CONTENTS

ACTIONS TAKEN AT SPRING MEETING

April 12-17, 1971

ELECTIONS - R. M. Berne was elected to the position of President-Elect. Bodil M. Schmidt-Nielsen was elected to a full four-year term on Council.

E. E. Selkurt was elected to fill the unexpired term (three years) of R. M. Berne on Council.

All candidates nominated by Council were elected to membership (See Newly Elected Members).

DUES - Council announced that beginning July 1, 1972 dues for regular members will be $25 per year. Associate members dues will be $10 per year. Retired members pay no dues. Dues run from July to July.

PUBLICATIONS - It was announced that the subscription price for Physiological Reviews will be raised to $20 per year and that for the Journal of Neurophysiology to $25 per year beginning January 1, 1972. Subscriptions run from January to January.

ABSTRACTS - All qualified abstracts received for 1971 were programmed. However for 1972 the Society will invoke the 1970 rule of limiting to 850 the number of papers for oral presentation.

MEMBERSHIP STATUS

April 1, 1971

Active Members	3391
Retired Members	192
Honorary Members	16
Associate Members	331
	3930

SUSTAINING ASSOCIATES

Abbott Laboratories
Ayerst Laboratories
Burroughs Wellcome & Co., Inc.
CIBA Pharmaceutical Products, Inc.
Eli Lilly & Co.
Gilford Instrument Laboratories
Gilson Medical Electronics
Harvard Apparatus Co.
Hoffman-LaRoche Laboratories
Lakeside Laboratories, Inc.
Merck Sharp & Dohme Research Laboratories
Narco Bio-Systems, Inc.
The Norwich Pharmacal Co.
Chas. Pfizer & Co., Inc.
Riker Laboratories, Inc.
A. H. Robins Co., Inc.
Smith Kline and French Laboratories
Warner-Lambert Research Institute
Waverly Press-Williams & Wilkins Co.
Wyeth Laboratories

DEATHS SINCE FALL MEETING 1970

Samuel Amberg - 11/29/70 - Assoc. Prof. Emeritus, Pediat., Mayo Clinic
Frank R. Blood - 1/31/71 - Dir., Div. Toxicol., Vanderbilt Univ.
Charles W. Crumpton - 2/6/71 - Prof. Med., Univ. of Wis., Madison
Percy M. Dawson - 9/27/70 - Lecturer, Stanford Univ.
Herbert M. Evans - 3/6/71 - Prof. Anat., Univ. of Calif., Berkeley
Eugene M.K. Geiling - 1/12/71 - Prof. Pharmacol., Howard Univ.
Frank A. Hartman - 3/21/71 - Prof. Emeritus Physiol., Ohio State
Charlotte Haywood - 2/6/71 - Prof. Emeritus Physiol., Mt. Holyoke
Shirley A. Johnson - 9/11/70 - Res. Physiologist, VA Hosp., Washington, D. C.
Joseph E. Markee - 11/27/70 - Prof. Anat., Duke Univ. Sch. Med.
Hans Molitor - 8/5/70 - Dir. Scientific Relations - Merck Sharp & Dohme Res. Labs.
Carl A. Moyer - 5/29/70 - Prof. Biol. Sci., Mich. Technological Univ. at Houghton
Leo Oliner - 1/9/71 - Chief, Res. Endocrinol. & Metabolism, VA Hosp., Washington, D. C.
Eugene L. Opie - 3/12/71 - Prof. Emeritus Physiol., Cornell Univ.
Wilhelm Raab - 9/21/70 - Emeritus Prof. Exptl. Med., Univ. Vermont
Arturo S. Rosenblueth - 9/20/70 - Dir., Ctr. de Investigacion & de Estudios Avanzados del IPN, Mexico City
Philip E. Smith - 12/8/70 - Prof. Emeritus, Dept. Anat., Stanford Univ.
Howard G. Swann - 9/14/70 - Prof. Physiol., Univ. of Texas Med. Br., Galveston
Harry B. Van Dyke-2/14/71-Prof. Emeritus Pharmacol., Columbia Univ.

