to apply them because of the differences in the physiology between the equator and the schedule and the body's response.

The decompression work by an aggerable gage has been recognized in a dry chamber controlled the temperature to evaluate different decompression schedules like the U.S. Naval table and the standard table.

Aggerable gage bubbles are only physically formed by pressure changes and it is obvious that decompression sickness is due to the bubble formation, plus our physiological body reaction after bubble formation, our physical conditions and so forth.

It is shown that bubble formation work by an aggerable gage has been recognized in a dry chamber controlled the temperature to evaluate different decompression schedules like the U.S. Naval table and the standard table.

The decompression schedules for this study were nine tables for both dives and compressed air workers. The i.e. different in each schedule, even though the depth and the bottom time are same. Aggerable gage was used to test the waterbath pressure and time decomposed according to the each schedule, counted bubble number in each 0.27 ml of the cells and those schedules were evaluated by the bubble number.

NON-HALITANT DECOMPRESSION SCHEDULES. T. T. J. J. Decompression schedules for both dives and compressed air workers. The i.e. different in each schedule, even though the depth and the bottom time are same. Aggerable gage was used to test the waterbath pressure and time decomposed according to the each schedule, counted bubble number in each 0.27 ml of the cells and those schedules were evaluated by the bubble number.

The development of non-halitant decompression schedules for both dives and compressed air workers. The i.e. different in each schedule, even though the depth and the bottom time are same. Aggerable gage was used to test the waterbath pressure and time decomposed according to the each schedule, counted bubble number in each 0.27 ml of the cells and those schedules were evaluated by the bubble number.

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THE DEVELOPMENT AND TEST IN DECOMPRESSION SCHEDULES USING EXTRACTED L. R. NAVY CRITICAL POINT PHASE DIAGRAM. C. A. Moore, H. H. C. Thompson and H. L. Borogart. Interpretive Research and Development Division, University of California, Los Angeles, California, U.S. The critical tissue pressure curves obtained by the U.S. Navy were extrapolated to obtain predicted critical tissue pressure values for comparison.

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NEUROPHYSIOLOGICAL AND BIOCHEMICAL STUDIES IN Rats

MOSKOWITZ AND BULGARIAN ACADEMY OF SCIENCES, BULGARIA

A joint Moscow-Bulgarian experiment "EURO-L-100" was carried out in the USSR in 1973. Three experiments were performed under conditions of 14-day stay in pressure chamber. 7 days at 14 ATA, using He-Na$_2$O atmospheres in different rates. The main aim of the experiment was to study the changes taking place in the human organism during and after continuous exposure to high pressure conditions in He-Na$_2$O medium.

In the course of the experiment recordings were made of the EEG, both spontaneous and in functional tests, of the averaged potentials (AP) after light stimulation, as well as polyphotographic sleep recordings. Lipid metabolism - total lipids, phosphatidyl choline, phosphatic acid - as well as the acid-base equilibrium, were studied parallel with the electrophysiological data. The electrophysiological analysis showed that the experimental conditions had different effects on the different subjects due to the individual adaptation possibilities and they were a factor influencing AP generation.

The longer latencies of the EP components observed in the course of the experiment should be assumed to be one of the indicators of the general physiological stress under hypobaric conditions. A readjustment of the metabolic process requires considerable energy expenditure which is compensated by a general intensification of lipid metabolism.

VISCERAL MALLIGATIONS, RESISTANCES, AND BEDWETTING AMONG PEAL RATS EXPOSED TO AIR AT INCREASED ATMOSPHERIC PRESSURE, M. N. PLOM and A. M. ALFRED, University of Florida, Gainesville, Florida, U.S.A.

Atmoic exposure to air at greater than 1 atmosphere absolute pressure (1.0 ATA) has been associated with various physiological and psychological changes in normal adult mice. Recently, postmortem examination of these animals has revealed increased frequency of total blood circulation and death. The purpose of this report was to determine if rats subjected to absolute pressures of 1.0 ATA and above after 48 minutes, and then subjected to an atmosphere of 4.5 ATA and above after 30 minutes, would exhibit an increased frequency of total blood circulation and death. The results of this study are reported in the following metabolic analysis.

PRESENTATIONS SESSION II

ANALYSIS OF MEDICAL ERROR IN RETROVIRAL MEDICAL CERTIFICATION IF FITNESS IN COMBINED DIVING IN THE U.K.

J. A. CHERY, King's College Hospital Medical School, London, and R.C.C.M., Great Yarmouth, U.K.

Diving regulations in the U.K. demand that a diver be medically examined every 18 months to assess his fitness in work underwater. A body of "approved doctors" administers the system in the U.K. but problems arise when a diver is found to have developed some minimal medical condition. The degree and type of medical condition is then considered and a period of work under water is recommended. Over the past 15 years, there has been a marked increase in the number of medical conditions found which prevent a diver from being fit for work underwater. The aim of this study was to determine the nature and frequency of medical conditions found which prevent a diver from being fit for work underwater.

The age range was 20-50 years and commercial diving experience 1-30 years. In 10 tests 73% of divers were fit to work underwater and 27% were found to be unfit. The majority of divers were found to be affected by the following medical conditions: asthma, allergic rhinitis, sinusitis, chronic bronchitis, phlebitis, and peptic ulcer disease.

In the region of the body where the symptoms were found to be the most significant were the following: cardiovascular, respiratory, gastrointestinal, and musculoskeletal systems. The most significant findings were found to be related to the cardiovascular system with 73% of divers found to have significant findings in this area. The results of this study indicate that the medical condition found in divers is related to the cardiovascular system and that divers must be carefully monitored to ensure their safety in work underwater.
OXYGEN I

Session IV

The Effect of Intrascholastic Oxygen Inhalation on the Activity of the Time-Antithetic, Incubated, Catalase-Mediated, Neutrophil Oxidative Metabolism.

Large quantities of hydrogen peroxide and hypochlorite were injected intravenously into mice. This resulted in the injection of free radicals into the mouse, which then produced excessive tissue damage in the vicinity of the injection site. The following day, the mice were sacrificed and the organs and tissues were harvested and examined. The results showed that the injection of free radicals into the mouse resulted in the production of free radicals in the vicinity of the injection site, which then produced excessive tissue damage. This suggests that the injection of free radicals into the mouse may be a useful method for the study of free radical-mediated tissue damage.
POSTER PRESENTATIONS

SESSION IV

OXYGEN I

OXYGEN AND POLYMYEAL DYSFUNCTION DURING INTERMITTENT EXPOSURE TO HYPOBARIC OXYGEN AND AIR. A. K. Fliedner, W. C. Krahl, A. L. Habraken, A. S. Carey, and H. G. Kollin, latest, University of California, Los Angeles, and Naval Medical Research Institute, Bethesda, Maryland. 3.

A blinded observer evaluated the quality of the data and classified the results as normal or abnormal. The results were then compared with the corresponding values obtained from normal subjects. The observers were able to differentiate between normal and abnormal data with a high degree of accuracy. The results of this study suggest that intermittent exposure to hypobaric oxygen may be beneficial in treating certain neurological conditions, such as stroke or brain injury. Further studies are needed to confirm these findings and to explore the potential mechanisms by which hypobaric oxygen may exert its effects.

SESSION V

PULMONARY FUNCTION IN HEALTH AND DISEASE. R. I. Alksnis, Naval Medical Research Institute, Bethesda, Maryland.

A novel noninvasive method for assessing pulmonary function in health and disease has been developed and validated. The method involves measuring the response of the respiratory muscles to a brief period of hypoxia, which is then used to calculate the work of breathing. The method is highly sensitive and specific for detecting subtle changes in pulmonary function, and has been found to be useful in the evaluation of patients with a wide range of respiratory diseases. The method is also relatively inexpensive and noninvasive, making it an attractive option for widespread use in clinical practice.

The results of this study demonstrate the potential utility of the novel method for assessing pulmonary function in health and disease. Further studies are needed to fully evaluate the method and to explore its clinical applications.
Emergency Thermal Protection for Saturation Diving. Glen H. Exargon and Anthony D'Ildefons. Commercial Diving Center, Wilmington, California

The loss of power and completion of saturation dives has resulted in casualties. In circumstances in which breathing gas supplies and CO elimination capability were adequate for an extended period of time, lack of facility control has quickly shifted ambient conditions to 98-99% relative humidity, 100% in all field environments, with the container short time in the unpressurized chamber of the dive. A study conducted in the Commercial Diving Center's saturation facility involved a series of saturation dives for up to 12 days. A 150-psig dry air supply was maintained in the chamber for breathing gas, with the air supply being kept below 150-watt outputs per hour. During the initial 24-hour exposure, the temperature was maintained at 95°F (35°C) and 90-95% relative humidity. Comparative data was recorded each 30 minutes for 24 hours. The results indicated that the thermal environment was constant at 95°F (35°C). Heat stress during dives in this environment was not observed during the 24-hour exposure.

1. Thermal protective equipment maintained dive comfort during a 24-hour exposure in a 75°F (24°C) environment. The diver's initial rectal temperature of 97°F (36°C) and the hour 24 rectal temperature of 97°F (36°C) were not observed.

2. Reduced metabolic activity during rest and sleep did not result in hypothermic discomfort or alterations of the dive environment.
POSTER PRESENTATIONS

SESSION V

OXYGEN II

SESSION VI

EFFECT OF NORMOPHASIC AND HYPERBARIIC HYDROGEN ON CYANIDE INHIBITION. Toshikazu Tsuchida, Yasuko Tsuchida, Yoko Yamauchi, Takehiko Ikeda, Mutsuo Nakamura. Department of Physiology, Kyushu University, Fukuoka, Japan.

In order to evaluate the effect of normobaric and hyperbaric hydrogen on cyanide poisoning, the intracellular inhibition of oxygen metabolism was studied in isolated rat hearts. The hearts were perfused with a solution containing either 1% or 4% hydrogen in nitrogen. The results showed that hydrogen had a protective effect on the hearts, reducing the effects of cyanide poisoning.

HYPOBARIAN OXYGEN TENSION IN HUMANS AT 30 KM WITH MULTIPLE CONVULSIVE T.C. Hjelmfelt, E. R. Lundgren and A. Sernbo, Laboratory of Aviation and Astronautical Engineering, Malmö University, Sweden.

In this study, the effect of hypobaria on oxygen tension in humans was investigated. The subjects were exposed to a simulated altitude of 30 km for 2 hours. The results showed that the oxygen tension decreased significantly, indicating the importance of oxygen supply in high altitude environments.

Irradiation of Sodium Lactate Systematic Analysis, T. H. Oldfield, Hyperbaric Medicine Division, Brooks AFB, Texas 78235, U.S.A.

The study investigated the effects of irradiation on the sodium lactate system. The results showed that irradiation had a significant impact on the system, with changes in the sodium lactate levels and pH observed.

ENDOTHELIAL CONFIGURATION IN CYANIC HYPOXIA AND ITS RELATIONSHIP TO THE CYANIDE INHIBITION. W. B. Brown, P. A. Estrella, and R. M. Estrella, Department of Physiology, University of Texas Medical Branch, Galveston, Texas.

The study explored the relationship between cyanide inhibition and endothelial configuration in cyanic hypoxia. The results showed that the endothelial configuration was affected by cyanide inhibition, indicating the importance of endothelial function in hypoxic conditions.

HEMODYNAMIC ADAPTATION: ISSUES CONSIDERED IN CYANIC, SOFT TISSUE WOUNDS. F. J. Newfield, Hyperbaric Medicine Division, Brooks AFB, Texas 78235, U.S.A.

The study investigated the issues considered in cyanic, soft tissue wounds. The results showed that the adaptation to cyanic hypoxia was crucial in managing these types of wounds, with a focus on oxygen therapy and wound care.

SUPPORT FOR NATIONAL SHAPELESS TECHNOLOGY DEVELOPMENT. Supported by National Agency for Technology Development.

The study reviewed the support for national shapeless technology development. The results showed that the development of shapeless technology was crucial for various applications, with a focus on innovation and technological advancement.

The full text of these presentations can be found in the respective proceedings of the conference.

This study investigated the capacity of the respiratory system to respond to increased workloads during exercise at increased depths of breath. The study was conducted in a hyperbaric chamber where subjects were exposed to increased oxygen tensions. The results showed that the capacity to deliver oxygen to the tissues increased with increased workloads and increased depths of exercise. The study also demonstrated that the respiratory system had the ability to adapt to the increased workloads and maintain adequate oxygen delivery to the tissues. This study has important implications for understanding the physiological responses to increased workloads and deep diving conditions, which is crucial for safety and optimal performance in such environments.
MINI-PAPERS

7TH SYMPOSIUM ON UNDERWATER PHYSIOLOGY

In the interest of space, references have been eliminated from the following mini-papers; however, all papers will be printed in full, including references, in the Symposium PROCEEDINGS.
OXYGEN TOXICITY

SESSION VII

MULTIPLAS AND METHODS

Backman:

The course of brain and blood catecholamines during hypoxia. Groups of rats were exposed to 5% oxygen for 7 hours. It was found that the intracellular and extracellular concentrations of norepinephrine, dopamine, and serotonin were increased at the end of the exposure. Blood and brain samples were taken for analysis of the norepinephrine and dopamine content at different time intervals. The results were compared with those obtained in control animals exposed to normal oxygen tensions.

McIntyre:

A study on the effect of hypoxia on the activity of tryptophan hydroxylase, an enzyme which catalyzes the conversion of tryptophan to serotonin. The enzyme activity was measured in brain tissue from rats exposed to 5% oxygen for 7 hours. The results showed a significant decrease in enzyme activity, indicating a decreased synthesis of serotonin under hypoxic conditions.

Dow:

The effect of hypoxia on the activity of the enzyme monoamine oxidase. The enzyme activity was measured in brain tissue from rats exposed to 5% oxygen for 7 hours. The results showed a significant decrease in enzyme activity, indicating a decreased metabolism of monoamines under hypoxic conditions.

Littler:

The effect of hypoxia on the activity of the enzyme catechol-O-methyltransferase. The enzyme activity was measured in brain tissue from rats exposed to 5% oxygen for 7 hours. The results showed a significant increase in enzyme activity, indicating an increased catabolism of catecholamines under hypoxic conditions.

Summary:

The study showed that hypoxia had a profound effect on the activity of enzymes involved in the synthesis and metabolism of monoamines. The decrease in tryptophan hydroxylase activity indicates a decreased synthesis of serotonin, while the decrease in monoamine oxidase activity indicates a decreased metabolism of monoamines. The increase in catechol-O-methyltransferase activity indicates an increased catabolism of catecholamines. These findings suggest that hypoxia may have significant implications for the regulation of monoamine neurotransmission in the brain.
advantage of the pathway where ammonia production once has proved to be prevented by Boyne et al. (1978). During hypoxia these authors observed that brain glutamine and IAA increased and glutamic acid was decreased. Hypoxia also stimulated ammonia formation but brain ammonia did not increase in the first hour of hypoxia where O2 fixation and amonia surfaced to buffer it. Glutamic acid concentrations naturally would have to increase to initiate the buffer action and early in the hypoxic period, one might assume that an enhanced buffer formation would also occur as was indeed observed. Thus amonia production became too great to be buffered by a buffering cycle. Reaction then glutamic and aspic acid concentrations declined. These limitations in the capacity of the O2 fixing systems provide an explanation for the above observations.

Figure 1 illustrates the multi factors placed upon glutamic acid concentrations during hypoxia as a potential, delvetor of oxidation, from the Kreb cycle as a component of the respiratory cycle (Hester, 1976) producing glutamic acid from nitrogen, acid transport and free radical scavenging, no process in the formation of GABA a neuronal depressant and emphasizing the complex hierarchy of events leading to cumulative action within which ammonia and glutamic acid occur in central roles.

In the experiments described, the authors have been able to determine the production of ammonia from an extract of brain and circulating catecholamine, confirming activity by glucose, and to determine the phenomena of cumulative activity in hypoxia.

References will appear in PREVIEW.

Table 1 and Figure 1 follow.

![Oxygen Toxicity](image)

**Fig. 1.** Contributing effects of catecholamine depletion and ammonia to cumulative activity in hypoxic states.

**Table 1.**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Dose (mg/ml)</th>
<th>Current (mA)</th>
<th>V O2 (ml/min)</th>
<th>H Concn. (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.00</td>
<td>2.50</td>
<td>10.00</td>
<td>0.30</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>0.01</td>
<td>2.50</td>
<td>10.00</td>
<td>0.30</td>
</tr>
<tr>
<td>DHCP</td>
<td>0.02</td>
<td>2.50</td>
<td>10.00</td>
<td>0.30</td>
</tr>
</tbody>
</table>

**Notes:**

- *p < 0.05 when compared with normal control.
- *p < 0.05 when compared with experimentally changed control.

**CHANGES IN CELLULAR OXYGEN TOLERANCE: A HYPOTHESIS FOR OXYGEN TOXICITY.**

In: Re oxygen and safety. Walker, University of Newcastle upon Tyne, 1964 (p. 1).

A decrease in blood flow has been observed in rabbit heart when air during hypoxia by Walker and Walker in 1964. It was speculated that the increase in oxygen cell volume might occur during hypoxia as an increase to blood flow during hypoxia and that the, by increasing the oxygen to hypoxia blood flow, could account for the observation. To confirm this hypothesis experiments have been performed to determine the cell volume distribution in a rat cell suspension and to determine the effect of changes in cell volume on the oxygen uptake of the cell cells at various gas mixtures at various gas mixtures.

Simultaneously, a study of the morphological appearance of fat cells, following hypoxic exposure, has been carried out.

For the purpose of measurement, the work was carried out in an investigation of red cell volume.

The suspensions of isolated fat cells were prepared from the epididymal fat pads of adult white male rats using a technique described by Smith in 1973. In this procedure, the fat pads were removed from one epididymal area and 3-4 whole epididymal fat pads were removed in Krebs-Ringer bicarbonate buffer containing collagenase. After incubation, the isolated fat cells were separated by centrifugation. This preparation provided both a control and a test suspension.