HONORARY MEMBER

Carlos Monge - 2/15/70 - Emeritus Prof. Med., Univ. of San Marcos, Lima, Peru

ASSOCIATE MEMBERS

Kenneth E. Kellogg-7/19/70 - Prof. Physiol., Coll. Med. Evangelists, Loma Linda
William Steinberger - 4/10/71 - Dept. of Physiol., Univ. of Michigan

50-YEAR MEMBERS

Edward F. Adolph
Walter C. Alvarez
Joseph C. Aub
George A. Baitsell
J. Percy Baumberger
Olaf Bergeim
Harold C. Bradley
Thorne M. Carpenter
Dayton J. Edwards
Lester M. Dragstedt
Mable P. Fitzgerald
Carl H. Greene
Charles M. Gruber
Harold L. Higgins
Paul E. Howe
Andrew C. Ivy
Dennis E. Jackson
Norman M. Keith
Edward C. Kendall
Benjamin Kramer
Henry Laurens
Edward Lodholz
David Marine
Jesse F. McClendon
Walter R. Miles
Frederick R. Miller
Clarence A. Mills
Sergius Morgulis
Stuart Mudd
Leonard B. Nice
Alfred C. Redfield
Andrew H. Ryan
Charles D. Snyder
Joseph T. Wearn
George H. Whipple
Rosalind Wulzen

NEWLY ELECTED MEMBERS

The following, nominated by Council, were elected to membership in the Society at the Spring Meeting, 1971.

HONORARY MEMBER

GUTMANN, Ernst: Prof. Physiol., Charles Univ., Prague, Czechoslovakia

REGULAR MEMBERS

ANDERSON, Nels C.: Asst. Prof. Physiol. & Pharmacol., Duke Univ.
BADE, Maria L.: Asst. Prof. Biol., Boston College
BARTLETT, Donald, Jr.: Postdoct. Res. Fellow Physiol., Dartmouth
BECK, Ronald R.: Asst. Prof. Physiol., Indiana Univ. Med. Ctr.
BITTNER, George D.: Asst. Prof. Zool., Univ. of Texas, Austin
BLANKENSHIP, James E.: Postdoct. Res. Fellow, Lab. Neurophysiol., NIMH
BLAUFOX, Morton D.: Asst. Prof. Med. & Radiol., Albert Einstein
BOERTH, Robert C.: Asst. Prof. Pharmacol., Emory Univ., Atlanta

BOLEY, Scott J.: Assoc. Prof. Surg., Albert Einstein Coll. Med.
BOVE, Alfred A.: Asst. Prof. Physiol., Univ. of Minnesota Grad. Sch. Med.
BRODY, Jerome S.: Asst. Prof. Med., Univ. of Pennsylvania
CHASE, Michael H.: Asst. Prof. Physiol., UCLA
COOKE, Allan R.: Assoc. Prof. Internal Med., Univ. of Iowa
COSTIN, Anatol: Res. Assoc. Anatomy, UCLA
COX, Robert H.: Asst. Prof. Physiol., Univ. of Pennsylvania
CRASS, Maurice F. III: Asst. Prof. Med. & Physiol., Univ. Florida
DOBRIN, Philip B.: Asst. Prof. Physiol., Loyola Univ., Stritch Sch. Med.
EFFROS, Richard M.: Asst. Prof. Med., New Jersey Coll. Med.
FISHER, Aron B.: Asst. Prof. Med. & Assoc. in Physiol., Univ. of Pennsylvania
FRANKLIN, Eleanor L. Ison: Assoc. Prof. Physiol. & Biophys., Howard Univ.
GALINDO, Anibal: Assoc. Prof. Anesthesiol., Univ. of Washington
GEE, J. Bernard L.: Assoc. Prof. Med., Yale Univ. Sch. Med.
GELPERIN, Alan: Asst. Prof. Biol., Princeton Univ.
GOOTMAN, Phyllis M.: Instructor Physiol., Albert Einstein Coll. Med.
HAGINO, Nobuyoshi: Head, Dept. Neurophysiol., Southwest Fndn. for Res. & Education
HALIKAS, George C.: Asst. Prof. Physiol., Univ. of Alaska
HAN, Paul W.: Asst. Prof. Physiol., Univ. of Pennsylvania Sch. Med.
HOMER, Louis D.: Assoc. Prof. Med. Sciences, Brown Univ.
HOPKINS, Thomas F.: Staff Scientist, Div. Regulatory Biol., Univ. of Connecticut
HUGGINS, Sara E.: Prof. Biol., Univ. of Houston
JAEGER, Marc J.: Assoc. Prof. Dent. & Physiol., Univ. of Florida
JOHNSON, Thomas F.: Asst. Prof. Physiol., Howard Univ.
KELLY, Keith A.: Asst. Prof. Surg., Mayo Grad. Sch. of Med.
KNOCHEL, James P.: Prof. Med., Univ. of Texas, Southwestern Med. Sch.
LAZZARA, Ralph: Lt. Col., US Army Med. Res. & Nutr. Lab., Physiol., Fitzsimmons Hosp.
LEVITAN, Herbert: Spec. Res. Fellow, NINDS, NIH
LEVITT, Melvin: Assoc. Prof. Physiol., Wake Forest Univ.
McCALLY, Michael: Chief, Environmental Med., Wright-Patterson AFB
MAACK, Thomas M.: Asst. Prof. Physiol., Cornell Univ. Med. Coll.
MASLAND, William S.: Asst. Prof. Physiol. & Neurol., Univ. of Pennsylvania Sch. Med.
MASSARO, Donald J.: Assoc. Prof. Med., George Washington Univ.
MOGENSON, Gordon J.: Prof. Physiol., Univ. of Western Ontario
MORAD, Martin: Asst. Prof. Physiol., Univ. of Pennsylvania Sch. Med.
MORKIN, Eugene: Asst. Prof. Med., Harvard Med. Sch.
MORRISON, Martin: Chrmn., Dept. Biochem., St. Jude's Children's Res. Hosp., Memphis
MURRISH, David E.: Res. Assoc., Dept. Zool., Duke Univ.
NAGEL, Eugene L.: Assoc. Prof. Anesthesiol., Univ. of Miami
NAVAR, Luis G.: Asst. Prof. Physiol. & Biophys., Univ. Med. Ctr., Jackson, Miss.
PAGE, Charles H.: Asst. Prof. Zool., Ohio University
PEARLMAN, Alan L.: Asst. Prof. Physiol. & Biophys., Washington Univ.

PERL, William: Assoc. Prof. Med., New Jersey Coll. Med. & Dent.
POMERANZ, Bruce H.: Asst. Prof. Zool., Univ. of Toronto
PUTNAM, Serpas J.: Asst. Prof. Med. Sci., Brown Univ.
RALPH, Charles L.: Prof. Biol., Univ. of Pittsburgh
RAMAZZOTTO, Louis J.: Chrmn., Physiol. Dept., Fairleigh Dickinson Univ.
RUPERT, Allen L.: Auditory Electrophysiol., Callier Hearing & Speech Ctr., Dallas
SACHS, John R.: Asst. Prof. Physiol., Yale Univ. Sch. of Med.
SAHA, Jagneswar: Asst. Prof. Physiol. & Biochem., Michigan Coll. of Osteopathic Med.
SANTOLUCITO, John A.: Leader, Physiol., Group, Perrine Primate Res. Br., USPHS
SANTOS-MARTINEZ, Jesus: Prof. Physiol., Univ. of Puerto Rico
SCHNEIDER, Edward G.: Postdoct. Fellow, Physiol., Univ. of Missouri
SCHRIER, Robert W.: Asst. Prof. Med., Univ. of California, S. F.
SENTURIA, Jerome B.: Asst. Prof. Biol., Cleveland State Univ.
SHAMOO, Adil E.: Asst. Physiol., Mt. Sinai Med. Sch., City Univ. of New York
SHARMA, Sansar C.: Res. Assoc. Biol. Dept., Washington Univ.
SHIELDS, Jimmie L.: Chief, Spec. Programs Br., Natl. Heart & Lung Inst., NIH
SPICKLER, J. William: Dir. Physiol. & Biol. Sect., Cox Heart Inst.
SUDDICK, Richard P.: Chrmn., Dept. Oral Biol., Creighton Univ.
TALBOTT, Richard E.: Res. Assoc. Physiol., Univ. of Oregon
TAUB, Edward: Chief, Neuropsychol. Dept., Inst. for Behavioral Res., Silver Spring, Md.
TAYLOR, Stuart R.: Instr., Pharmacol., State Univ. of New York., Downstate Med. Ctr.
THURBER, Robert E.: Assoc. Prof. Physiol., Jefferson Med. Coll.
VERRIER, Richard L.: NIH Postdoct. Fellow, Univ. of Michigan
WEISS, Harvey J.: Dir., Div. Hematol., Columbia Univ. Coll. P & S
WHITEHORN, David: Asst. Prof. Physiol. & Biophys., Univ. of Vermont
WURSTER, Robert D.: Asst. Prof. Physiol., Loyola Univ., Stritch Sch. of Med.
YORK, Donald H.: Asst. Prof. Physiol., Queen's Univ., Kingston, Ont.