The control suspension was maintained at 37°C under anaerobic conditions. The test suspension was placed in a thermostatically controlled compressor chamber and maintained at 37°C during exposure to compressed air at 3-6 A.T.P. for periods of up to 3 h. At the end of this time an increase in the volume of the fat cells in the suspension was determined by a Coulter counter and a flow cytometer which displayed the result as a volume distribution curve.

By superimposing the volume distribution curve obtained from the test suspension on that of the control, any change in the volume distribution of the fat cell suspension occurring as a result of exposure to compressed air could be detected and the direction of the change determined.

Microscopic examination of both stained and unstained preparations of the fat cell suspensions after exposure to compressed air was carried out by direct microscopy, dark ground and phase contrast techniques.

From the recordings obtained, illustrated in Fig. 1, it can be seen that the change in density of the cell suspension exposed to compressed air is shown to the right of the control suspension. This was found to be the case for all suspensions exposed to compressed air at temperature range of 3-6 A.T.P. for periods of time of 3-3 h. The results of the gas mixture in the cell of the test suspension was found to be the same as the cell of the control suspension. These results indicate that an increase in fat cell volume occurs in vitro as a result of exposure to compressed air.

To evaluate the mechanism of the observed increase in fat cell volume, the separate effects of increased O2 and pressure were investigated. Measurements were made at temperatures of 37°C to determine the effect of increased oxygen on the oxygen uptake of the suspending medium. The effect of hyperbaric oxygen on cell volume was also determined.

Using the techniques described above, the effect of exposing fat cell suspensions to the following gas environments was determined:

- *Tris buffer* (pH 7.4) and *Friedel’s agent* (pH 7.4).
OXYGEN TOXICITY

a. Oxygen mixture: Normal H2 with N2 to 6 A.T.A.
b. Oxygen 100% at 1 A.T.A.

Subsequently, the effect of increasing the partial pressure of oxygen in the suspending medium was investigated by repeating the experiments with fat cells suspended in a hypo-ionic saline medium buffered containing hemolyzed albumin at concentrations of 2% or 4%.

Finally, brain venous blood samples, 2.0 ml volume, were placed in separate plastic containers, maintained at 37°C and exposed to compressed air at pressures ranging from 3-4 A.T.A. - 6-8 A.T.A. for periods from 5 to 24 hours. At the end of this time the volume distribution curves of the test sample was determined, and the volume changes observed were compared with that of a control sample from the same donor kept at atmospheric pressure. The effect on the observed cell volume changes of introducing lithium into the venous blood sample prior to exposure to compressed air was also investigated.

The results of these experiments may be summarized as follows:

1. The volume distribution curves of fat cells exposed to 100% oxygen at 1 A.T.A. were used to the right of those of control suspensions exposed to air at 1 A.T.A. (Fig. 1).

2. The volume distribution curves of fat cells exposed to high partial pressures of helium or nitrogen buffered containing hemolyzed albumin at concentrations of 2% or 4% were also shifted to the right of those of control suspensions exposed to air at 1 A.T.A.

3. When the cells were suspended in a medium of Kussmaul-Hamburger bicarbonate buffer containing hemolyzed albumin at 0% or 2% the volume distribution curves of fat cells exposed to compressed air at pressures ranging from 3-4 A.T.A. and then fat cells exposed to 100% oxygen at 1 A.T.A. were to the left of those of control suspensions. No volume change occurred in cells exposed to high partial pressures of helium or nitrogen when suspended in this medium.

4. The volume distribution curves of fat cells exposed to compressed air at pressures ranging from 3-4 A.T.A. in the absence of those of control suspensions. No volume change was found to be prevented or reversed by the presence of lithium ions in the blood samples.

From these results it was concluded that:

1. Fat cells in suspension increased in volume on exposure to 100% oxygen at 1 A.T.A. This increase in volume was similar to that seen following exposure to compressed air at 3-4 A.T.A.

2. Hepatocytes are exposed to any mixture of helium or nitrogen containing oxygen at a normal partial pressure no effect on the volume of fat cells.

3. A decrease in fat cell volume was seen following exposure to both compressed air and 100% oxygen when Albumin 4% had been added to the suspending medium prior to exposure. However, no change in volume of fat cells was observed in a medium containing albumin was produced by hyperoxic exposure to any mixture containing nitrogen or helium.

4. Red cells in vitro increase in volume when exposed to compressed air at high partial pressures of oxygen, synthetic changes in which the H2O content remained normal in no change in cell volume, excluding pressure per se as a causal factor.

In presence of the test cell volume of fat cells in vitro have been demonstrated following exposure to both compressed air and 100% oxygen.

The size of the cellular volume and the changes in the volume distribution of particulate materials in whole blood and the increase in the relative cell volume in blood containing added oxygen were not observed in cells exposed to compressed air at pressures ranging from 3-4 A.T.A. and then fat cells exposed to 100% oxygen at 1 A.T.A.

As the maintenance of plasma volume is a basic function of mammalian cells, this increase in fat cell volume is a modification of oxygen toxicity.

In summary, the volume of fat cells exposed to increased partial pressures of oxygen has been demonstrated to occur in vitro. The importance of this finding in the role of these cells in the context of the observed mechanism of the action of lithium is still to be determined. A consideration of these results has been accentuated by the recent findings of oxygen toxicity in the intact organism as well as the role of oxygen in the mechanism of the action of lithium in C.S.H. toxicity.

In summary, the increase in the volume of fat cells exposed to increased partial pressures of oxygen has been demonstrated to occur in vitro. The importance of this finding in the role of these cells in the context of the observed mechanism of the action of lithium is still to be determined. A consideration of these results has been accentuated by the recent findings of oxygen toxicity in the intact organism as well as the role of oxygen in the mechanism of the action of lithium in C.S.H. toxicity.

Let us examine the data that we have collected so far. These data show that by increasing the partial pressure of oxygen, we can observe a significant increase in the volume of fat cells. This increase is not only limited to the fat cells in whole blood, but also occurs in isolated, oxygenated fat cells. These findings are consistent with the hypothesis that oxygen, by increasing the volume of fat cells, plays a role in the mechanism of lithium toxicity.

However, it is important to note that these results are preliminary and further studies are needed to fully understand the mechanisms involved. Future research will focus on the role of oxygen in the mechanism of lithium toxicity and its potential relevance to other diseases.
SESSION VII

OXYGEN TOXICITY

PROTECTION FROM PULMONARY OXYGEN TOXICITY BY TREATMENTS WITH LOW DOSES OF BACTERIAL PROTEIN.

1. Frank S. Young and D. McConnel, Jr.

A new approach was taken to study the effect of oxygen on the lungs. The animals were subjected to hyperoxia and then subjected to protracted periods of high oxygen exposure. The results indicated that the administration of low doses of bacterial protein protected the lungs against oxygen toxicity.

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OXYGEN TOXICITY

After the surviving animals from three experiments were maintained in room air for a week recovery period, special studies for fibrinous lung changes revealed a much reduced deposition of collagen and irregular fibrils in the lung of endotoxin-treated rats compared to the increased fibrils demonstrable in the untreated O2-exposed survivors. Analysis for lung hydroxyproline content showed a biochemical evidence for a reduction in chronic lung changes (fibrosis) in the endotoxin-treated animals.

We have further explored the biochemical basis by which endotoxin confers tolerance to hypoxia by measuring the effect on lung DNA, RNA, and the ratio of RNA to DNA. In rats breathing room air endotoxin caused an increase within 36 hrs in total lung DNA and RNA without any change in the RNA/DNA ratio; these findings persist for at least 72 hrs. In rats exposed to 95% O2 at one atm. but not given endotoxin, there is a smaller rise in total lung DNA and RNA but no change in the RNA/DNA ratio except at 72 hrs. In the few rats who survive without endotoxin treatment, in contrast, 1% O2-exposed rats given endotoxin, a significant rise in the ratio of RNA to DNA occurs by 48 hrs. of O2 exposure. This suggests an "activation" of the lung to increased cell division plus biochemical activity.

We conclude that 1) endotoxin confers protection against acute O2 toxicity even when given in a single dose (300 mg/kg or 3/10th LD50 level) as late as 36 hours after the onset of O2 exposure; 2) the antioxidant enzyme of the lung - ribose, CAT, and O2 play an important role in the protective effect produced by endotoxin; and 3) endotoxin treatment may protect against the delayed (fibrotic) changes which follow prolonged O2 damage. We suggest that endotoxin acts as a mitigator in the lung (increasing DNA and reducing the ability of activated cells to respond to hypoxia); free radical action in the endotoxin-treated O2-challenged animal.

Studies to further define the mechanism for the marked protective action of endotoxin against pulmonary O2 toxicity may hopefully lead to the development of still weaker agents with similar protective actions but perhaps less toxic potential than endotoxin itself, agents that may be of future clinical use in helping to circumvent the lung injury associated with prolonged treatment with life-giving O2.

Acknowledgements: initial studies with endotoxin were performed in cooperation with Dr. Robert W. Roberts, Department of Pharmacology and Pediatrics, University of Iowa Medical School, to whom the authors express their appreciation and gratitude.

**Enhancement of Pulmonary Defining Capacity After Brief Hypoxia Dives with High O2 Level During Decompression.**

High O2 pressure during high altitude decompression can be used to enhance the oxygen transport capacity of the lung. This was demonstrated by measuring the pulmonary diffusing capacity (Dl) of a group of subjects who were exposed to a hypoxic environment and then subjected to a rapid decompression. The Dl was measured before and after the exposure, and the results showed a significant increase in Dl after the exposure.

The beneficial effects of breathing oxygen during a decompression have been recognized. However, it is very little known about the optimum oxygen level for a long exposure to high pressure. The calculation of oxygen and the decrease in formed vital capacity had been shown satisfactory when the FIO2 is low and when oxygen is combined with other gases. For long dives saturation dives with helium it is recommended not to exceed 4 days with FIO2 of 0.6 or 0.8.

We studied the evolution of carbon monoxide lung diffusing capacity (DlCO) after two saturation dives. The first one was a 45 m depth saturation dive with inflation at 350 meters in open sea. The profile of this dive during the 4 days of decompression was a series of decrements function curves from 0.0 to 0.4 ATA. The 6 divers were subjected to decompression mixtures at the end of decompression. The second dive was a 45 m depth saturation dive with a profile of 45 m depth, the 4 days of decompression was an exponential decrement function of pressure from 0.6 to 0.4 ATA, 4 of 8 divers were subjected to decompression mixtures in series of 124 mm Hg. 3 to 4 times a day during 7 days after the start of decompression and the last two days of decompression. We measured these by SAPO directly after the subject at test and breathing a mixture of 100% P02 of 14 l/min.

Compared to control measurements, DlCO decreased in all but one subject during the first 3 days of measurements obtained 12/14 by after termination of the dive. The range was 8.5 to 20.5 with a mean decrease for all subjects of 4.1 l/min (B). At the time of follow-up measurements determined 3-5 days after termination of the dive, DlCO measured on 2 subjects was below the control values in all but one subject, compared to control values the mean decrease was 17.8% with a range between 5 to 52.7 (B). Two weeks after the termination of the dive the DlCO was below the control value in all but one subject. Compared to control values the mean decrease was 10.8% with a range between 11 to 26.5.

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The abnormal changes in DlCO two weeks after the termination of the dive indicate changes in pulmonary function which are slowly reversible. For the determination of optimal oxygen levels for a long exposure to high pressure it is necessary to consider the oxygen transport, the oxygen time, and the physiological sensitivity of the divers but leads and potentiometric studies.
SESSION VIII

PSYCHOMOTOR PERFORMANCE
AND HIGH PRESSURE NERVOUS SYNDROME

A THEORY OF EVENT-RELATED RESPONSES
Boris Portrait
York University, 4700 Keele St., Toronto, Ont., Canada.

The approach to the analysis of the behavioral effects of stress is to determine the processing of one of the main components of the various information processing mechanisms that control performance. If a pattern of effects is found, it is established, beyond that performance on complex tasks can be predicted. The focus is on the number of studies using either hypnotizable or R.O. (office worker) which can be interpreted in terms of this model of its description and form of a coherent pattern.

Talismans affects the kinesthetic system (Cheney, et al., 1972) but not vision (Fischer, 1972) or audition (Foster, in press). Unanimous increased reaction time by a considerable amount, irrespective of the number of trials in a single task (Sperling, 1964) and irrespective of the task of the animal in the visual recognition task (Sperling, et al., 1979). On the other hand, in a task where a response was required to precisely timed area of digit pad, a proportion increase in reaction time was found as a function of set (Stuhle and Fixler, 1965). Following the reasoning of Stuhle (1965), the lack of an interaction in the visual and visual recognition tasks and its presence in the digit reaction task implies a different effect on some aspect of memory processing but not in California response or visual processing. Memory and learning deficits in this state may have represented a number of workers. This evidence has been generalized by Foster, et al., 1979, who argued that this effect related to the changes in the visual reaction time and deficit that was (short-term memory) is unaffected.

The purpose of this paper is to explore the effects of nort on a neglected but important information processing checking attention. The model, to propose a model of nort effects on the basis of the current evidence.

In the first experimental steps, several subjects were required to produce a list of words presented in a random order and in a crossover order, or in an invariant condition. In the other order when breathing either 15% or air. A recognition paradigm was used to test recall and the results were illustrated in Fig. 1. A parametric effect is apparent. The distinctive habit of opposite effect on recall when breathing the noradrenaline mixture time was made in these observations for these results. The first is that nort affects the degree of recognition of the most effective of the two lists, the second is that the distinctive habit of opposite effect of the noradrenaline is blocked by this condition in some manner. These hypotheses were tested in a second experiment which followed a similar paradigm to the first but included the following conditions: 1) condition of target words in the list during presentation, 2) recall of the list after presentation, 1) recognition of words after presentation. The results from this experiment suggest that the second effect is related to the direction of attention.

The present evidence suggests that, at least under moderate levels, noradrenaline has a remarkably specific effect on some mechanisms, namely the kinesthetic memory system and certain other memory systems, namely the visual recognition system (Sperling, et al., 1979). A model which takes these facts into account and which can explain a wide range of pharaco effects involves three assumptions. First, nort causes a slowing in the rate at which information enters to some extent into the visual memory system (Smith, et al., 1979). A critical mechanism in the information processing model (Cheney, 1972, 1977; Stuhle, 1977) is that nort causes a slowing in the rate at which information enters to some extent into the visual memory system. A second assumption is that the disruption in the rate at which information enters to some extent into the visual memory system is related to a shift in the sensory sensitivity criterion (K entsik, 1971) rather than the interaction of some processing mechanisms.

[Diagram of experimental setup]

**APPENDIX**

ABSTRACT OF THE HIRE PSYCHOPHYSIOLOGICAL STUDIES: A NEW METHOD IN MEASURING PSYCHIC STRESS AND STRESS INDUCED SOMATOFORM DISORDERS

The present study was designed to evaluate the effects of stress on the physiological responses of individuals. The results of the study suggest that stress is associated with increased heart rate, respiratory rate, and blood pressure. The findings suggest that the stress response is mediated by the sympathetic nervous system and that stress may play a role in the development of somatoform disorders.

[Diagram of experimental setup]
PSYCHOMOTOR PERFORMANCE AND HIGH PRESSURE NERVOUS SYSTEM

The onset of tremor, as the pressure is increased, can be seen as an abrupt burst of tremor, lasting for up to 900 ms over every two seconds. These tremor bursts are characterized by high frequency, low amplitude, and no observable tremor threshold at the onset of the tremor burst. The tremor intensity is not consistent with the tremor frequency, and we have found it to remain constant over a period of 0-10 Hz. An examination of the tremor shows a significant decrease in tremor intensity during the state of the tremor and suggests a reduction in amplitude, followed by abolition of the basic tremor frequency.

Tremor is a minor stimulus in the important parameter used in the study of BPNS, but its measurement is very difficult to quantify. Problems include: distinguishing it from microseismic (including small microseismic), movement, manual, small stimulus, and the unfamiliarity of the recording task. A classification analysis in terms of both amplitude and geometry for a new classification technique which requires manual adjustment during the course of the high pressure experiment. The need to define the features of independence of the natural variability must be evaluated, temperature, and high pressure conditions.

We report our findings with a simple strain gauge device specifically developed for the purpose, which appears to overshadow the majority of the above constraints and which we are now using in the pharmacological studies of BPNS.

References will appear in PAPER.

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References will appear in PAPER.
SESSION VII

PSYCHOMOTOR PERFORMANCE AND HIGH PRESSURE NERVOUS SYNDROME

Eight subjects were selected to make the dive to 450 meters, three from group 0 and 1, and two from group 2. Three of the eight, two from group 0 and one from group 2, were resuscitated to 100 meters with an H2-N2-O2 mixture (90-3.5-6.5%)

The subjects were presented at the start of the test. The EEG tests were performed immediately after the subjects were resuscitated to 100 meters. The EEG tests were performed during confinement (duration 48 hours; H2: 0.6%; N2: 99.4%; O2: 0.0%). During the confinement, the EEG test was performed every 60 minutes.

Results:

1) EEG

a) At arrival at the surface, the EEG modifications found during this dive compared to those obtained during the trials dives to 100 m, gave the following observations:

- The two subjects who were resuscitated to 100 meters (group 2) show a marked increase in the activity of the EEG at 450 m (0000 and 0035).

- In the two subjects of group 2, the power spectrum of the theta activity clearly increases and is located between 10 and 20.5 m.

b) During the stay at 450 m, the EEG record in the same way in all the subjects so that at the end of the stay, they remain classified as they were at the time of arrival at the bottom.

2) Psychomotor performance

- The tests provided by psychomotor tests show that on the group level, average variations in performance between the surface and 100 m, and between the surface and 450 m, are a function of the depth.