ASSOCIATE MEMBERS

ARMOUR, John A.: Res. Assoc. Physiol., Loyola Univ., Stritch Sch. of Med.
BEEUWKES, Reinier III: Instr. Physiol., Harvard Med. Sch.
COULTER, Dwight B.: Asst. Prof. Physiol. & Pharmacol., Iowa State Univ.
DHINDSA, Dharam S.: Res. Fellow Med., Univ. of Oregon Med. Sch.
ELLIOTT, John C.: Staff Physiologist, Lovelace Foundation
FARMER, Robert W.: Res. Specialist Neuroendocrinol., Texas Res. Inst. Mental Sci.
GARRICK, Rita Anne: Teaching Asst. Zool.-Physiol., Rutgers Univ.
GEIS, William P.: NIH Postdoct. Res. Fellow, Loyola Univ., Stritch Sch. of Med.
GOLDBERG, Jack M.: NIH Predoct. Training Grantee, Loyola Univ.

HARTMAN, H. Bernard: Asst. Prof. Zool., Univ. of Iowa
JOHNSON, Dale R.: Predoct. Student Physiol., Marquette Sch. Med.
KASTELLA, Kenneth G.: Res. Assoc. Physiol., Univ. of New Mexico
KEYES, Jack L.: Grad. Student Physiol., Cornell Univ. Med. Coll.
KOHLMEIER, Ronald H.: Asst. Prof. Physiol. & Pharmacol., Iowa State Univ.
KRAFT-HUNTER, Frances S.: Asst. Prof. Physiol., Howard Univ.
KUNESH, Jerry P.: Asst. Prof. Physiol. & Pharmacol., Iowa State Univ.
MACKOWIAK, Robert C.: Asst. Prof. Physiol., Thomas Jefferson Univ.
RAINBOLT, Mary L.: Chrmn., Biology Dept., Illinois College
RICHARDSON, Daniel R.: Asst. Res. Bioengineer, Univ. of California, San Diego
SPURGEON, Harold A.: NIH Predoct. Fellow Physiol., Loyola Univ., Stritch Sch. Med.
VIDOLI, Vivian A.: Asst. Prof. Biol., Fresno State College
YELLIN, Herbert: Res. Physiol., NINDS, NIH

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IUPS CONGRESS HISTORY

The APS Office holds some copies of the History of the International Congress of Physiology, 1889-1938, edited by Wallace O. Fenn. The volume includes Franklin's History of the Physiological Congresses during the first 50 years, Zotterman's account of the Minnekahda Voyage from Europe to the Boston Congress in 1929, accounts of the Congresses, 1938-1968 by officials of those Congresses, and a history of IUPS from 1953 to 1968 by the editor. Copies are available for one dollar from the APS, 9650 Rockville Pike, Bethesda, Maryland 20014, as long as the supply lasts.

ANNOUNCEMENT
JANE NUGENT COCHEMS COMPETITION

The University of Colorado School of Medicine announces the Tenth Annual Cochems Competition, funds for which were provided in the will of the late Mrs. Jane Nugent Cochems. A prize of $2500 will be awarded to the author of the best paper in the field of "Thrombophlebitis and Basic Vascular Problems." Basic vascular problems under consideration in this instance should be concerned with the underlying mechanisms or processes of vascular disease, particularly those associated with thrombosis, but not necessarily restricted to it.

The competition is open to all persons holding the doctorate degree and entries must be received in triplicate, including all charts, illustrations and photographs, on or before November 15, 1971. For income tax reasons, eligibility is limited to those physicians who are subject to U. S. income tax regulations.

The Colorado National Bank of Denver, Trustees under the will of Jane Nugent Cochems, has requested the Dean of the University of Colorado School of Medicine to conduct the competition. The judges appointed by the Dean are Dr. Sol Sherry, Professor and Chairman of the Department of Medicine, Temple University School of Medicine, and Dr. Michael E. DeBakey, Professor and Head of the Department of Surgery, Baylor University College of Medicine. Decisions of the judges are final, and they may elect at their discretion not to award the prize.

Papers submitted in the competition may not be published until after the winner has been announced early in 1972. At that time, the winning paper and all others may be published at the discretion of individual authors. It should be noted, however, that sponsors and judges of the competition will not assume any responsibility for submitting manuscripts for publication nor for any costs incident thereto. The winning paper, if published, must carry the designation, "Awarded the Jane Nugent Cochems Prize."

No entry blank or application form is required. There are no restrictive rules regarding length or format of the manuscript, joint authorship, or inclusion of such materials as pictures, charts, figures, etc. It is not required that the paper include results of original experimental work, nor that it be based on personal clinical experience. All manuscripts must be typed with double spacing and each copy together with accompanying illustrations, etc., must be submitted in a folder or cover. On request, the original copy of the manuscript will be returned if accompanied by a stamped, addressed envelope. Papers will be judged on originality, content, clarity, and critical value.

Inquiries regarding the competition and all manuscripts should be submitted to Dr. David W. Talmage, Dean, School of Medicine, University of Colorado Medical Center, 4200 E. Ninth Avenue, Denver, Colorado 80220.

1970 FISCAL REPORTS

The Bylaws of the Society (Article VII) identify the three principal funds which are used for the fiscal management of the Society's affairs. The behavior of these funds during the year 1970 is summerized below.

SOCIETY OPERATING FUND

This fund is used for direct services to members through arrangement of meetings, programs, etc.; the expenses and activities of Council and its committees (other than publications); the generation and distribution of educational materials; and the supervision of the business affairs of the Society.

INCOME

Membership Dues	$57,806	(67%)
Sustaining Associates Contributions	5,147	(6%)
Reimbursement for services rendered in connection with the Fed. Spring Meeting	15,198	(18%)
Interest (on advance monies received)	4,884	(6%)
Fall Meeting (net)	2,188	(2%)
Other Income (Sale of educational and other material, etc.)	876	(1%)
Total	$86,099	

EXPENSES

Salaries and Benefits	$38,763	(42%)
Dues to Fed. and other Organizations	23,471	(26%)
Office Rental (From Fed.)	4,660	(5%)
Travel and Subsistence for officers and committees	5,448	(6%)
Education Committee and Office	4,336	(5%)
Bowditch Lecture	500	
Mail, Telephone, Supplies & Misc.	5,146	(6%)
Business Office expenses	9,187	(10%)
Total	$91,511	

Excess of Expenses over Income (deficit) ($5,412)

PUBLICATIONS OPERATING FUND

This fund represents the functions of the Society as a publisher of scientific journals.

INCOME

Subscriptions	$459,080	(63%)
Sale of Reprints (net)	66,668	(10%)
Sale of Back and Single Issues	15,287	(2%)
Page Charges	141,837	(19%)
Advertising (net)	11,039	(2%)
Interest (on advance subscriptions, etc.)	24,391	(3%)
Royalties	5,461	(1%)
Miscellaneous	- 5,674	(1%)
Total	$729,437	

EXPENSES

Printing and Engraving	$451,168	(55%)
Salaries and Benefits	150,262	(18%)
Mail, Telephone, Supplies, etc.	58,276	(7%)
Office Rental	16,675	(2%)
Section Editor Expenses & Professional Services	50,104	(6%)
Travel & Subsistence for Officers, Committee, and Editors	9,303	(1%)
Miscellaneous	1,656	
Business Office Expenses	82,680	(10%)
Subtotal	$820,124	
Allocated to Handbook Operations	(11,087)	
Allocated Advertising Costs	(4,556)	
Adjusted Total	$804,481	
Excess of Expenses over Income (deficit)	(75,044)	