- The results are not thus on an individual level. The great inter-individual variation at 100 meters does not exist at 450 meters where the subjects have a more homogenous behavior.

- If the subjects are classified by the distance between the two situations, the classification varies little from the surface to 450 meters.

Conclusions:

- On the basis of the psychomotor tests, it is possible to predict that there will be a worsening in the performance of all the subjects. However, there are subjects with an inter-individual difference at the surface with or without hypoxia and 450 meters that it is impossible to predict the behavior of each subject at 450 meters without considering constraints that influence this variability.

- The EEG behavior of a subject at 450 meters can be predicted by a dive to 100 meters with a rapid decompression as if the same type of mixture was used. The subjects who present the greatest modifications at 100 meters with the H2-N2-O2 mixture will also show the greatest modifications at 450 meters with the H2-N2-O2 mixture. The subjects who present the least modifications at 100 meters with the H2-N2-O2 mixture will also those who will be the most likely to have the least modifications at 450 meters with the H2-N2-O2 mixture, but this is not always true.

- The trial at 100 meters with the H2-N2-O2 mixture is not sufficient; it would be necessary to have a trial at 450 meters with the same mixture so that result at 450 meters. We have seen that a subject could have little modification of the EEG at 100 meters with the H2-N2-O2 mixture, and show substantial modifications at the greater depth, while the EEG remains stable.

- These results suggest that the subject reacts differently to pressure as can be seen in their EEG, their clinical symptoms, and their performance.

- Furthermore, in a given subject, the sensitivity of each of these symptoms may differ according to the model of compression as well as the gas mixture used at the pressure level, which remains to be defined in the subject who will present the most useful in selecting the best divers to already reached depths, especially between 450 and 600 meters, but also other divers who could react differently to pressure at greater depth.

- It would be interesting to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement with the H2-N2-O2 mixture, and to analyze the EEG modifications in the light of the psychological test to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement with the H2-N2-O2 mixture, and to analyze the EEG modifications in the light of the psychological test to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement with the H2-N2-O2 mixture, and to analyze the EEG modifications in the light of the psychological test to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement with the H2-N2-O2 mixture, and to analyze the EEG modifications in the light of the psychological test to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement with the H2-N2-O2 mixture, and to analyze the EEG modifications in the light of the psychological test to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement with the H2-N2-O2 mixture, and to analyze the EEG modifications in the light of the psychological test to compare the EEG symptoms of the psychological test performed at 450 meters with those obtained at the surface and during confinement.
SESSION IX

Cardio-respiratory Effects

Acute, comparable to that found in older adults, is not unique, and was not given.

Pendulum, a phenomenon that is long ago more subtle and can be difficult to observe, involves physiological dead space, impairs gas exchange (Ventura, 1974), and results in increased pulmonary ventilation rates at some respiratory frequencies, but reduces it at zero and in other respiratory frequencies. Effects of renin are not clear, but they further reduce the consequences of meal-induced ventilation rates at those frequencies.

Initially, some recent work to reiterate the present theoretical observations. In two studies using either the J android or J androids, Bocelli et al. (1984) and Taylor et al. (1984), acute was seen to decrease. But, in both studies respiratory frequency (f1 to f4 BPM) were lower than those expected to produce a major acute effect. Furthermore, there are unobserved density-dependent phenomena not included in the model that may influence the results. Thus, it is not surprising that these studies did not find acute in 2.5 L per minute flow rates, which correspond to a normal tidal volume.

Inhaled and inspired air was the highest at a (41 L per minute) and therefore might have been the most susceptible to acute effects if the study had been related to a wide range of ventilation rates. The inhaled fraction presented in this paper was therefore likely to explain some of the variability seen in other works.

Unfortunately, the possible model is limited to the scatter of variables that may be considered. Consequently, the degree of scatter in the data and the results of each model expected to produce a major acute effect are not clear. While more data may be relevant, it is not surprising that these studies did not find acute in 2.5 L per minute flow rates, which correspond to a normal tidal volume.

Figures 1, 2, and 3 show the clearance of inhaled air as a function of frequency and tidal volume. The ventilation rate at the start of the inspiration (Vtrig) is shown to be a function of the tidal volume and the frequency of the inspiration.

In conclusion, acute, comparable to that found in older adults, is not unique, and was not given.
CARDIO-RESPIRATORY EFFECTS

In the other two examples of pressure temperature stress an oscillation occurred in the conduction time between the stimulating and recording sites. These were sinus node activity at 100 A.M., suggesting that moderate hypo- and hyperthermia, and profound hypoxia, were alternately disturbed at 12.5 degrees a.m. Following these, the tissue at 30% abolished the arrhythmia.

Combinations of rate stress and pressure are also potentially arrhythmic. As noted in Table II, arrhythmias developed in 24% of the rabbit atria (11) and 55% of dog atrial preparations (12), always in conjunction with rate changes and higher pressures. Asynchronous atrial conduction appeared as a 3:2 block at 200 A.M. when the pacing rate was increased to 200 impulses/min, increasing the pressure during the rate of the intraparaseptal atrial stimulus. At 150 A.M. the 2:1 block was evident at a slower rate of 150 impulses/min. Increasing the rate to 250 impulses/min increased the conduction defect, resulting in a 2:1 block.

A 2:1 conduction block was also encountered in 2 atrial fiber preparations (see V in Table II) subjected to rapid stimulation at 250 A.M. In these fibers, the A.M. of the conducted impulse was markedly longer than the stimulus cycle length (2850 m.sec). This, the rate was delivered during the relative refractory period of the tissue, and therefore unable to evoke a propagating response. The resultant dropped beat enabled the tissue to recover sufficiently to respond to the subsequent stimulus, establishing the 2:1 conduction pattern.

Other rate and pressure related arrhythmias in atrial fibers (see Table II) were identified by an oscillation in impulse conduction time. In these examples, every other stimulus pulse occurred during the terminal repolarization phase of the preceding cardiac action potential. The resultant response was initiated from a depolarized level of normal potential. As a result, this action potential had a reduced duration and conducted more slowly than the preceding response. The 3:2 of the "slow" response was uncertain such that repolarization was complete prior to the occurrence of the next stimulus. The next response, originating from the fully repolarized membrane, propagated more effectively. Thus, an oscillatory conduction pattern was thereby established.

The present findings offer insight into the arrhythmic potential of elevated intraventricular pressure and velocity during vascular blocking, the safety margin for cardiac conduction by decreasing ventricular rate, and prolonging the refractory period. These pressure-induced arrhythmias may be of sufficient degree, under certain circumstances, to lead to the development of overt arrhythmias.

In terms of resistance to pressure, frequency has an additive effect. This effect is further reduced the safety margin for conduction. The fact is evident in the present research, where arrhythmias were encountered at 150-150 A.M. when either the temperature was lowered to 250C or when the stimulus rate exceeded 250 impulses/min.

This may have direct application in diving trauma. Typically, depth, work load, and hypothermia are three of the primary safety concerns during open-water diving. The effects of various factors can be studied to determine those factors that are critical to human physiology, emphasizing the importance of maintaining a balance between the body's metabolic requirements and the environmental conditions.

Arrhythmia will appear in Proceedings, Figure 1, Table 1, follow.

SESSION IX

TABLE II

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Pressure</th>
<th>Temp.</th>
<th>Rate</th>
<th>Arrhythmia</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Sinus node</td>
<td>150 A.M.</td>
<td>25°C</td>
<td>150</td>
<td>3:2 block</td>
<td>Present report</td>
</tr>
<tr>
<td>II. Atrial</td>
<td>150 A.M.</td>
<td>25°C</td>
<td>150</td>
<td>2:1 block</td>
<td>Present report</td>
</tr>
<tr>
<td>III. Darkling</td>
<td>150 A.M.</td>
<td>25°C</td>
<td>150</td>
<td>2:1 block</td>
<td>Present report</td>
</tr>
<tr>
<td>IV. Darkling</td>
<td>150 A.M.</td>
<td>25°C</td>
<td>150</td>
<td>2:1 block</td>
<td>Present report</td>
</tr>
</tbody>
</table>

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The subject, a 33-year-old healthy male, engaged in physical activity, ranged from 20 to 40, with normal results. He had a single lung function test and site calculated over a heat source at the rate of 0.4 m/sec. Blood alcohol levels were estimated by sampling and analysis. Alcohol was consumed after the initial alcohol test, and subjects tested for 6 hours. The subject was tested cooperatively by the heat source and after the rate of 0.4 m/sec. Blood alcohol levels were measured by taking a blood sample at the rate of 0.4 m/sec. Blood alcohol levels were measured in the second test and a blood sample was taken after the test. Blood alcohol levels were measured at the rate of 0.4 m/sec. Blood alcohol levels were measured at the rate of 0.4 m/sec. Blood alcohol levels were measured at the rate of 0.4 m/sec.

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SESSION IX

CARDIO-RESPIRATORY EFFECTS

Influence of the ventilation defects on the cardio-respiratory system was studied in a group of healthy subjects. The subjects were divided into two groups: Group A, consisting of 10 subjects without any defects; and Group B, consisting of 10 subjects with various defects. The defects included chronic bronchitis, emphysema, and asthma. The subjects were exposed to various experimental conditions, such as hyperventilation and hypoventilation, at different levels of exercise intensity. The results showed that the defects significantly affected the cardio-respiratory system, with Group B subjects exhibiting lower heart rates and higher oxygen consumption than Group A subjects. The study concluded that the defects significantly impaired the cardio-respiratory system, and that further research is needed to understand the underlying mechanisms and develop effective treatments.

REGULATION AND FREQUENCY OF HEART RATE DURING OPEN-CHAIR SATURATION DIVING

S. M. Gougeon and A. L. Inglis,
Naval Medical Institute and Institute of Aviation Medicine, Nieuport, Yegatania.

The ECG and instantaneous heart rate of a group of divers during a four-day open-air saturation diving with the air in the 120% O2 with an increase in the heart rate recorded on an electronic heart rate recorder. The heart rate was determined by the instantaneous heart rate measurements of the divers. The ECG was recorded by a synchronized heart rate monitor. The results show that the instantaneous heart rate was significantly lower in the divers during saturation diving than in the control group. The heart rate measurements were performed at different levels of exercise intensity, and the results showed that the instantaneous heart rate was significantly lower during saturation diving than during control conditions. The study concluded that saturation diving significantly decreased the instantaneous heart rate, and that further research is needed to understand the underlying mechanisms and develop effective treatments.
CARDIO-RESPIRATORY EFFECTS

In this study, the effects of respiration on various physiological variables were investigated. Respiratory movements were associated with changes in cardiac output, blood pressure, and heart rate. The respiratory cycle produced significant variations in arterial blood pressure, which were most pronounced during expiration.

In summary, the results of this study suggest that respiration has a significant impact on cardiovascular function. Further research is needed to fully understand the complex interactions between the respiratory and cardiovascular systems.
hypertonic (15% NaCl) at 1 ATA positive. It should be pointed out that the pre-immersion arterial flow and speciality were quite comparable in all three experimental conditions (i.e., 1 ATA positive, 31 ATA, and 1 ATA positive). The relative clearance of radioactivity during immersion at 31 ATA positive was only 10% greater than that at 3 ATA positive, indicating that the observed differences in the immersion studies are not due to differences in the glomerular filtration rate.

Despite such large differences in the degree of immersion studies, there were no differences in the rate of excretion of NaCl and in total cardiac index under the three experimental conditions. This indicates that the fractional excretion of NaCl (i.e., the relative clearance of radioactivity) at 31 ATA and at 1 ATA positive was not different from that at 1 ATA. A possible difference in the excretion of NaCl during immersion at 31 ATA and at 1 ATA positive led to a significant difference in the free water clearance. The latter value was around 2.0 ml/min during immersion in all experiments, and was not statistically different from 1.5, 0.5 and 1.2 ml/min during the first hour of immersion at 1 ATA positive, 31 ATA, and 1 ATA positive, respectively. This indicates that the free water excretion during 31 ATA immersion was about 30% higher than at 1 ATA positive. The total amount of sodium excretion was negatively correlated with the urinary NaCl excretion (r = -0.60, p < 0.005). However, the NaCl was not always accompanied by an increase in urinary NaCl excretion. Although the underlying mechanisms for this phenomenon have not been specified, it may be related to the fact that the degree of inorganic excretion during immersion (as indicated by the NaCl clearance) was less than the total cardiac index, and the cardiac index was lower at 31 ATA and 1 ATA positive than at 1 ATA positive. These findings also suggest that the sodium retention during immersion is different from that for the inhibition of ADH (which is considered to be primarily responsible for immersion diuresis).

![Image]

**Fig. 1:** The effect of head-out immersion of cardiac index at 1 ATA air (pre- and positive) and 31 ATA (positive) and 31 ATA (negative). The cardiac index was calculated from the values of heart rate, stroke volume, and mean arterial blood pressure. Each point represents the mean of 4 subjects.

**CARDIO-RESPIRATORY EFFECTS**

**SESSION IX CARDIO-RESPIRATORY EFFECTS**

The effect of water temperature on vital capacity during head-out immersion.


**Table 1:** Vital capacity (VC) in the sitting position during and following head-out immersion at 31 ATA and 1 ATA positive. Data are expressed as a percentage of pre-immersion value. Thirteen healthy male subjects were studied. The mean VC was 43.9 ± 0.5% (SEM) of the pre-immersion value. The VC decreased by 49.3 ± 1.4% (SEM) at 31 ATA and by 24.4 ± 1.2% (SEM) at 1 ATA positive.

**Fig. 2:** The effect of head-out immersion on vital capacity during head-out immersion at 31 ATA and 1 ATA positive. The vital capacity decreased by 51.0 ± 1.3% (SEM) at 31 ATA and by 26.0 ± 1.5% (SEM) at 1 ATA positive. The decrease was significant at 31 ATA (p < 0.001) but was not significant at 1 ATA positive (p > 0.05).

**Table 2:** Vital capacity (VC) in the sitting position during and following head-out immersion at 31 ATA and 1 ATA positive. Data are expressed as a percentage of pre-immersion value. Thirteen healthy male subjects were studied. The mean VC was 43.9 ± 0.5% (SEM) of the pre-immersion value. The VC decreased by 49.3 ± 1.4% (SEM) at 31 ATA and by 24.4 ± 1.2% (SEM) at 1 ATA positive.
CARDIO-RESPIRATORY EFFECTS

OXYGEN SUFFICIENCY AND UTILIZATION WITHIN THE CELL

SESSION IX

SESSION X

Fig 2: Vital capacity (VC) in the sitting position during non-immersion (dry) and head-out immersion in 20°C water. The effects of periods of arterial and venous occlusions by tourniquets on area and volume and the Valsalva maneuver are shown. The values are means ± S.E. from 3 to 4 experiments (see abstract) in 3 or 4 subjects. Dashed lines indicate mean values for measurements obtained during relaxed time up.

Discussion: The immediate 4-7% reduction in VC upon immersion was unaffected by widely differing water temperatures (24°C, 32°C and 40°C). The lowering of the lung capacity to solid air was presumably caused by a sudden redistribution of blood from the peripheral into the thoracic vessels in this position during some support. It is a fact that arterial stasis on the extremities prevented (by first drop in VC in 20°C water. The slight reduction (2%) in VC despite the use of an external heart pump may be ascribed to blood redistribution prevented by the tourniquets. The VC in water of neutral temperature (30°C) remained at the initial immersion levels. The mean reduction of 4-6% in agreement with 39 other studies yielded an average VC reduction of 5.1% (cf. 2). It may therefore be concluded that the initial redistribution of blood due to hydrostatic immersion effects, there were no further major adjustments in blood distribution in the 30°C water. However, such adjustments apparently occurred in the lungs and the warm water. After the initial VC drop in VC by 10% water there was a further reduction by 2.7% during the immersion period. The difference in the lower change is still of the order of 0.05% to the hydrostatic effect on VC evident in 30°C water, there was possibly an element of cold vasoconstriction in 20°C water accounting for part of the term and increasing drop in VC. The possibility remains to be examined, however, that some of the VC reduction toward the end of the exposure to 20°C water was caused by lessening of neurovascular performance.

The crucial role of blood redistribution for the observed effects is further borne out by the gains in VC achieved by the application of venous stasis and the Valsalva maneuver (5.2%) and 6.2%, respectively). The effects of the venous stasis in to allow blood to accumulate distal to the tourniquets. The increase intrathoracic pressure generated by the Valsalva maneuver forces blood out of the chest cavity (cf. 4). At the initial drop in VC by 5.1% upon immersion in 40°C water the VC rapidly recovered almost to the preimmersion level. It is likely that this reflected, after the initial increase in intrathoracic blood volume, a redistribution of blood from the chest cavity to peripheral vessels which were subject to thermodilutional vasoconstriction.

Nevertheless, when the subjects were protected by a wet suit and comfortable in 10°C water the loss in VC was the same (13.7), and equally rapid, as it was (10.2%) when they were naked and shivering in 20°C water. It is therefore reasonable to conclude that both part of the intrathoracic blood redistribution which depends on peripheral cold vasoconstriction and that caused by hydrostatic effects of the immersion were of the same magnitude in the two conditions.

It follows from the present observations that when lung volumes are measured during immersion, and possibly non-immersion, the subject's thermal situation should be considered. In addition, to the extent that intrathoracic blood pooling has secondary effects on cardiorespiratory function, e.g. causing air trapping, changes in compliance, changes in cardiac output, these effects may also be modified by changes in thermal stress. One should also note that warm water immersion, the normally through peripheral vasoconstriction most completely counteracted the hydrostatic effect evidenced throughout the neutral temperature range in this and another work. After the initial dip the hydrostatic load during immersion may be overcome by intrathoracic hydrostatics, favoring sites of particular importance in the present study is the new piece of evidence presented by the observations that physically significant cooling may occur in the suit shaded subject in the absence of subjective sensations and shivering (cf. 8).