PUBLICATIONS CONTINGENCY AND RESERVE FUND

This is a reserve fund which the Society has accumulated over many years. Its existence is dictated by prudent business practice, in case of any severe reversals etc. the journals can continue to be published for at least one year following such reversals. The Society has very few tangible, salable assets that could be used as collateral for borrowing money. The fund's size should be from one to two times the annual operating costs of the publication operations, including the Handbooks. It is held in long term investments managed by an investment counselor. Its uses are carefully spelled out in Article VII, Section 3 of the Society Bylaws.

Balance Dec. 31, 1969 (market value)	$1,009,315
Dividend and Interest paid to APS in 1970	43,548
Balance Dec. 31, 1970 (market value)	1,039,508
Gain in market value during 1970	30,193

Note: For long-range financial operations see The Physiologist, Vol.13, No.2, May 1970, pp.60-69.

SECOND NATIONAL BIOLOGICAL CONGRESS

The Congress, to be held 23-26 October 1971 at the Fountainbleu Hotel, Miami Beach, Florida, is the second in a series of Congresses sponsored by the American Institute of Biological Sciences. The Congresses are aimed at a better public understanding of environmental problems and the efforts of biologists to resolve them. The mornings will be utilized for various symposia and the afternoons by programs of various biological Societies. The last day is to be set aside for various local field trips. There will be one evening session devoted to a program titled Biology in National Policy. For further information write AIBS, 3900 Wisconsin Avenue, Washington, D. C. 20016.

SYMPOSIUM ON CEREBRAL BLOOD FLOW

An International Symposium on Cerebral Blood Flow Regulation in Acute Brain Lesions will be held in Rome and Siena, Italy, October 28-31, 1971. Deadline for application and submission of abstracts is May 31, 1971. Further details may be obtained from Dr. C. Fieschi, Department of Neurology, University Hospital, Siena, Italy.

FALL MEETING

University of Kansas at Lawrence and
University of Kansas Medical Center at
Kansas City, Kansas, August 16-19, 1971

The physiologists of both campuses of the University of Kansas cordially invite all members of the American Physiological Society and their guests to attend the 1971 Fall Meeting. The first three days of the meeting, August 16, 17, 18, will be held on the campus at Lawrence, with the final day, August 19, at the Medical Center in Kansas City, Kansas.

The refresher course on Neuroendocrinology has been organized by Dr. William F. Ganong. Participating in this teaching effort with Dr. Ganong will be Drs. Wylie Vale, S. M. McCann, Ernst Knobil, Leonard Share, Donald Pfaff, and Robert Gorski. This refresher course will be held on Monday, August 16 from 9:00-5:00. Scientific sessions will be held from 9:00-12:00 and 1:30-4:00 on Tuesday, August 17, and Wednesday, August 18.

Five symposia have been organized.

1. Secretory Processes in the Endocrine System, organized by Dr. William F. Ganong. 9:00-12:00 Tuesday, August 17.

2. Blood-Brain Barrier, organized by Dr. Alan M. Thompson 9:00-12:00 Wednesday, August 18.

These symposia will run simultaneously with scientific sessions on the campus at Lawrence.

The following three symposia will be presented from 9:30-12:30 on Thursday, August 19, at the University of Kansas Medical Center in Kansas City, Kansas.

3. Fibrous Organelles, organized by Dr. Paul Burton.

4. Tissue Buffering, organized by Dr. E. B. Brown, Jr.

5. Mechanisms of Muscle Contraction, organized by Drs. R. L. Clancy and Merrill Tarr.

Look for the detailed programs of these symposia in the preliminary announcement brochure.

Dr. J. Alan Herd, Associate Professor of Physiology, Harvard Medical School, will deliver the Bowditch Lecture on Wednesday afternoon, August 18 at 4:30 PM. His subject will be, "The Physiology of Strong Emotions: Cannon's Scientific Legacy Re-examined."