References and acknowledgments will appear in PROCEEDINGS.

Fig. 1: Cardiac output (CO) in the absence of other major drug interventions. Aortic chemoreceptors were activated by aortic occlusion.

Fig. 2: Cardiac chemoreceptor activity in the absence of other major drug interventions.
SESSION X

OXYGEN SUFFICIENCY AND UTILIZATION WITHIN THE CELL

The progression of the filling with perfusion duration for serial patelastrian and tulitinae muscles is shown by Table 1. Filled fraction (F) for individual fields varied widely, particularly for short perfusions, but means increased regularly with time. For small increments in H, between 1 and 5% per unit time were observed.

The fraction of open capillaries occurring in this definition fell between 0.25 and 0.4 for four fields of each tissue and was 0.32 for the fields of all capillaries. Mean 0.33 for open capillaries were inversely related to H, and varied from 0.6 to 1.2. Distribution was almost linear to the log-normal pattern (Fig. 4-1-3) and variability was increased as fast for milar tissues. Log range from 0.10 to 0.81, 0.10 to 0.31 (a < 0.3) is equivalent to 0% of individual values falling between 0.3 and 1.0 and to 0.2 and 0.5. If the distribution patterns represented by these sections are left in time, tissue volume lying beyond the unit distribution range (0.25, equivalent to radius of a rough cylinder) must determine the critical parameters for blood supply to these tissues.

The contour maps for both ventilatory and tulitinae muscles show "islands" of well-filled capillary networks surrounded by "lakes" of tissue lying from open capillaries. In mixed tissues, the islands are clustered around groups of F 50 and 30 fibers. However, groups of these cells are represented proportionally in the remote areas. Although the arrangement of the entire capillary is related to the arrangement of muscle fiber types in plant musculature, the distribution of open capillaries appears to result from characteristics of the vascular supply to these muscle fibers, and is not associated with localization of the different fiber types.

(Supported by NIMHD Grant R.1998)

References will appear in PAPERBACK.

Fig. 1 follows

<table>
<thead>
<tr>
<th><strong>TABLE 1</strong></th>
<th>Fraction of perfused capillaries (mean ± SD)  (N fields counted)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PERFUSION</strong></td>
<td>0.25</td>
</tr>
<tr>
<td><strong>Field</strong></td>
<td>0.16-0.20</td>
</tr>
<tr>
<td><strong>Saline</strong></td>
<td>0.14-0.16</td>
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</tbody>
</table>

Location of tissue mass with respect to distribution for the nearest capillaries was evaluated by plotting contour intervals for each field at multiples of h mean SD (Fig. 1-1). For mixed tissues, F 64 to 75% of each field type present (F 50 male, F 66 female, F 70 mixed), and the same volume, F 90-100 different each, 0-100, 0-100 mixed). Mean fiber diameters ranged from 0.08 to 0.21 mm (mean SD 0.11). Mean fiber diameters range from 7.6 to 49, the mean SD practically equal to 10% of the capillary diameter. Individual values were distributed approximately as log-normal the SD of the field is shown such as Fig. 1-1.1-1.1.

By plotting contour intervals for each field at multiples of h mean SD (Fig. 1-1.1), for mixed tissues, F 64 to 75% of each field type present (F 50 male, F 66 female, F 70 mixed), and the same volume, F 90-100 different each, 0-100, 0-100 mixed). Mean fiber diameters ranged from 0.08 to 0.21 mm (mean SD 0.11). Mean fiber diameters range from 7.6 to 49, the mean SD practically equal to 10% of the capillary diameter. Individual values were distributed approximately as log-normal the SD of the field is shown such as Fig. 1-1.1.1.

**SESSION X**

**Retinal blood with hypercapnia and hyperbony cyan (J. S. Neiss, J. S. Neiss, W. D. Neiss, B. J. Neiss)**

BLOOD DELIVERY WITH HYPERCAPNIA AND HYPERBOXY CYAN (J. S. Neiss, J. S. Neiss, W. D. Neiss, B. J. Neiss)

**Introduction**

Under hyperbony conditions it is theoretically possible to raise the Pao2 (arterial blood) to a level where oxygen extraction by the tissues does not take place in the tissue homology system in transit. The notion that a small amount of arterial blood in a large arterial oxygen extraction and its unique optical access. Using a reflection absorption technique and pressures into the hyperbony region, we have investigated the effect of increasing the partial pressure of inspired oxygen on the hyperbony saturation characteristics of arterial capillary blood (Fig. 4-1.1). In the left eye, more specifically the choroidal. Because essentially all of the oxygen from the retina is absorbed in the retina, this animal has been to the best of our knowledge, O2 is known to be able to penetrate to the choroid cells and it is possible to determine that oxygen tension at which the oxygen needs of the retina are met by physiological means, i.e., from tissue. This allows for the measurement of sequentially greater carbon dioxide pressures without sacrificing oxygenation in the systemic oxygen effect of extracorporeal shock wave treatment on hyperbony respiratory state could be, and was, investigated as well. Finally, the effect of these unusual respiratory conditions on retinal function has not been fully explored before. The goal of this study is to change the state of the "fasting blood." Additional observations were therefore made on the electroretinographic (ERG) activity which accompanied these gas changes.
OXYGEN SUFFICIENCY AND UTILIZATION WITHIN THE CELL

In order to study the effects of oxygen on cellular processes, researchers have developed techniques to measure oxygen levels within cells. One such method involves monitoring the oxygen tension in various parts of the cell, such as the mitochondria and the cytoplasm. This can be achieved through the use of specialized sensors and imaging techniques.

A key aspect of this research is understanding how oxygen changes affect cellular functions, particularly in the context of oxidative metabolism. For example, changes in oxygen levels can impact the rate of ATP production, which is crucial for cellular energy production. Additionally, oxygen levels can influence the expression of certain genes and the activity of enzymes.

In summary, understanding oxygen sufficiency and utilization within the cell is essential for advancing our knowledge of cellular biology and physiology. Further research in this area is likely to provide valuable insights into the mechanisms underlying cellular processes and may have implications for the treatment of various diseases, such as cancer and neurological disorders.
SESSION X

MATERIALS AND METHODS

A 10 gauge needle was inserted percutaneously into the left tibial metaphysis of a few tumor-bearing rabbits. The needle was removed, the tumor was excised, and the needle was reinjected into the same site. The injection was made in a sterile manner with the animal in the supine position. The needle was inserted through the skin into the intramuscular tissue, directed toward the tibia, and advanced with the needle point. The tumor was aspirated from the needle and placed in a sterile container. The injection was made in the same manner as the tumor was excised.

Histological examination of the tumor-bearing rabbit revealed a well-circumscribed, firm, gray, firm, and homogenous mass. The tumor was excised and placed in formalin solution for histological examination. The needle was removed, and the injection was made in the same manner as the tumor was excised.

RESULTS

Three animals were used in this experiment. Two animals were injected with the tumor-bearing rabbits, and one animal was injected with the normal bone. The normal bone was excised from the tibia of a normal rabbit. The injection was made in the same manner as the tumor was excised.

The results showed that the tumor-bearing rabbits had a significantly higher oxygen tension than the normal bone. The tumor-bearing rabbits also had a significantly higher oxygen tension than the normal bone. The oxygen tension was measured in millimeters of mercury (mm Hg) and was significantly higher in the tumor-bearing rabbits than in the normal bone.

DISCUSSION

Oxygen sufficiency and utilization within the cell...

Comparison of the different in percent of normal of 5, oxygen at 2 hours under one atmosphere of oxygen (n=10) and in tumor-bearing rabbits (n=5) showed that the difference between 5% and 20% was significant (p<0.05). The difference between 20% and 100% was not significant (p>0.05).

The viability of the PMN was assessed in all five oxygen tensions at relative viability of 100% at 2 hours as shown by the exclusion of trypan blue dye.

Mass spectrometer oxygen data...
OXYGEN SUFFICIENCY
AND UTILIZATION WITHIN THE CELL

Figure 2. Bactericidal killing of E. coli (E) by rabbit peritoneal leucocytes (PMN) with oxygen as a variable. Mean accumulated oxygen at different oxygen tensions. Ordinates represent percent original inoculum at 60 minutes in presence of 80% PMN and 20% controls without oxygen. Each line represents one of 4 experiments.
METABOLISM AND THERMAL PHYSIOLOGY

SESSION XI

Contribution of Metabolic and Respiratory Heat to Core Hypothermic Gain after Cold Water Immersion. M. L. Cown, F. A. Higley and J. B. Fitchett, Department of Kinesiology, Kent State University, Kent, Ohio. Coady and Adlibility

Rate of metabolism increases in cold water exposure. Inhalation of cold air increases respiratory heat production. The increase in respiratory heat production is proportional to the change in core temperature. The relative contribution of metabolic and respiratory heat to core temperature is determined by the rate of heat production and the rate of heat loss. The rate of heat production is determined by the metabolic rate and the respiratory rate. The rate of heat loss is determined by the core temperature and the environmental temperature.

In this study, the relative contribution of metabolic and respiratory heat to core temperature was determined using a metabolic calorimeter. The subjects were placed in a water tank and their metabolic rate and respiratory rate were measured. The rate of heat production was calculated from the metabolic rate and the respiratory rate.

The results of this study indicate that the relative contribution of metabolic and respiratory heat to core temperature is determined by the rate of heat production and the rate of heat loss. The rate of heat production is determined by the metabolic rate and the respiratory rate. The rate of heat loss is determined by the core temperature and the environmental temperature.

References will appear in PROCEEDINGS.
Molecular and Cellular Effects of Hydrostatic Pressure

The effects of hydrostatic pressure on cellular and molecular processes have been extensively studied in various organisms. Changes in pressure can alter the structure and function of biological membranes, affect protein folding and stability, and influence the activity of enzymes and other macromolecular systems. These effects are mediated through a variety of mechanisms, including changes in solvation, ionization, and partition coefficients of molecules.

In studies on fish species, it has been observed that moderate changes in hydrostatic pressure can alter the expression of certain genes and affect the dynamics of protein-protein interactions. For example, in salmonid fish, an increase in pressure has been found to induce the expression of genes involved in stress response pathways, such as heat shock proteins and antioxidant enzymes.

In mammals, hydrostatic pressure can also influence cellular processes. For instance, pressure-induced changes in intracellular pH can trigger signaling pathways and affect the activity of ion channels and transporters. Moreover, pressure can alter the localization and function of membrane receptors, leading to changes in cellular signaling.

The specific mechanisms by which hydrostatic pressure affects cellular and molecular processes are complex and depend on the pressure level, duration, and the specific organism or cell type involved. Further research is needed to fully understand the intricate interplay between pressure and cellular function at the molecular level.
MOLAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE

The observation that pressure above 100 atm does not alter the osmotic fragility of human red cells, and that the fragility is greater at 37°C than at 4°C, suggests that each has different effects at action. This we suggest that pressure alters the osmotic fragility of the membrane, whereas pressure protects the cell by increasing the osmotic fragility of the membrane. In the experiments above 100 atm, the effect of pressure below 100 atm is difficult to interpret.

The results of these experiments show that red cells become more sensitive to pressure at pressures above 100 atm and may be explained by pressure causing an extensive disruption of the protein network. Presumably the protein network is only partially reversibly denatured in ice water at 100 atm, indicating that such cells are more fragile in subsequent experiments. At these high pressures there may be additional pressures of interpretation due to temperature changes on compression and alterations to red cell unit differences.

References will appear in this volume.

Figures 1 and 2 follow.

Fig. 1. Human red cell equilibrium at 4°C, 21 atm and 37°C (†) and at pressure subjected to an osmotic head of 132 m0N.
Molecular and Cellular Effects of Hydrostatic Pressure

A mathematical analysis of high pressure and anaesthetic effects.

The pressure reversal of anesthetics in a well established phenomenon in both animal models and man. Since 1975, the quantitative data on the decrease of anaesthetic potency with increasing pressure have usually been analyzed in terms of the critical volume hypothesis. This predicted that there should be a linear relationship between the percentage increase in anaesthetic potency and the increase in pressure of the agent used. This has been demonstrated in the case of high pressure helium as an anaesthetic agent (Miller et al., 1975). It has also been shown that the increase in anaesthetic pressure (Miller et al., 1975) is different from that for the inhalational anaesthetics (Kaslo et al., 1975; Miller and Wilson, 1975). However, these last two studies are not in agreement as to whether the reversal in anaesthetic action occurs when all experimental errors are included.

1. Are the percentage increases in anaesthetic requirements non-linear when all experimental errors are included?

2. If they are non-linear, do they fit a mathematical model based on a simple extrapolation of the non-linear relationship (Kaslo et al., 1975; Miller et al., 1975)?

3. Alternatively, do they fit a more complex model which would predict additional effects as the pressure is increased and which would be applicable to all physiological systems (Muller, 1977; 1978)?

We have used the data obtained for the increase in anaesthetic pressure of the intravenous agents (Kaslo et al., 1975; Miller et al., 1975) to determine the effect of pressure on the anaesthetic action of the agents. The data were fitted to a linear relationship between the percentage increase in anaesthetic potency and the increase in pressure of the agent used. The results are shown in Fig. 2. The data were fitted to a linear relationship between the percentage increase in anaesthetic potency and the decrease in percentage change in anaesthetic potency. The data were fitted to a linear relationship between the percentage increase in anaesthetic potency and the decrease in percentage change in anaesthetic potency. The data were fitted to a linear relationship between the percentage increase in anaesthetic potency and the decrease in percentage change in anaesthetic potency.
SESSION XII

Molecular and Cellular Effects of Hydrostatic Pressure

As mentioned, hydrostatic pressure is generally considered to have negative biologic outcomes, and it attenuates the net division of animal cells. In contrast, it was observed that hydrostatic pressure is capable of enhancing the growth inhibition effects of low pressure on the growth of certain cultures. This enhancement is observed even at low pressures, although it is less pronounced at high pressures. Therefore, these observations suggest that hydrostatic pressure can have a complex effect on cell growth, with potential implications for biologic systems.

Further experiments indicated a similar enlarging effect for animal cells in the absence of pressure. It was observed that hydrostatic pressure is capable of enhancing the growth inhibition effects of low pressure on the growth of certain cultures. This enhancement is observed even at low pressures, although it is less pronounced at high pressures. Therefore, these observations suggest that hydrostatic pressure can have a complex effect on cell growth, with potential implications for biologic systems.

### References


### Figures

- Figure 1: Schematic representation of hydrostatic pressure effects on cell growth. The figure illustrates the relationship between hydrostatic pressure and cell growth, showing that pressure can have a direct effect on cell proliferation.
- Figure 2: Enlarging effect of hydrostatic pressure on cell growth. The figure demonstrates that hydrostatic pressure enhances the growth inhibition effects of low pressure on cell growth, with implications for biologic systems.
MOL.ECULAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE

**Figure 1**: Hypothalamic response profile.

**Figure 2**: Effects of different arterial hydrostatic pressures on blood glucose, lactate, acetate, lactate, and glycogen expressed as percentage of control at 0 time.

**Table 1**: Summary of experimental data.

In conclusion, the effects of hydrostatic pressure on the hypothalamus have been investigated using a novel technique. The results indicate that hydrostatic pressure can significantly alter the hypothalamic response to various stimuli. Further studies are needed to fully understand the mechanisms involved.

SESSION XII

The effects of hydrostatic pressure on the hypothalamus have been studied using a novel technique. The results indicate that hydrostatic pressure can significantly alter the hypothalamic response to various stimuli. Further studies are needed to fully understand the mechanisms involved.

**Figure 3**: Effects of hydrostatic pressure on the hypothalamus.

**Figure 4**: Summary of experimental data.
SESSION XIII

INERT GAS EXCHANGE AND DECOMPRESSION

EVALUATION OF DECOMPRESSION TABLES AT A MODEL DESCRIBING DYNAMIC IN TISSUE. J. Hakii, A. Talmon, and D. Rabin. Dept. of Chemical Engineering and Div. of Physiology & Biophysics, Faculty of Medicine, Hebrew University, Jerusalem, Israel.

Decompression following a hyperbaric exposure may cause formation of gas bubbles in tissue and blood. It is widely accepted that this gas phase in the case of inert gases in decompression bubbles. It is often suggested that the formation of bubbles could also occur during the decompression process carried out by following conventional diving tables, in which the diver is always without decompression.

We believe that bubble formation and its dynamics are the key to a correct rationale in computing decompression tables. To pursue this concept further, we have developed in this paper a mathematical model which describes bubble dynamics in tissues, in relation to environmental parameters characteristic of a dive, such as bottom time and depth.

We assume that a gas phase is already present in the tissue undergoing decompression, and potentially so even under normal conditions due to the low permeability of the tissue. This gas phase is considered to be first dissolved in the tissue and then bubbles that grow up by diffusion by physical expansion and then diffusion of inert gas from containing supercritical fluid. At the same time bubble formation by diffusion from supercritical fluid is also possible due to the adverse conditions of the tissue. Moreover, not only the growth of bubbles but also the overall rate of bubble formation is affected by the environmental conditions.

We assume the bubbles to be spherical and to be connected to the infinite medium of supercritical fluid, by the factors taken into account in a properly distributed mass volume. A mass balance on the bubble yield (after defining a conservative form factor to be of zero significance) an expression that can be written in a dimensionless form as:

\[ \frac{d}{dt} \left( \frac{1}{\text{r}} \right) = R - F \]

where \( \frac{1}{\text{r}} \) is the dimensionless bubble radius, \( R \) is the rate of bubble formation, and \( F \) is the fraction of bubble destruction.

\[ \text{r} = \frac{\text{r}_0}{\text{r}_0 + \text{t}} \]

where \( \text{r}_0 \) is the initial bubble radius, \( \text{t} \) is the time, \( \alpha \) is the cavity coefficient, and \( \beta \) is the dimensionless radial coordinate.

\[ \frac{\partial}{\partial \text{r}} \left( \frac{\alpha}{\beta} \right) = \frac{1}{\text{r}} \frac{\partial}{\partial \text{r}} \left( \frac{\beta}{\alpha} \right) \]

where \( \alpha \) and \( \beta \) are the cavity coefficient and the diffusion parameter, respectively.

\[ \frac{\partial}{\partial \text{r}} \left( \frac{\text{r}}{\alpha} \right) = \frac{1}{\text{r}} \frac{\partial}{\partial \text{r}} \left( \frac{\beta}{\text{r}} \right) \]

The dimensionless pressure in the bubbles is given by:

\[ \frac{p}{p_0} = \frac{1}{\text{r}} \frac{\partial}{\partial \text{r}} \left( \frac{\beta}{\alpha} \right) \]

where \( p \) and \( p_0 \) are the pressure in the bubble and in the infinite medium, respectively.
where the three terms in the numerator stand for the inherent unsaturation, tissue elasticity and the surface tension. $K$ is the elastic modulus of the tissue and $s$ is the surface tension.

An expression for $F_B$ in the case of air breathing, is obtained from Hills and Lemberger (1982).

$$ F_B = F_B^0 + \frac{K}{2s} \left( \frac{F_B}{F_B^0} \right)^{1/2} $$

Numerical integration of the above equation yields $F_B(t)$ for a step change in alveolar inert gas tension, assuming steady state values of $F_B^0$ and $K$. This model can predict the behaviour of a decompressed bubble for various degrees and saturation fractions ($F_B$), for different breathing gas mixtures, and can be used for the evaluation of decompression tables.

Our basic assumption is that a lung-like tissue becomes overdistended at pressure in a semi-solid tissue exceeds a critical value, if $F_B$ is the concentration of bubble in tissue and $s$ the added pressure of the gas phase volume then we have (Hills, 1985):

$$ s = \frac{1}{2} F_B^0 $$

If the critical $s$ for inducing symptoms is 11 mmHg (after Leman and Saunders, 1982), then $s_B$ can be easily estimated.

Thus, bubble radius change following a step decompression can be calculated and symptoms can be expected when $s = 5$ mmHg.

To illustrate this procedure we present two figures. Fig. 1 shows the pattern of bubble radius change after a saturation pressure ($F_B(t)$) is 30 mmHg. The unperturbed model, as it includes time at 3 m and 20 min more time spent at the shallow stop, is typical of conventional decompression tables.

Fig. 2 shows equivalent patterns after a 30 min exposure at saturation fraction of 0.03 and 0.06. The effect of a proper uptake factor was allowed by simply choosing $F_B$ values. The first stages of the decompression reveal bubble resolution because of the low degree of supersaturation, but upon further decompression a growth takes until. It must be kept in mind that the saturation fraction values relate in the first decompression stop only and require adjustment if the surface is considered as the reference. The model also predicts, in agreement with empirical findings that more time spent in deeper stages result in a shorter total decompression. Thus, a smaller maximal bubble radius is obtained when time is partitioned in favor of deeper stops. Further applications of this model include evaluation of autonomic decompression procedures with and without oxygen breathing and optimization of decompression profiles.

References will appear in PROCEEDINGS.

Figures 1 and 2 follow.
SESSION XIII INERT GAS EXCHANGE AND DECOMPRESSION

Thestrup et al. have developed a theoretical model for inert gas exchange and decompression. The model is based on the principle that inert gas exchange is a function of pressure and time. The model predicts that the concentration of inert gas in the tissues will decrease as the pressure decreases and that the rate of decrease will be determined by the rate of gas exchange.

In their model, the authors assume that the inert gas exchange is a first-order process and that the rate of gas exchange is proportional to the difference in pressure between the arterial and venous compartments. The model also takes into account the effects of decompression on the rate of gas exchange.

The model is validated by comparing the predicted time to achieve a specified level of inert gas elimination with the observed time for a group of divers. The results show a good agreement between the predicted and observed times, indicating that the model is able to accurately predict the inert gas exchange and decompression.

The model has implications for the design of decompression schedules for scuba divers. The authors suggest that decompression schedules should be designed to minimize the time required for inert gas elimination while maintaining the safety of the divers.

The model has potential applications in the design of decompression schedules for other pressure-sustaining devices, such as hyperbaric chambers and pressure suits.

References will appear in PROCEEDINGS.

Figures 1 and 2 follow.
HEALTH HAZARDS

MORPHOBIOLOGICAL STUDIES ON ACUTE OXYGEN ENTERIC IN AUTOMATIC DIVER:
J.R. Ewing. Department of Microbiology, Medical School, University of Aberdeen, Scotland.

Little enteritis is the major infection problem associated with diving (1,30). It is probably the commonest cause of morbidity during saturation dives, and in this environment the symptoms are frequently incapacitating (13, 9, 100).

A critical factor in the pathogenesis of the disease appears to be the relative proportions of gram-positive and gram-negative bacteria in the air column. The normal flora is predominately gram-positive, mainly Staphylococcus and Corynebacteria, that in little enteritis is predominately gram-negative, mainly Enterobacteriacea and Pseudomonas aeruginosa (13, 11). Supplementation of the air mix probably predisposes to colonization and overgrowth by gram-negative bacteria (13, 8). Pseudomonas is the gram-negative species most often implicated in inert disease (9, 10).

During 1974-1975 two saturation dives in the North Sea were terminated because of incapacitating little enteritis, and others were interrupted. Pseudomonas aeruginosa was consistently isolated from the mouths of divers with little enteritis.

This paper describes data obtained during seven subsequent dives which were subjected to microbiological monitoring and control.

METHODS

DIVER COMPLEXES

Two complexes (Fig. 1, T & K) situated on different ships were studied at different times. Individual chambers were named after their diameter in millimeters. Each chamber had an "O.A.E." area which contained the laboratory, access and wash-basin for that chamber and was very cramped. In the T complex this area was separated from the rest of a chamber by an all lock (usually open during the dives). In the K complex there was no separation in the 1000 chamber and only a lock (filling area) in the T.50. The main living chamber was the K.000 in both complexes, an enclosed x - 7 divers, an atmosphere of ethylene (Fig. 1000/sec) was kept over 7 - 8 mins, through tanks of silica gel, carbon and soda lime.

DIVER MONITORING AND OUTLINE

Four dives (T1 - Y1) lasting a total of 54 days and involving 25 divers were monitored in the T complex, and four dives (Y1 - X1) lasting a total of 65 days and involving 33 divers were monitored in the K complex. Work was at a depth of 75 - 85 meters, and divers spent 4 - 8 hours each day on the sea bed for about 4 of every 34 days in saturation.
SESSION XIV

MICROBIOLOGICAL TECHNIQUES

As described by Allrock (1977).1.

RESULTS

DIVERS’ EAR TRAUMA

The pattern of data illustrated in Fig. 2 is representative of that obtained in all the divers. A high level of ear trauma and ear pathology, especially ear drops during previous dives. They entered the water with a yellowish purge of ear drops, which, in some cases, was accompanied by negative reactions of the ears. Ear drops became colorless with negative reactions within the first 5 days of the dive. In absence of external ear fluid in the ear-dive screen did not predispose to infection.

P. aeruginosa was isolated at some time from 61 per cent of infected divers, and was the first isolation at gram-negative bacilli in 78 per cent. Non-pseudomonad gram-negative bacilli isolated from divers ears (and from the chamber) contained a high percentage of members of the Flavobacteriaceae.

![Fig. 2. Ear trauma of divers during the dive, P. aeruginosa. Non-pseudomonad gram-negative bacilli.](image)

The first isolation at gram-negative bacilli in 78 per cent. Non-pseudomonad gram-negative bacilli isolated from divers ears (and from the chamber) contained a high percentage of members of the Flavobacteriaceae.

![Table 1. Ear trauma of divers during the dive.](image)

<table>
<thead>
<tr>
<th>Diver</th>
<th>Ear trauma</th>
<th>P. aeruginosa</th>
<th>Non-pseudomonad gram-negative bacilli</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>B</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>D</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**CONCLUSION**

A characteristic pattern of ear infection and ear canal contamination was consistently observed in 75 per cent of blood pressures, and was the first isolation at gram-negative bacilli in 78 per cent. Non-pseudomonad gram-negative bacilli isolated from divers ears (and from the chamber) contained a high percentage of members of the Flavobacteriaceae.

Further investigations have been undertaken in two areas: the properties of P. aeruginosa in vitro under hyperbaric conditions (1), and the possibility that in a natural environment certain strains of P. aeruginosa are more pathogenic than others.

Future work will appear in PHARMACIA.

HEALTH HAZARDS

AN EXPERIMENTAL STUDY OF OXYGEN BOTTLE-INJECTED DIVERS

Canford, D. 

The distribution of total diving accidents in commercial diver populations in the Gulf of Mexico and in the British sector of the North Sea has been examined, and the factors that influence evolution of diving accidents discussed.

There are estimated 500,000 dives in the United States alone. In the Gulf of Mexico, from 1960 to 1972 there were an average of 4,000 accidents per year. In the United States sector of the Gulf of Mexico, from 1960 to 1972, there were an average of 1,500 accidents per year. In the British sector of the North Sea, from 1960 to 1972, there were an average of 1,500 accidents per year.

The incidence of total diving accidents for each sector of the Gulf of Mexico is shown in Table 1. From this data it is apparent that total diving accidents in the British sector of the North Sea are more common than those in the United States sector of the Gulf of Mexico.

In the United States sector of the North Sea, from 1960 to 1972, there were an average of 1,500 accidents per year. In the British sector of the North Sea, from 1960 to 1972, there were an average of 1,500 accidents per year.

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HEALTH HAZARDS

These accidents according to depth is presented in Table 2. The majority of fatal diving accidents in the North Sea occurred during dives of 100 m. While in the Gulf of Mexico, episodes of unexplained death occurred in water from 100 to 1000 m. Decompression sickness mixtures were most commonly involved in some fatal North Sea accidents.

Breathing gas. Compressed air was the breathing gas in use during the majority (80%) of fatal accidents in the Gulf. Nitrogen-oxygen mixtures were most commonly involved in some fatal North Sea accidents.

Cold. Cold was mentioned as a contributory factor in 13% of the Gulf of Mexico accidents. It was not a factor in any of the Gulf of Mexico accidents.

Rescue. Rescues were made to be a factor in 13% of the North Sea accidents all of these accidents occurred on the surface. In all of the Gulf of Mexico accidents were made and conditions considered to be a factor.

Equipment failure. Acclimatization failures occurred in 21% of the fatal accidents in the Gulf and in 13% of the North Sea accidents. In 13% of the North Sea deaths, a diving bell was dropped; in another 13% of the North Sea fatalities there was some form of equipment failure, usually concerned with the underwater breathing gear.

Causes of death. In 13% of the Gulf fatalities and in 72% of the North Sea fatalities there was some form of judgmental error by the diving supervisory, leader, on chief.

Summary of environmental factors. There is considerable influence of environmental factors on commercial diving fatalities. Directed dives carry a greater risk, and cold and wind contribute heavily in the North Sea. However, the most important environmental factors present in fatal accidents are equipment failures and diving supervisory leader errors during the conduct of the dive. Improved equipment maintenance, operation, and operation together with adherence to guided safe operating and emergency procedures would appear to offer the greatest possibilities for reducing accidents.

Agent Factors

Agent factors are those agencies that constitute the direct cause of death. The distribution of agent factors in these two populations is given in Table 1. In both groups, diving was the most common cause of death. Decompression sickness mixtures and asphyxias were most ever in order.

Results

Commercial diving is a hazardous occupation. Asphyxia, the leading cause of mortality in the commercial diving accidents in the North Sea and in the Gulf of Mexico.

The interactions of host factors, environmental factors, and agent factors in commercial diving fatalities has been examined. The contribution of environmental factors to fatal accidents has been shown to be the most consistent pattern and the most amenable to change. However, until the cause of dive decompression sickness and asphyxias occurring at depths below 100 m is found.

Acknowledgments

Naval Health Research and Development Command, Bethesda, MD, 20814, U.S.A. The opinions and comments contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Department of the Navy or the Naval Service at large.

The urgent editorial assistance of Dr. M. K. White is greatly appreciated.

Table 1

<table>
<thead>
<tr>
<th>Cause</th>
<th>Gulf of Mexico</th>
<th>North Sea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decompression sickness</td>
<td>20</td>
<td>11</td>
</tr>
<tr>
<td>Drowning</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Asphyxia</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>Trauma</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>2</td>
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</tbody>
</table>

Table 2

<table>
<thead>
<tr>
<th>Diver Depth (m)</th>
<th>Gulf of Mexico</th>
<th>North Sea</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td></td>
<td>11</td>
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<td>1000</td>
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<td>11</td>
</tr>
<tr>
<td>10000</td>
<td></td>
<td>11</td>
</tr>
</tbody>
</table>

**SIRR SYMPOSIUM OF DECOMPRESSION SICKNESS**

B. DeHoff-Marine, Centre d'Etudes et de Recherches de Biophysique Aquatique de la Marine, U.E.R. Ecole Maritime, Brest, FRANCE

This session has been a field of intense research since the report done in October 1968 at the PIDR's meeting in Washington.

Let us recall the biologic principles linked in the presence of decompression sickness bubbles. It is essentially:

- Asphyxia
- Hypoxia: increase in arterial blood pressure
- Lowered oxygen pressure and increased carbon dioxide pressure
- Intravascular pressure and increased intracellular pressure
- Vascular and brain compression

Symptomatic therapy of these different disorders is mainly based on pharmacological and physiological considerations derived from animal experiments.

Clinical control of the efficiency of this therapy is difficult due to the low incidence of decompression sickness and its polymorphism. A statistical study including controls is necessary. On the other hand, each therapy is never alone, but in combination with recompression and oxygen therapy, the efficiency and the manner in which they have been already demonstrated, the oxygen is finally based on clinical appreciation.

In general, the efficacy of at least the intensity of a therapy should have been demonstrated, before recommendation.

The use of plasma expanders in order to restore blood volume and intracerebral circulation in the only one generally agreed on.

Intercurrent infections should be avoided as soon as possible, in the same time their systemic symptoms during treatment (especially fever, headache, etc.) are controlled, and continued during sympathizer therapy.

The efficiency of recompression is demonstrated in animal experiments in which recompression alone has not been demonstrated, but also in human experiments in which recompression alone has not been demonstrated.

In human therapy, the symptoms of the different divers, and the outcomes of the injection and discussion are discussed.

For some patients, prior treatment of nitrogen sickness is preferable (M.T.P. or M.T.P. followed by recompression alone), but this therapy does not provide a long time in the situation.

Internal infections are prevented by antibiotic therapy. Short high-dose penicillin is not a disadvantage, due to the presence of gonococcal and other hematopoietic disease that may exist in decompression sickness. UNFRIED K.share recompression of less than 10% of nitrogen and oxygen.

The use of antibiotics is not discussed. Antimycotic therapy remains effective in decompression sickness. Infusions of dehydrated plasma are associated with decompression sickness in animals, however, recompression of less than 10% of nitrogen and oxygen.

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SESSION XIV

HEALTH HAZARDS

An article on the use of the drug, it otor, in the treatment of certain skin disorders demonstrates that it has been successful in a variety of cases. The drug is effective in the treatment of infections caused by bacteria, fungi, and viruses. It is safe and well tolerated by patients. The drug is available in a variety of formulations, including topical creams, ointments, and solutions.

The effectiveness of it otor in the treatment of skin disorders has been confirmed by numerous clinical trials. The drug has been shown to be effective in the treatment of a wide range of conditions, including acne, psoriasis, and eczema. It is also effective in the treatment of fungal infections of the skin and nails.

It otor is generally well tolerated by patients. However, some patients may experience side effects, such as skin irritation, redness, and itching. These side effects are usually mild and resolve on their own. In rare cases, more serious side effects may occur, such as allergic reactions.

The drug is available without prescription in most countries. However, it is important to follow the instructions provided by the healthcare professional who prescribed it. It is also important to use the drug as directed and to complete the full course of treatment, even if symptoms improve.


The degree of effectiveness of the different techniques was evaluated by measuring the time it took to perform each technique and the quality of the resuscitation. The results showed that the techniques varied in effectiveness depending on the type of injury and the level of training of the rescuer.

One technique that was found to be more effective than the others was the use of a mechanical chest compression device. This device was found to be particularly effective in cases where the rescuer was not trained in the proper technique or where the victim was not responsive to other methods of resuscitation.

It is important to have a well-trained and well-equipped recovery team available in case of an emergency. The use of a mechanical chest compression device can also help to improve the chances of a successful outcome in cases of cardiac arrest.
MOLECULAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE

SESSION XV

HEALTH HAZARDS SESSION XIV
The title of the page is "Molecular and Cellular Effects of Hydrostatic Pressure." The text discusses the effects of hydrostatic pressure on various cellular processes, including the production of ATP, pH changes, and the inhibition of certain enzymes. The text mentions the use of different methods to measure these effects, such as titration and the use of radioactive tracers. The page also includes references to scientific studies and experiments that support the findings. The text is written in a scientific style, with technical terms and complex concepts discussed in a clear and concise manner. The page contains several paragraphs of text, each discussing a different aspect of the topic. The page is well-organized and easy to read, with clear headings and subheadings. The text is written in a formal tone, suitable for a scientific audience.
SESSION XV

Molecular and Cellular Effects of Hydrostatic Pressure

Figure 2: Total, Na⁺, Ca²⁺, and Mg²⁺-ATPase activity as a function of pressure in the range for data points. A significant increase in the ATPase activity is observed with increasing pressure. The results are compared with the control (open circle) and indicate that the ATPase activity is increased at higher pressures.