Dr. A. Clifford Barger will deliver the Past-President's Address on the subject: "To Assist Young Men and Women of Promise in the Study of Physiology: The Porter Development Program" at the Annual

Banquet which will be held at 7:00 Wednesday evening, August 18.

Registrants will be housed in McCollum Hall - a new, air-conditioned dormitory which will accommodate 1,000 persons. Participants who prefer to live off the campus will have their choice of several new motels within one mile of the campus.

Registration will begin Sunday, August 15, at 1:00 PM and continue from 9:00 until 5:00 throughout the meetings. Transportation from the airport in Kansas City will be available Sunday afternoon and evening, and all day Monday. The University of Kansas in Lawrence is adjacent to the Kansas Turnpike and adequate parking space is available at the residence halls for all who wish to drive to Lawrence.

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INTERNATIONAL CONFERENCE ON MEDICAL PHYSICS

The Third International Conference on Medical Physics will be held at Gothenburg, Sweden, July 30 to August 4, 1972. For further information contact the following before October 1971.

Roland Kadefors
Secretary General, 3rd ICMP
Dept. of Applied Electronics
Chalmers University of Technology
402 20 Göteborg, Sweden

A HANDBOOK OF PHYSIOLOGY IS OFF TO THE PRINTER

About a year and a half ago the Society established a new office in Bethesda for the coordination of activities concerning the Handbook of Physiology. This office is now pleased to announce that the first manuscripts for the Handbook volume on the endocrine pancreas have been sent to the printer and we expect the publication date to be at the beginning of 1972.

The volume on the endocrine pancreas is the first in an extensive series that will make up a Section on Endocrinology. This vast undertaking has only been possible through the cooperation of the Society and a large number of devoted individuals. The Handbook Editorial Committee presently composed of J. M. Brookhart, G. F. Cahill, L. D. Carlson, C. L. Prosser, and A. P. Fishman (chairman) proposes topics, recruits appropriate editors and helps editors organize the volume contents. The choice of Drs. Roy O. Greep and E. B. Astwood as editors for the Section on Endocrinology was indeed wise. The section will be composed of about 250 chapters in six volumes. These volumes and their editors are:

The Endocrine Pancreas	D. F. Steiner N. Freinkel
The Thyroid and Parathyroid Glands Thyroid	M. Greer D. H. Solomon
Parathyroid	G. Aurbach
The Reproductive System - Female	R. O. Greep
The Reproductive System - Male	R. O. Greep
The Adrenal Glands Adrenal Cortex	G. Sayers
Adrenal Medulla	H. Blaschko A. D. Smith
The Pituitary Gland and Its Control Adenohypophysis	E. Knobil
Neurohypophysis	W. Sawyer

Drs. Freinkel and Steiner invited authors to write 47 chapters on various aspects on the endocrinology of the endocrine pancreas. These chapters have been subdivided into 8 subareas:

1. Morphogenesis and Development

2. Chemistry and Biosynthesis of Hormones

3. Biochemical Organization of Islet Tissue

4. Pathology of the Endocrine Pancreas

5. Molecular Basis of Pancreatic Hormone Action

6. Pancreatic Hormones and the Metabolism of Exogenous Fuels (The Fed State)

7. Pancreatic Hormones and the Metabolism of Endogenous Fuels (The Fasted State)

8. Clinical and Experimental Disorders of Insulin and/or Glucagon Secretion

The coverage is thorough and similar topics discussed from different viewpoints pinpoint critical areas and should stimulate further study. The papers range from traditional reviews to reports containing much previously unreported data.

The contribution of devoted individuals needed to produce such a series does not end with the authors and scientific editors. The editorial office has been fortunate in being able to hire two experienced copy editors for work on this volume, Mrs. Doris Morton and Mrs. Kathleen Lamar and to retain the services of Mrs. Ruth Ballard, who prepares figures for the engraver Mr. Baker.

All manuscripts are returned to authors, so that they may answer questions and verify editorial suggestions, before the chapters are marked by the copy editors for the printer. Miss Louise Pastuck is responsible for the smooth flow of manuscripts between the various parties and for the many important details involved in the production of a book.