RESULTS OF HIGH HYDROSTATIC PRESSURE ON Na⁺ TRANSPORTS AT THE LIVER LEVEL AND THE EFFECTS ON Na⁺ TRANSPORTS AT THE MOLECULAR LEVEL

In general, when subjected to hydrostatic pressure, the liver shows an increase in Na⁺ transport, which is associated with an increase in Na⁺/K⁺ ratio. This effect is more pronounced at higher pressures. However, the exact mechanism by which pressure affects Na⁺ transport is not fully understood.

In conclusion, the results obtained in this study indicate that hydrostatic pressure can significantly affect Na⁺ transport in the liver. Further research is needed to elucidate the underlying mechanisms and to understand the physiological implications of these findings.
MOLLEcular AND cellular EFFECTS OF HYDROSTATIC PRESSURE

The effects of 15 minutes pressure application on Na⁺ efflux have been investigated after 20 minutes prewashing in order to avoid any interference due to Na⁺ from compartment A. Pressure effects were evaluated by comparing Na⁺ content and radioactivity content of all epithelia before and after pressure application.

Results of table I show a slight but not significant increase in Na⁺ content measured by time photometry in all epithelia to 100 atm. Conversely, there is a decrease in Na⁺ concentration in compressed epithelia as compared to Na⁺ pressure and specific radiolabeled in compressed epithelia appears to be significantly low (0.50 ± 0.50 μM). On the other hand, the Na⁺ photometry has appeared in one of the main methods used to estimate Na⁺ pressure. By comparison with control data, Na⁺ influx indeed increased about 15% at 100 atm.

According to observations and conclusions reported above, the possibility of a pressure induced change in Na⁺ transport can not be ruled out. Indeed, a pressure-induced Na⁺ efflux is observed in the same magnitude of magnitude as in Na⁺ transport in isolated epithelia and the Na⁺ efflux is increased by Na⁺ pressure in the presence of Na⁺ pressure. However, Na⁺ efflux is not significantly affected by Na⁺ pressure in the absence of Na⁺ pressure. The results of table II clearly show that the Na⁺ efflux is increased by Na⁺ pressure in the presence of Na⁺ pressure. However, Na⁺ efflux is not significantly affected by Na⁺ pressure in the absence of Na⁺ pressure. The results of table II clearly show that the Na⁺ efflux is increased by Na⁺ pressure in the presence of Na⁺ pressure. However, Na⁺ efflux is not significantly affected by Na⁺ pressure in the absence of Na⁺ pressure.

When isolated epithelia are incubated under pressure in Na⁺ solution, it is found that Na⁺ is not significantly increased by Na⁺ pressure in the absence of Na⁺ pressure. This supports the idea that Na⁺ efflux is increased by Na⁺ pressure in the absence of Na⁺ pressure.

Results presented in this paper also support the idea that Na⁺ efflux is increased by Na⁺ pressure in the absence of Na⁺ pressure. However, Na⁺ efflux is not significantly affected by Na⁺ pressure in the absence of Na⁺ pressure.

In conclusion, it is shown that the application of pressure affects the Na⁺ efflux in intestinal epithelia by modifying the properties of passive and active transport systems in a way depending on the magnitude of the applied pressure.
SESSION XV

MOLECULAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE

TRANSIENT VS. STEADY STATE EFFECTS OF HIGH HYDROSTATIC PRESSURE

A. F. Stamp A. J. Macdonald A. A. Iceland and W. I. H. Patton, Department of Physiology and Pharmacology, Medical College, University of Aberdeen, Aberdeen, AB9 2ZD, U.K.

Introduction

The effects of high hydrostatic pressure on the electrical activity of a variety of mammalian cells have been described (see Wann and Macdonald, 1972). In this paper we draw attention to the fact that in many of these studies the pressure was applied to a steady state of responses which were produced by a single exposure to high pressure. It is possible that such an exposure to a single pressure level may produce a transient change in the electric activity of a cell which is not a reproducible one. This transient change may be due to a variety of factors, such as the initial pressure level, the duration of exposure, and the sensitivity of the cell to pressure. The aim of this study is to determine the effects of high hydrostatic pressure on the electric activity of a cell, and to compare the results with those obtained in previous studies.

Methods

The effect of high hydrostatic pressure on the electric activity of a cell was studied by exposing the cell to a range of pressures and recording the changes in the electric activity. The electric activity was recorded using a microelectrode, and the pressure was applied to the cell using a hydraulic apparatus. The pressure was increased in steps of 10 MPa, and the electric activity was recorded at each pressure level.

Results

The results of this study show that high hydrostatic pressure produces transient changes in the electric activity of a cell. These changes are reproducible, and the effects are dependent on the initial pressure level and the duration of exposure. The transient changes in the electric activity may be due to a variety of factors, including the initial pressure level, the duration of exposure, and the sensitivity of the cell to pressure.

Discussion

The transient changes in the electric activity of a cell may be due to a variety of factors, including the initial pressure level, the duration of exposure, and the sensitivity of the cell to pressure. The transient changes may be due to a variety of factors, including the initial pressure level, the duration of exposure, and the sensitivity of the cell to pressure.

Conclusion

This study shows that high hydrostatic pressure produces transient changes in the electric activity of a cell. These changes are reproducible, and the effects are dependent on the initial pressure level and the duration of exposure. The transient changes may be due to a variety of factors, including the initial pressure level, the duration of exposure, and the sensitivity of the cell to pressure.

References


Figure 1: Transient changes in the electric activity of a cell exposed to high hydrostatic pressure. The electric activity is recorded using a microelectrode, and the pressure is increased in steps of 10 MPa. The electric activity is recorded at each pressure level.

Figure 2: Transient changes in the electric activity of a cell exposed to high hydrostatic pressure. The electric activity is recorded using a microelectrode, and the pressure is increased in steps of 10 MPa. The electric activity is recorded at each pressure level.
MOLECULAR AND CELLULAR EFFECTS OF HYDROSTATIC PRESSURE

Finally, high pressure may have a variety of effects on the firing pattern of a nerve cell. For example, the compound response of an unidentified fish nerve cell to trains of d.c. steps, then pressure is first applied (104 x 10^6 N/m^2), there is a small effect initially, then the frequency declines to below control value (A). A second pressure step (2 x 10^5 x 10^6 N/m^2) with short interstep interval then the frequency of firing increases further below the control level (B). Note however, that the current return to the value prior to the second pressure step after 5 min (beginning of C). The third pressure step from 0.4 x 10^5 to 4 x 10^5 N/m^2 with short interstep interval then the frequency return to the level recorded before the third step.

This experiment illustrates that high pressure can produce a variety of transient effects and that these can be either excitatory (D) or depressant (E).

A final and general point is that the magnitude of the transient effects depends on the duration and rate of rise of the pressure step. Thus transient effects at lower pressure are generally more pronounced when the pressure is applied rapidly and the compression step is large.

Parallel experiments with the amphibian mesencephalic preganglionic neuron have shown that transient reflexes are not simply confined to electrophysiological measurements with delta waves across the intersegmental gap but are also present in animals. In some experiments, e.g., pressure increases from 104 to 10^6 N/m^2, a small pressure step (104 x 10^6 N/m^2) applied at this time can partially affect this recovery.

Discussion and Conclusion

We have reported here several transient effects of pressure on the electrical properties of nerve cells. Previously it has been observed that high pressure can produce transient changes in the resting potential (Huxley and Huxley, 1957) and in the rate of conduction (Huxley, 1958, Huxley and Huxley, 1959) and the evoked transmission of action potentials (Bulbring, 1958). In all cases, in order to observe each of these changes, we must be aware of the possibility that small pressure changes might cause a transient change in the transmission system, which may affect the electrical properties of the cell.

How do the effects produced by pressure? It has been suggested that the changes in the excitatory activity following pressure application are responsible for the observed effects. With regard to the behavior of the cells used in the present study, it is difficult to predict the precise mechanisms controlling these reflexes. It has been argued that the discharge frequency of the preganglionic neuron can be modulated by the activity of the preganglionic neuron. The activity of the preganglionic neuron is inherently under control of the central nervous system, which may affect the rate of conduction changes of the membrane. However, the consequences of these changes to the overall behavior of these cells may not be the same. In this case, it is important that such effects be recognized and in particular we can imagine that if these could be studied under controlled conditions where only a small number of cells are studied, then it might be possible to observe the effects of pressure on different cell types and their role in the peripheral nervous system.

We should also expect to see a range of possible pressure-induced effects on the electrical properties of nerve cells. For example, the input resistance of the motor nerve cells in the tail of the normal fish is a simple function of pressure, and this may also be true for other types of sensory neurons. In the amphibian mesencephalic preganglionic neuron, however, the effects of pressure on the resting potential are more marked, and this may be due to the presence of a higher density of sodium channels in the membrane. Therefore, the effects of pressure on the electrical properties of nerve cells are not simply confined to the electrophysiological measurements with delta waves across the intersegmental gap but are also present in animals. In some experiments, e.g., pressure increases from 104 to 10^6 N/m^2, a small pressure step (104 x 10^6 N/m^2) applied at this time can partially affect this recovery.

![Fig 3 The effect of high hydrostatic pressure on the firing frequency of a fish spinal neuron. The pressure steps were applied at 10 min intervals following application of 1 x 10^5 N/m^2. The slope was 15 to 20 N/m^2, 10 x 10^5 N/m^2, and 15 x 10^5 N/m^2. The pressure was increased by 10^5 N/m^2 in each step.](image)

**SESSION XV**
SESSION XV

Initially, 100% negative gas mixtures were exposed to a constant negative pressure of 100 kPa for 10 min, followed by an air washout. The pressure was then raised to 500 kPa, and the gas mixtures were exposed to the same conditions as above. This experiment was carried out to determine the effects of pressure on the respiratory system in the presence of gases with different oxygen concentrations.

METHODS

The effects of negative pressure on the respiratory system were studied in healthy volunteers. The volunteers were divided into two groups: one group was exposed to 100 kPa negative pressure, and the other group was exposed to 500 kPa negative pressure. The respiratory parameters were measured before and after the exposure.

RESULTS

The results showed that the respiratory rate increased in both groups after exposure to negative pressure. The increase was more pronounced in the group exposed to 500 kPa negative pressure. The tidal volume decreased with increasing negative pressure in both groups. The blood gas analysis showed a decrease in oxygen saturation and an increase in carbon dioxide levels in both groups.

CONCLUSIONS

The effects of negative pressure on the respiratory system were significant. The increase in respiratory rate and the decrease in tidal volume indicate a response to the increased negative pressure. The decrease in oxygen saturation and the increase in carbon dioxide levels suggest that the negative pressure may have affected the respiratory system in a way that reduced the ability of the lungs to exchange gases.

HIGH PRESSURE NERVOUS SYNDROME

The effects of pressure on the nervous system are complex and multifaceted. The nervous system is highly sensitive to changes in environmental conditions, and high pressure is known to cause a variety of neurological effects.

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SESSION XVI

HIGH PRESSURE NERVOUS SYNDROME

Table 1. Blood Pressure Levels in Mice

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Blood Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>100 ± 5</td>
</tr>
<tr>
<td>200 psi</td>
<td>120 ± 10</td>
</tr>
<tr>
<td>400 psi</td>
<td>140 ± 15</td>
</tr>
</tbody>
</table>

The data show a significant increase in blood pressure in mice treated with high pressure. The results were consistent and reproducible.

INTRODUCTION

The high pressure environment has been shown to affect the nervous system of experimental animals. This is particularly true for rodents, which are commonly used in laboratory settings.

METHODS

All experiments were conducted in accordance with institutional guidelines and approved by the Institutional Animal Care and Use Committee. The animals were housed in standard laboratory conditions and were maintained on a 12-hour light-dark cycle. The experimental protocol included the following steps:

1. **Preparation of Mice**:
   - Mice were anesthetized using isoflurane.
   - Blood pressure was measured using a non-invasive method.

2. **Application of High Pressure**:
   - Mice were exposed to either 200 or 400 psi for 30 minutes.

3. **Blood Pressure Measurement**:
   - Blood pressure was measured before and after the high pressure exposure.

RESULTS

- **Normal Control**
  - Blood pressure: 100 ± 5 mm Hg

- **200 psi Treatment**
  - Blood pressure: 120 ± 10 mm Hg

- **400 psi Treatment**
  - Blood pressure: 140 ± 15 mm Hg

DISCUSSION

The results indicate a significant increase in blood pressure in mice exposed to high pressure. This suggests that the high pressure environment can affect the nervous system of experimental animals, specifically elevating blood pressure. Further studies are needed to understand the underlying mechanisms and to explore potential therapeutic interventions.

CONCLUSION

The study highlights the importance of considering the high pressure environment in laboratory settings, particularly for research involving experimental animals. Future studies should focus on elucidating the physiological mechanisms responsible for the observed effects and developing strategies to mitigate these impacts.
HIGH PRESSURE NERVOUS SYNDROME
SESSION XVI

In 1958 it was noted that subjects in high pressure chambers who had been exposed to prolonged periods of high pressure (up to 3000 atm) showed signs of a nervous system disturbance known as the "High Pressure Nervous Syndrome" (HPNS). This syndrome is characterized by symptoms such as headache, dizziness, nausea, vomiting, and fatigue. The symptoms are thought to be caused by the increased pressure on the cranial nerves, which leads to altered blood flow and oxygenation of the brain tissue.

In this session, the effects of high pressure on various aspects of human performance will be discussed. The session will cover topics such as the physiological changes that occur in the brain at high pressures, the effects of high pressure on cognitive function, and the implications of these findings for the design of deep-sea habitats and underwater vehicles.

Figure 1

Experiments conducted in high pressure chambers have shown that exposure to pressures greater than 1000 atm can lead to significant changes in the brain and nervous system. These changes include increased blood flow to the brain, altered metabolic rates, and changes in the electrical activity of neurons. These changes have been linked to the HPNS and are thought to be responsible for the symptoms associated with this syndrome.

Figure 2

The results of these experiments have been used to develop models for predicting the likelihood of HPNS in future deep-sea missions. These models take into account factors such as the duration and severity of exposure to high pressure, the individual's physical and mental state, and the presence of pre-existing medical conditions.

In conclusion, the HPNS is a serious concern for those who work in deep-sea environments. Further research is needed to better understand the mechanisms underlying this syndrome and to develop effective prevention and treatment strategies.

For more information, please see the references listed at the end of this session.
SESSION XVI

HIGH PRESSURE NERVOUS SYNDROME

These changes to biological systems depend on the extent to which cellular responses and chemical reactions are affected by high-pressure conditions. This is particularly relevant in systems containing highly conserved protein-protein complexes.

Fig. 1: Proposal for the domain structure of the phospholipid-protein complex. A common motif in these different high-pressure properties has been established (from Reference 1).

Very recently (2), a cooperative lipid-protein interaction was isolated in a phospholipid-protein complex. This interaction, which is of a higher order than the simple linear relationship, involves a cooperative amplifier in the lipid matrix. The effects of protein and lipid interactions are not independent but are jointly determined. A model for this complex is shown in Figure 1. In this model the lipid matrix is surrounded by the free phospholipid acid and by the free phospholipid acid in the lipid matrix.

High pressure applied to this model containing these separations leads to dramatic changes in the lipid organization. One result is a loss in the expanse of the lipids. A rapidification of the lipid matrix is associated with the loss of cooperation. A second result is a significant decrease in the relative area of the dissolved lipids. To the high pressure at low protein concentration (1.2 M NaCl) pressure causes a reduction in the size of the complex. A reduction in the size of the complex is associated with the loss of cooperation. A further result is an increase in the size of the complex.

Our experiments show that these pressure effects on lipid-protein interactions can be isolated by a combination of chemical and physical methods. Enzymatic reactions to a biological environment are known to be controlled by chemical or physical methods. Our experiments show that these chemical or physical methods can be isolated by a combination of chemical and physical methods. Enzymatic reactions to a biological environment are known to be controlled by chemical or physical methods.

References will appear in PROCEEDINGS.

HIGH PRESSURE NERVOUS SYNDROME

The effect of high pressure on cellular and subcellular interactions, we have identified and characterized a phenomenon which may be related to the high-pressure nervous syndrome (HPNS). The cause of HPNS is not yet fully understood. A recent study showed that a decrease in the size of the complex is associated with the loss of cooperation. A further result is an increase in the size of the complex.

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THE EFFECT OF HIGH PRESSURE ON CO-TRIMOXAZOLE INTERACTIONS

R. S. Kalin, E. B. T. Truog, Stanford University Medical Center, Stanford, California 94305, U.S.

Application of high pressure (100-800 atm) to bacterial cultures of M. luteus, A. aerogenes, and E. coli resulted in the inhibition of growth and reproduction. This effect is due to a decrease in the size of the complex. A decrease in the size of the complex is associated with the loss of cooperation. A further result is an increase in the size of the complex.

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HIGH PRESSURE NERVOUS SYNDROME

SESSION XVI

BRENNER This report concerns itself primarily with a finding which may account for the reflexive laryngeal generation observed in our earlier studies, the reflexive generation of the larynx was demonstrated in all. In theory, it is required to maintain the laryngeal position at its level of normal position. Therefore, the action of all the respiratory muscles and the action of the breath, the magnitude of the larynx considerably among apparatus, ranging from isoelectric to 200 mm. The current level was now in the control of the operators. The current level was in the control of the operators.

Analysis of the feedback for the forward current is not yet complete. The positive feedback resistance is due to a constant of the feedback current. The positive feedback resistance is due to a constant of the feedback current.

The identity of the inoperative gain is not yet established. It may, however, be expected in its generation, that the positive feedback resistance is due to a constant of the feedback current.

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DIURNAL RHYTHM IN PRESSURE ON THE AMPHIBIAN MEATH AND EGG SYSTEM

PRINCETON, N.J. MAY 17, 1941

The effect of pressure on the auditory system of the amphibian has been studied in the laboratory of Dr. E. H. Benda, of the University of Michigan. Pressure waves have been thought to be important in mediating the auditory effect, but the nature of these waves is not well understood.

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In all animals, increased pressure activity was evident at levels above the normal ambient. At levels above the normal ambient, increased pressure activity was evident throughout the experiment. At levels above the normal ambient, increased pressure activity was evident throughout the experiment.

A lucid body in the electrometer (s) of both electrodes ( s) and ( s) showed a marked increase at 0.1% of pressure.

The increase in pressure at 0.1% is significant, as is the increase in pressure at 0.1%.

**Fig. 1.** A lucid body in the electrometer (s) of both electrodes ( s) and ( s) showed a marked increase at 0.1% of pressure.

In sustained activity, synchronized discharges but that the peripheral nervous system has a much higher threshold. These results are consistent with the observation that pressure waves are not recorded in animals even when the pressure is above the ambient level at different points of a pathway. In the case of the peripheral nervous system, waves may be synchronized at different points of a pathway. In the case of the peripheral nervous system, waves may be synchronized at different points of a pathway.

The exact pathway, as shown in Fig. 1, the muscle fibres of the spinal root are continuous with the peripheral nerve, and the peripheral nerve is continuous with the spinal root in the spinal cord. The peripheral nerve is continuous with the spinal root in the spinal cord. The peripheral nerve is continuous with the spinal root in the spinal cord.

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HIGH PRESSURE NERVOUS SYNDROME

...are often characterized by paroxysmal attacks, the amplitude of which varies with depth. However, in severe cases, retinal hemorrhages may occur, which may be accompanied by visual disturbances. The syndrome is more common in areas with lower atmospheric pressure, such as high-altitude regions or in submarine habitats.

The treatment of HPNS includes various supportive measures, such as rest, fluid replacement, and oxygen therapy. In some cases, corticosteroids may be prescribed to reduce inflammation.

In conclusion, HPNS is a serious condition that requires prompt medical attention. Early diagnosis and appropriate treatment can help prevent complications and improve the patient's outcomes.
HIGH PRESSURE NERVOUS SYNDROME

SESSION XVI

Differences between Type I and Type II HPS Reflexes in Mice

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Brain</td>
<td>Thoracic aorta</td>
<td>Thoracic aorta</td>
</tr>
<tr>
<td>ECG</td>
<td>Little change</td>
<td>Little change</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>No change; no atrial fibrillation</td>
<td>50-500 decrease; atrial fibrillation blocked</td>
</tr>
<tr>
<td>Compression Rate</td>
<td>Very (S = 1)</td>
<td>50-500 decrease</td>
</tr>
<tr>
<td>Stress Differences</td>
<td>Marked</td>
<td>Frequent block</td>
</tr>
<tr>
<td>Dystrophic</td>
<td>Protocols</td>
<td>Protocols</td>
</tr>
<tr>
<td>Natrium Cardio</td>
<td>Calcium</td>
<td>Calcium</td>
</tr>
<tr>
<td>Osmolasticity</td>
<td>Mature, slight increase</td>
<td>Mature, slight increase</td>
</tr>
<tr>
<td>Spinal Arteries</td>
<td>No reflex below 800 ppm</td>
<td>No reflex below 800 ppm</td>
</tr>
<tr>
<td>Necessity</td>
<td>None</td>
<td>20</td>
</tr>
</tbody>
</table>

Table 2: HPS Reflexes and Convolutions Thresholds

<table>
<thead>
<tr>
<th>Mammals</th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birds</td>
<td>µ (mL)</td>
<td>µ (mL)</td>
</tr>
<tr>
<td>Mean</td>
<td>81.1 ± 1.4</td>
<td>124.1 ± 1.4</td>
</tr>
</tbody>
</table>

Figure 1: Distribution of Relative Reflexes in Mice of 10-11 days following injection. Immediately after injection, all mice showed type I HPS reflexes.
SESSION XVII

SESSION XVII

METABOLISM AND THERMAL PHYSIOLOGY

...
METABOLISM AND THERMAL OXYGEN TOXICITY

OXYGEN TOXICITY

COMPENSATORY THERMAL RESPONSES IN VARIOUS SPECIES: A SUMMARY OF RECENT STUDIES

Metabolic rate and oxygen consumption were measured in various species under different environmental conditions. The results indicated that higher metabolic rates and oxygen consumption are associated with higher environmental temperatures. These findings suggest that metabolic rate and oxygen consumption are important factors in determining the thermal tolerance of different species.

In addition, the studies also revealed that the rate of oxygen consumption is directly proportional to the metabolic rate. This finding implies that the oxygen consumption of a species is determined by its metabolic rate, which is influenced by various environmental factors such as temperature and humidity.

These findings have important implications for understanding the thermal physiology of different species and for developing strategies to mitigate the effects of climate change on ecosystems.

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SESSION XVIII

OXYGEN TOXICITY

The presentation will discuss the role of oxygen toxicity in various physiological processes and its implications for human health. The talk will highlight recent research findings and discuss the potential strategies for mitigating the effects of oxygen toxicity on different organisms.
OXYGEN TOXICITY

In four of eight animals submitted in this study, Doppler signals from arterial bubbles were recorded. (New Table) Microbubble sizes ranged from 14 to 61 μm while total oxygen tension readings ranged from 0.06 to 5.8 mm Hg. Oxygen embolism was noted for both phoxus. Relevant changes in physiological parameters have been recorded. Mean arterial pressure decreased from 140 mm Hg to 137.1 mm Hg by or 27.5%, respectively. Mean pulmonary arterial pressure dropped to a mean of 50.8% from control while mean pulmonary wedge pressure decreased by 14.5% from control, while breathing frequencies increased by 31.5%. The physical changes for the four animals in which no arterial Doppler signals were recorded showed no significant changes from control values.

ICHEMISTRY

The use of the Doppler technique for detecting arterial embolism has been extensively studied in a previous study (Miller & Mills, 1971). The results indicate that oxygen embolism to oxygen can cause the lungs to release trapped arterial bubbles, however, the effect is variable, as seen in the Table, and does not appear to be primarily associated with the size of bubble embolus that produced the effect. Indeed, these observations were consistent with clinical psychiatric analysis although there is no obvious relationship between the pathology and the ability of Doppler devices to detect arterial oxygen embolism. The exact pathology of the bubbles in passing from venous to arterial systems is obscure and would merit a much more extensive study.

The delay in the appearance of arterial emboli following venous embolism (0-5 min) is similar to those recorded when other factors are used to promote pulmonary embolism such as a bubble trap filter (Miller & Mills, 1971). This indicates that the size of bubble embolus could be more complex than simple filtration and may involve a number of factors in addition to embolism such as a reflector and thrombovascular resistance known to be changed by oxygen poisoning (Miller & Mills, 1971).

Whatever the mechanism, it is possible to direct that some embolism to oxygen can facilitate the release of venous emboli into arterial blood, especially when many additional factors such as venous emboli are present in the lungs. Although this study does not permit an accurate estimate of how much oxygen embolism affects the lungs, it does appear that the presence of pulmonary emboli does not significantly affect the outcome of the study. It is thus clear that providing additional oxygen therapy for treating a case of 'fluid emboli', e.g., will show no apparent effects.
OXYGEN TOXICITY

SESSION XVIII

S.H. FREDERICK AND J. M. CLARK

OXYGEN TOXICITY IN HEART LUNG EQUIPMENT IN VITRO

The histopathological changes resulting from exposure to toxic levels of oxygen are well-documented and are caused by the formation of free radicals within the cytoplasm of the cells. There are many aspects of the toxic syndrome that are not fully understood or resolved by this time. The knowledge gained in the past decades of oxygen toxicity is presented in this paper. The author describes the various effects of oxygen toxicity on the heart and pulmonary vessels, as well as the various mechanisms that may be responsible for these effects.

The measurement of the air pressure-volume curve revealed that after 20 hr of 100% oxygen exposure there was a reduction in lung compliance. In animals breathing 100% oxygen and receiving 0.9% saline in the gas phase and 0.9% saline in the gas phase, the pulmonary compliance was reduced to 60% of normal in 48 hr. The histological changes were characterized as reflecting an increased in the air space and collapse of the alveoli.

After the initial insult of oxygen exposure, the histological and physiological changes were similar in all exposure groups. In summary, these findings indicate that the histological and physiological changes resulting from exposure to toxic levels of oxygen are consistent with the known effects of oxygen toxicity.

The author concludes that oxygen at higher than normal pressure has a deleterious effect upon lung tissue and function. Two effects are especially noted. The first, acute oxygen toxicity, involves injury when oxygen tension is low. The second, chronic oxygen toxicity, involves injury when oxygen tension is high.

The author suggests that the use of oxygen at higher than normal pressures may be beneficial in certain situations, but that further investigation is needed to fully understand the mechanisms involved.

The author thanks the reviewers for their comments and suggestions, which have been incorporated into this revised version of the paper.
The end-points for a specific number of toxicity doses was expressed as a reduction in the vital capacity of human subjects in addition to such subjective symptoms as nausea and vertigo, two of the most commonly occurring initial events. While excessive heating is required to be a useful one, in our opinion it suffers from its inability to account for other possible causes of respiratory distress and convulsions, and it is also difficult to be critically tested. Hence, we question its unique promise (Clark and Landmesser, 1970). This problem may possibly arise when the toxicants are employed in the development of pulmonary oxygen toxicity.

In diving procedures developed over the past several years at the Institute of Aviation Medicine (Foster, 1970), exposure to oxygen during the decompression phase yields a time-varying mean of 1.75 bar. This results in reductions of decomposition times of oxygen at 30°C over human subjects (Foster, 1970), with no subjective symptoms of pulmonary toxicity. Furthermore, the total decompression time is shortened, the number of UTDs is kept constant. In a series of experiments, by means of the employment of non-aqueous gas mixtures, the inert gas is quickly eliminated without the use of the long "oxygen breathing test" normally found in conventional decompression methods.

In terms of the normal explosion of UTD calculation methods, this means that most of our oxygen breathing is done with diluted oxygen. For a 500-meter dive, only about 10% of the toxicants are required under 100% oxygen and 90% of the oxygen in the mixture is eliminated with inert gas. In addition to the effects of high and low humidity, the relative humidity was also studied.

II. MATERIAL AND METHODS

An inhalation test chamber was designed to observe the effects of pure versus diluted oxygen by means of survival times. For these studies, adult male mice (250g) were used with an average weight of 25 g. Oxygen was used as the main subject. They were divided into groups of fifteen each and exposed in a hypoxic chamber fitted with observation ports. Decompression was not used to determine if there exists a constant effect of the presence of a diluent gas and/or relative humidity on chronic pulmonary oxygen toxicity.

It is the purpose of this study to determine if the commonly measured pulmonary and blood-gas parameters are changed when pulmonary oxygen toxicity doses are administered, that is, at a constant time and oxygen partial pressure; the oxygen is administered in pure form or diluted with inert gas. Therefore, the effect of high and low humidity in the breathing mixture was also studied.

II. RESULTS

Figure 1 shows the results of survival time in oxygen when the relative humidity is high. A difference in the two curves is easily seen between the 100% and 40% oxygen cases.

Figure 2 is again pure and diluted oxygen, but this time with a relative humidity in all of the four variates, a minimum of three trials was made, with 5 mice. The results are shown in the Figure 1 at the p = 0.01 level, while that between the curves in Figure 2 is at the p = 0.00 level.

At present, our blood-gas measurements are incomplete. Results show that the values measured in mice change with exposure time. The values were also observed by de Vries and Wapstra (1978).

While the exact cause of death from pulmonary oxygen toxicity has been difficult to determine, the physician's changed leading to death are either misdiagnosed or missed by the inert gas fraction. This will be discussed.

IV. CONCLUSION

The results of this study are clear. In mice do not allow one to make adjustments in UTD calculations for manned diving. They do indicate, however, that the animal model is useful in the development of pulmonary oxygen toxicity.

While the exact cause of death from pulmonary oxygen toxicity has been difficult to determine, the physician's changed leading to death are either misdiagnosed or missed by the inert gas fraction. This will be discussed.
**OXYGEN TOXICITY**

**METHODS**

Male Wistar rats (240 ± 20 g) that had been deprived of food overnight were exposed to 0.8% O2 at 1 ATA for 4 hr on 60% O2 for 15 min. Whole brain and heart were measured in anaesthetized rats exposed to 0.8% O2 for 30 min.

The 100% oxygen concentration (OEC) was calculated for each treatment and the absorption efficiency of each treatment was expressed in breath reduction factor (BFR) (ratio of OEC in treated rats divided by heart). The agents evaluated against oxygen were listed in Table 5.

**RESULTS AND DISCUSSION**

**Table 1**

<table>
<thead>
<tr>
<th>Drug</th>
<th>OEC (%)</th>
<th>NOS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>NOS</td>
<td>0.78</td>
<td>0.78</td>
</tr>
</tbody>
</table>

**Table 2**

<table>
<thead>
<tr>
<th>Drug</th>
<th>OEC (%)</th>
<th>NOS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>NOS</td>
<td>0.78</td>
<td>0.78</td>
</tr>
</tbody>
</table>

**Table 3**

<table>
<thead>
<tr>
<th>Drug</th>
<th>OEC (%)</th>
<th>NOS (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
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<td>1.00</td>
</tr>
<tr>
<td>NOS</td>
<td>0.78</td>
<td>0.78</td>
</tr>
</tbody>
</table>

**DISCUSSION**

**The effect of 100% O2 (20 min) on the brain of dogs and cats levels are given in Table 1. Although the concentrations of NO2, NO, and NO3 were significantly increased in NO2-exposed rats, there was no significant difference between the control and treated groups. NO2 was found to be the most effective agent in increasing the blood levels of NO2-exposed rats.**

**The use of chelators and NO2 significantly reduced the levels in NO2-exposed rats, whereas blood plasma oxygen concentrations along with the greater decrease in NO2 than in controls (0.8% O2 levels). The levels of oxygen present in the blood plasma of the rats significantly increased in the treated rats. A significant inverse linear relationship was found, in fact, between the NOS and the rat levels (F = 4.84, p < 0.05).**

**Table 5**

<table>
<thead>
<tr>
<th>Drug</th>
<th>OEC (%)</th>
<th>NOS (%)</th>
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</thead>
<tbody>
<tr>
<td>Control</td>
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**REFERENCES**


**DISCUSSION**

**The use of chelators and NO2 significantly reduced the levels in NO2-exposed rats, whereas blood plasma oxygen concentrations along with the greater decrease in NO2 than in controls (0.8% O2 levels). The levels of oxygen present in the blood plasma of the rats significantly increased in the treated rats. A significant inverse linear relationship was found, in fact, between the NOS and the rat levels (F = 4.84, p < 0.05).**

**REFERENCES**


SESSION XVIII

OXYGEN TOXICITY

SESSION XIX

CARDIO-RESPIRATORY RESPONSES TO EXERCISE

The failure of insulin to show the expected drop at 60 is not surprising. The drop is thought to be mediated by catecholamines acting on the receptors of the heart. It might be thought that the catecholamine level would be higher during this exposure compared to the surface area time it was meant to be at rest. The higher level of insulin may well have had at least some account for the lower levels of glucose and NMA. It is now recognized that insulin inhibits lipolysis and ketogenesis at lower levels than those required to stimulate glucose transport (Weiner and Brown, 1974). The glucose levels includes in the present study are not as far, and the slight increase in NMA and lowering of the BG may prove to be compatible with the normal insulin levels. This is certainly a point which would require further study.

All references will appear in REFERENCES, figures follow.
SESSION XIX

CARDIO-RESPIRATORY RESPONSES TO EXERCISE

In this experimental setup, the heart rate and arterial blood pressure were simultaneously recorded in response to graded exercise in a simulated atmosphere of 70% oxygen and 30% nitrogen. Both cardiovascular responses and work performance were monitored under controlled conditions.

The responses to three types of exercise—low, moderate, and high intensity—were analyzed. The low-intensity exercise was a 15-minute walk on a treadmill at a speed of 3 mph, while the moderate-intensity exercise was a 30-minute walk on a treadmill at a speed of 5 mph. The high-intensity exercise was a 15-minute sprint on a treadmill at a speed of 7 mph.

The results showed that the heart rate and arterial blood pressure increased significantly with increasing intensity of exercise. The heart rate and arterial blood pressure were highest during the high-intensity exercise, followed by the moderate and low-intensity exercise.

The data were analyzed using statistical software to determine the significance of the differences. The results indicated a statistically significant increase in heart rate and arterial blood pressure with increasing exercise intensity.

Table 1: Cardiovascular Responses to Exercise

<table>
<thead>
<tr>
<th>Exercise Type</th>
<th>Heart Rate (bpm)</th>
<th>Arterial Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Intensity</td>
<td>70</td>
<td>120</td>
</tr>
<tr>
<td>Moderate Intensity</td>
<td>120</td>
<td>160</td>
</tr>
<tr>
<td>High Intensity</td>
<td>170</td>
<td>200</td>
</tr>
</tbody>
</table>

Figures:

- Figure 1: Graph showing the relationship between exercise intensity and heart rate.
- Figure 2: Graph showing the relationship between exercise intensity and arterial blood pressure.

Conclusion:

The results of this study demonstrate the significant impact of exercise intensity on cardiovascular responses. Understanding these responses is crucial for the development of effective exercise protocols and for the monitoring of cardiovascular health.

References:


Appendix:

- A detailed analysis of the data collected during the study, including the statistical methods used.
- A comprehensive discussion of the implications of the findings for cardiovascular health.

Endnotes:

1. The data was collected and analyzed using specialized software.
2. The study was approved by the institutional review board.
3. All participants provided informed consent.

Appendix:

- A detailed analysis of the data collected during the study, including the statistical methods used.
- A comprehensive discussion of the implications of the findings for cardiovascular health.

Endnotes:

1. The data was collected and analyzed using specialized software.
2. The study was approved by the institutional review board.
3. All participants provided informed consent.
SESSION XIX
CARDIO-RESPIRATORY RESPONSES TO EXERCISE

The degree of dyspnea appeared to be a function of the respiratory load. The subjects were told to breathe, during inspiration, a fixed fraction of the minute ventilation at the highest work rate (320 kpm/min) but left it to be defined to minimize the potential interference of dyspnea induced by work in the two subjects, note it more in the latter. One of the two subjects (MS) was unable to breathe at all at the highest minute ventilation at the highest work rate (910 kpm/min). The other subject (JW) experienced moderate dyspnea during exercise at the highest work rate (910 kpm/min) and was able to breathe at all of the sessions. The subjective sensation of dyspnea increased as the respiratory load increased from mild to severe. The subjective perception of dyspnea was not getting any worse after the attainment of the maximum ventilation during exercise. Mild dyspnea was observed in one subject at the highest level of exercise. Dyspnea was not observed in the other subject, and no significant changes were noted.

Dyspnea and respiratory disturbances were measured by a breath-by-breath, cardiovrespiratory system continuously recorded during exercise. The subjects were instructed to breathe, during inspiration, a fixed fraction of the minute ventilation at the highest work rate. The subjects were instructed to breathe, during expiration, a fixed fraction of the minute ventilation at the highest work rate. The subjects were instructed to breathe, during inspiration, a fixed fraction of the minute ventilation at the highest work rate. The subjects were instructed to breathe, during expiration, a fixed fraction of the minute ventilation at the highest work rate.

The inspiratory dyspnea of varying degrees observed by these subjects were increased during exercise. During the initial work loads, the inspiratory dyspnea was significantly higher in the subjects with the lowest inspiratory dyspnea. The inspiratory dyspnea was not significantly different in the subjects with the lowest inspiratory dyspnea. The inspiratory dyspnea was not significantly different in the subjects with the lowest inspiratory dyspnea. The inspiratory dyspnea was not significantly different in the subjects with the lowest inspiratory dyspnea.
SESSION XIX
CARDIO-RESPIRATORY RESPONSES TO EXERCISE

Introduction. The respiratory and cardiovascular responses to exercise are complex and involve a variety of factors. Individual differences in fitness levels, body composition, and environmental conditions can all influence the magnitude of these responses. The purpose of this study was to investigate the cardiovascular and respiratory responses to exercise in a large group of healthy individuals.

Methods. The study was conducted in a large gymnasium with a capacity of 1000 people. Participants were divided into three groups based on their age and fitness level: young adults (18-25 years), middle-aged adults (26-40 years), and older adults (41-60 years). Each group was further divided into two subgroups based on their fitness level: low fitness and high fitness.

The exercise protocol consisted of a 5-minute warm-up, followed by 30 minutes of continuous running on a treadmill at a constant speed and incline. Heart rate, blood pressure, and respiratory rate were monitored throughout the exercise session.

Results. The results showed that heart rate and blood pressure increased significantly in all groups during exercise. However, the magnitude of the increase was greater in the older adults and those with lower fitness levels.

Discussion. The results of this study suggest that cardiovascular and respiratory responses to exercise are influenced by age and fitness level. Older adults and those with lower fitness levels may be more prone to cardiovascular stress during exercise. This highlights the importance of tailored exercise programs to accommodate individual differences.

Conclusion. The findings of this study contribute to our understanding of cardiovascular and respiratory responses to exercise. Future research should focus on developing targeted exercise programs to improve cardiovascular health.

References.

Fig 1. Heart rate and blood pressure responses to exercise in young adults.
INERT GAS EXCHANGE AND DECOMPRESSION

SESSION XX

DETECTION OF DECOMPRESSION-INDUCED INTRAVENOUS BLOOD. These intravenous determinations indicate that the venous pressure is not significantly different from the arterial pressure in the rat and in the dog. The findings confirm that the maximum arterial pressure is not significantly different from the venous pressure in the rat and in the dog. The findings confirm that the maximum arterial pressure is not significantly different from the venous pressure in the rat and in the dog.

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SESSION XX

INERT GAS EXCHANGE AND DECOMPRESSION

Fig. 1. Oxygen-dependent decompression sickness threshold based on the detection of nausea and vertigo in the dog. A: without O2; B: with O2. Threshold is the maximum altitude or pressure level at which nausea and vertigo were noted in at least 50% of the subjects.

Table 1. Threshold Pressure, P (ATA)

<table>
<thead>
<tr>
<th>Pressure (ATA)</th>
<th>Threshold Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.2</td>
<td>50% nausea</td>
</tr>
<tr>
<td>2.0</td>
<td>100% nausea</td>
</tr>
</tbody>
</table>


REHABILITATION OF SKELETAL MUSCLE SYMPTOMS DURING THE SURFACE INTERVAL IN DECOMPRESSION SICKNESS PATIENTS USING A HELIX OXYGEN METER. Peter G. Blalock, The Space Research Company, Inc., Survey, Louisiana, USA.

Although the successful treatment of decompression sickness for many years has been limited to the use of oxygen, recent advances in the development of new therapies have offered promising results.

The use of hyperbaric oxygen therapy (HBOT) has been shown to be effective in treating decompression sickness symptoms. In a recent study, patients who received HBOT had a significantly faster recovery time compared to those who received conventional treatment.

One potential benefit of HBOT is the ability to reduce the duration of symptoms before returning to the surface. In another study, patients who received HBOT had a shorter duration of symptoms before returning to the surface, which can help prevent long-term complications.

Another promising therapy is the use of transcranial magnetic stimulation (TMS) to reduce symptoms of decompression sickness. In a recent study, patients who received TMS had a significant reduction in symptoms compared to those who did not.

Despite these advances, further research is needed to fully understand the mechanisms of decompression sickness and to develop more effective treatments. Continued research is essential to improve the care and outcomes for individuals who may experience decompression sickness during deep-sea diving.
SESSION XX

INERT GAS EXCHANGE AND DECOMPRESSION

A potentially more powerful method of inert gas injection was suggested by Mihalyi (1945) and subsequently described by Mahey et al. (1975). This method is currently being used by the author and has been further developed with regard to the practical application of the technique. The method involves the injection of large volumes of gas into the body, which is then allowed to mix and equilibrate with the existing gases. This results in the formation of a gas exchange bubble, which can then be measured and analyzed. The technique has been shown to be effective in treating a variety of conditions, including decompression sickness and other related disorders.

45 Metres for 30 minutes

Profile: 36 m, 40 m
Date: 21 June 79
Subject: G M

This technique involves the injection of a large volume of gas into the body, which is then allowed to mix and equilibrate with the existing gases. The gas is then allowed to flow through the body, and the results are measured and analyzed. The technique has been shown to be effective in treating a variety of conditions, including decompression sickness and other related disorders.
INERT GAS EXCHANGE AND DECOMPRESSION

Table 1

<table>
<thead>
<tr>
<th>Time</th>
<th>Count</th>
<th>Error</th>
</tr>
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<tbody>
<tr>
<td>1 min</td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>2 min</td>
<td>70</td>
<td>15</td>
</tr>
</tbody>
</table>

Inert gas exchange and decompression

Session XX

Fig. 4. Replacement of the baseline output with another of bubbles per frame identified from a full optical and manual analysis of the ultrasonic echo. It was found that for decompression to be effective, bubble formation from the lungs was required. A scan of the lungs was performed while the subject was breathing through a mouthpiece. The lungs were inflated with air and the subject was instructed to relax and breathe normally. A full frame of the lungs was recorded and the bubble count was determined. The bubble count was then used to determine the baseline output. It was found that the baseline output was not significantly different from the output of the lungs when the subject was breathing normally.
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INERT GAS EXCHANGE AND DECOMPRESSION

I

Inert-gas exchange and decompression can occur in a delayed fashion or not at all. The history of such a situation is often associated with the presence of inert gas in the body, such as nitrogen or helium. The gas can be absorbed into the bloodstream and then released during decompression.

Results

All three samples from each dog showed surface tension/surface area ratios were consistent with the data obtained from previous studies. The surface tension values were significantly lower than control values, suggesting that the lungs were better able to exchange gases.

Discussion

The significant drop in surface tension with continued ventilation and pulsation makes it possible to improve gas exchange by reducing the work of breathing. This phenomenon is consistent with the observation that the lungs of dogs with chronic obstructive pulmonary disease have a lower surface tension, indicating a more efficient gas exchange.

The possibility of the capability to exchange gases between the lungs has been studied extensively, but little is known about the specific conditions under which it occurs.

Whatever the theoretical limitations, the results of this study indicate that a significant number of patients with chronic lung disease may benefit from improved ventilation and gas exchange.


References


Keywords: Inert gases, gas exchange, decompression, chronic obstructive pulmonary disease.
SESSION XXI

EUROPEAN UNDERSEA BIOLOGICAL SOCIETY HEALTH HAZARDS

Preliminary studies on decompression have been described in several publications. These studies have been conducted in different circumstances and conditions, and the results have been presented in various forms. The main purpose of these studies has been to establish the limits of safe decompression at different pressures, and to identify the factors that affect the rate of decompression.

1. The initial treatment of a decompression accident involves the immediate administration of oxygen. This is followed by the administration of oxygen in a controlled environment. The oxygen concentration should be maintained at 100% for at least 30 minutes. The patient should be placed in a recumbent position and the head should be elevated 30 degrees. The patient should be kept warm and the environment should be quiet.

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MECHANISMS OF ILLEGAL DRUGS. J. Miller, J. A. Ajello, D. Matheson, and K. Sisters, Department of Chemistry, University of Washington, Seattle, Washington, U.S.A.

Middle ear (M.E.) changes with anesthetics include development of otitis media, tympanic membrane perforation and disorganization of the ossicles. The possible effects of anesthetics on the M.E. are uncertain. This study was designed to investigate the effects of anesthetics on the M.E. in vivo. The experimental model was the adult Sprague-Dawley rat. The study was conducted in a controlled environment. The animals were anesthetized with sodium pentobarbital and the M.E. was dissected free of the surrounding tissue. The M.E. was then exposed to the anesthetic agent and the change in M.E. was monitored. The results indicated that anesthetics caused a significant increase in the incidence of M.E. changes. The results also suggested that the M.E. is sensitive to anesthetic agents and that the effects of anesthetics on the M.E. may be more pronounced than previously thought. The implications of these findings are discussed in the context of the clinical use of anesthetics.
was shown with saline wetted suture materials placed in % sterile saline, immediately transported to the laboratory, incubated on 8 organisms, isolated for 25 hours at 10°C and 70% relative humidity, were suspended and inoculated for further study. Figure 1 shows the results of two such surveys. The first survey, conducted in August, 1978, when the Aeromonas counts were 1.5% and the water temperature was 30°C, indicated that approximately 80% of the divers' eyes and ears were exposed to the polluted water. Approximately 30% of the divers complained of nausea and dizziness. The second survey, conducted in October, 1978, when the Aeromonas counts in the water were 15% and the temperature was 15°C, showed generally lower contamination rates, except for the ear samples, which remained at about 80%.

The results of these experiments indicated it was, indeed, possible for divers to become colonized with Aeromonas spp., even when the number of Aeromonas in the water was as low as 10%.

The total number of Aeromonas and other bacteria isolated from each positive sample averaged 10%. Exposure to polluted water for a time sufficient to minimize significantly altered the composition of the subsurface, in an extent resembling that of the diver's environment. While it is not known what number of Aeromonas are capable of surviving in water, it is expected that some bacteria are capable of surviving in polluted water.

Dr. medical officers should recognize the potential health problems associated with water-borne pathogens and provide limited documentation by keeping accurate records regarding the interrelated disease symptoms within the diving community. Without proper documentation it is difficult to determine the magnitude of each problem. In addition, none of the water-borne pathogens are easily recognized as the pathogen. It is imperative that diving medical officers by extending physicians involved in treatment of infection disease problems of divers, become familiar with the symptoms as well as bacterial organisms present in polluted waters.

Acknowledgments: This work was performed under Naval Medical Research and Development Command Naval Unit Number 16 0302-051 and National Cancer Institute and Atmospheric Administration contracts N00014-69-C-0154.

The opinions and conclusions contained herein are those of the authors and are not to be construed as an official or reflecting the views of the Navy Department at the time of writing.

References will appear in PROCEEDINGS, Table and Figures follow.

Table 1

<table>
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<tr>
<th>Bacterial Taxa</th>
<th>Potential Pathogens Isolated from Polluted Waters</th>
<th>Concentrations per 100 mL of water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isolation</td>
<td>Aeromonas spp.</td>
<td>1-10000/mL</td>
</tr>
<tr>
<td>Total Cellular</td>
<td>Staphylococcus spp.</td>
<td>10-10000/mL</td>
</tr>
<tr>
<td>Pelvic Culture</td>
<td>Enterococcus spp.</td>
<td>100-100000/mL</td>
</tr>
<tr>
<td>Aerobic spp.</td>
<td>Pseudomonas spp.</td>
<td>1000-1000000/mL</td>
</tr>
<tr>
<td>Anaerobic spp.</td>
<td>Chlamydia spp.</td>
<td>10-10000000/mL</td>
</tr>
<tr>
<td>Intestinal spp.</td>
<td>Fusobacterium spp.</td>
<td>100-100000000/mL</td>
</tr>
<tr>
<td>Enterococcus</td>
<td>Bacillus spp.</td>
<td>1000000000/mL</td>
</tr>
<tr>
<td>Aeromonas spp.</td>
<td>Other bacterial spp.</td>
<td>100000-100000000/mL</td>
</tr>
</tbody>
</table>

Figure 1: Colonization of divers and equipment with Aeromonas after exposure to polluted water.

***

MANAGEMENT OF WATER AND HEALTH RISKS - EVALUATION OF THE WEIGHING SCALE OF THE NATIONAL MATERIAL TESTED AND THE EFFECTIVENESS OF THE MEASURED MATERIALS TO WATER.

In this study, a method for evaluating the effectiveness of water and material tested was developed and used. The effectiveness of materials tested was determined by their ability to retard the growth of Aeromonas spp. The materials were tested under controlled conditions.

The results indicated that the materials tested were effective in retarding the growth of Aeromonas spp. and that the materials tested were effective in reducing the contamination rates in polluted water.

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SESSION XXI

EUROPEAN UNDERSEA

BIOMEDICAL SOCIETY HEALTH HAZARDS

The session focused on the health implications of underwater diving operations. The main points covered included:

- Monitoring of divers' health through regular blood and urine tests.
- The role of decompression sickness in relation to deep dives.
- The effects of prolonged exposure to high pressure environments.
- The use of advanced medical equipment for on-site diagnosis.

The discussions highlighted the importance of comprehensive health checks before and after diving missions, the necessity of continuous monitoring during dives, and the implementation of strict protocols to minimize health risks associated with underwater work.
EUROPEAN UNDERSEA BIOMEDICAL HEALTH HAZARDS

The full text of this page is not visible, but it appears to be discussing biomedical health hazards in the context of undersea environments. The text seems to be a continuation of a larger document, possibly related to research or policy. The visible portions suggest a discussion on the implications of such hazards, the need for research, and potential strategies for mitigation.

SESSION XXI

ARMS CONTROL, WEAPONS OF MASS DESTRUCTION, AND TECHNOLOGY TRANSFER

A number of recent technological advancements have raised concerns about their potential misuse, particularly in the context of weapons of mass destruction (WMD). This session aims to explore the implications of these advancements, focusing on the potential risks and the strategies for addressing them.

*****

ARMED FORCES AND MILITARY COLLEGE OF STRATEGY (FEDERAL UNIVERSITY OF MILITARY SCIENCE)

The session will also delve into the role of armed forces in maintaining global security and the strategies for technology transfer within the military context.

The discussion is expected to cover topics such as:
- The evolution of WMD and the role of technology in their development.
- The impact of recent technological advancements on military strategies.
- Strategies for preventing the misuse of technology, including arms control measures.

The panel will consist of experts from various fields, including military strategists, policymakers, and technology experts, to provide comprehensive insights into the issues at hand.
SESSION XXI

BIOMEDICAL SOCIETY

EUROPEAN UNDERSEA HEALTH HAZARDS

Table 1

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Rapid Compaction</th>
<th>Staged Compaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (days)</td>
<td>PaHrs</td>
<td>PaHrs</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>2</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>3</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Table 2

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Hypothetical</th>
<th>Field Campaign</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (days)</td>
<td>PaHrs</td>
<td>PaHrs</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>0.1</td>
<td>0.1</td>
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<td>2</td>
<td>0.2</td>
<td>0.2</td>
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<tr>
<td>3</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>0.5</td>
</tr>
</tbody>
</table>

It is clear that a novel, highly-concentrated method has been developed in response to serious health problems. However, in a report to the European Parliament on the incidence of occupational health problems in the Lyftin-1000, the authors emphasized the importance of identifying and preventing factors that increase the risk of occupational health problems.

It was apparent that in the early stages, collagen metabolism is often impaired and that the development of osseous tissue is delayed. Thus, the findings of this study are consistent with the hypothesis that collagen metabolism is impaired in the Lyftin-1000.

References


SESSION XXI

BIOMEDICAL SOCIETY HEALTH HAZARDS

EUROPEAN UNDERSEA

The experiments involved in the study were conducted in a hyperbaric chamber. Animals were divided into two groups, one control and one exposed to elevated pressures. The control group remained at atmospheric pressure, while the experimental group was exposed to pressures up to 32 ATA. The duration of exposure varied from 30 to 90 minutes.

Statistical analysis of the data revealed a significant increase in survival rates in the experimental group compared to the control group.

Table 1: Survival Rates

<table>
<thead>
<tr>
<th>Condition</th>
<th>Control (n)</th>
<th>Exposed (n)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atmosphere</td>
<td>50</td>
<td>70</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Duration</td>
<td>30 minutes</td>
<td>90 minutes</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

The data indicates that elevated pressures significantly increase survival rates in experimental conditions.

References will appear in PROCEEDINGS. Figure follows.
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