Miss Sara Leslie advises the executive editor, particularly in his dealings with the printer, Waverly Press. Here too we are fortunate to have an experienced hand to help, as Mrs. Caral Nolley continues as the representative of Waverly Press. Some changes will be noticed in the format of the new volumes, e.g. there will be more text per page and the half tones will have greater resolution. We believe the choices that have been made are a proper balance between highest quality and ever increasing costs.

The executive editor, Stephen R. Geiger, acts as intermediary between authors, scientific editors, editorial staff, and printer.

In 1971 the Handbook editorial office began work on the remaining volumes of the Section on Endocrinology. Excellent progress has been made on the volumes on the reproductive system and we hope to provide you with a similar favorable progress report on these volumes and the others in the section in the near future.

ACTIVITIES OF THE EDUCATION OFFICE

ORR E. REYNOLDS

Dr. C. Ladd Prosser, then President of the American Physiological Society proposed the establishment of an Education Office by the Society in this publication in November 1969 (The Physiologist, Vol. 12, No. 4, Nov. 1969, p. 434). After a favorable response to this suggestion from the membership, the Education Office was established and the author was employed as Education Officer in November 1970.

Since then several activities generated by the Education Committee, now chaired by Dr. Jack L. Kostyo, Chairman of the Physiology Department, Emory University, Atlanta, Georgia, have been brought to fruition and several others initiated with the participation of the Education Office.

A survey of physiologists teaching in college biology departments has indicated strong interest in publication by APS of a newsletter for physiology teachers. The first issue of this newsletter, The Physiology Teacher appeared in April 1971. Subscribers to The Physiologist were included in the initial distribution of the newsletter. Although the subscribers to The Physiologist do not represent the focus of the audience to whom this newsletter is primarily addressed, it was felt that some of these recipients might wish to subscribe, and those that did not, being APS members, would wish to know of this activity of the Education Office.

Another proposed activity of APS, strongly supported by the survey of biology departments, was for workshops run by outstanding physiology departments, for the communication of new teaching approaches to physiology teachers in college biology departments. A pilot workshop of this type was successfully developed and conducted by Dr. David F. Bohr of the physiology department of the University of Michigan on two weekends in November and December 1970. With the success of this pilot workshop, additional ones are planned in other geographical areas for 1971.

The Education Committee has been considering matters of priorities in physiological education with respect to educational level and particular approaches that might be adapted. Although the spread of interest is very wide among the members of the Education Committee, a predominant interest in physiology teaching at the undergraduate college level and in medical education exists. Approaches that are considered highly desirable in these areas are the provision of teaching materials, i.e. audiovisual aids, programmed instruction modules, and stimulation of teaching by such programs as the newsletter and workshop described above.

In the matter of audiovisual aids, the Education Office has been working closely with the National Medical Audiovisual Center of the National Library of Medicine with the objective of providing as wide a range of audiovisual materials on physiology, as can be located and reviewed, and as can be developed. The author recently visited a number of departments of physiology around the country and is impressed by the number and variety of approaches to educational methods, including

audiovisual aids, that are being developed in these departments. The Education Office would therefore like to use the mechanism of this publication as a means of securing further information on this subject from the membership of the Society. Included in the envelope in which you receive this copy of The Physiologist is a one page questionnaire which we hope you will find time to answer. In the future this same mechanism will be used for surveying the activities, opinions and attitudes of members on other topics related to physiological education of interest to physiologists. The results of an analysis of the enclosed questionnaire and such subsequent ones as may be circulated will be presented in future brief articles in The Physiologist.

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FIRST ANNUAL MEETING OF THE SOCIETY FOR NEUROSCIENCE

The Society for Neuroscience wishes to announce that its first annual meeting will be held in Washington, D. C., October 27-30,1971. Abstracts of communications are invited which provide new data or are concerned with new concepts and principles in any of the fields of the neurosciences.

Special program groupings are planned in such areas as: Critical Periods in CNS Development; Neural Nets in Small Brains and in Tissue Culture; Synaptic Mechanisms, the Relation Between Morphology and Function; Order and Disorder in Movement; The Relation Between Psychophysical Measures of Sensation and Neural Mechanisms; Macromolecular Mechanisms in the Neuron.

Abstracts will be accepted from any member, and from any person whose abstract is sponsored by a member of the Society.

For detailed information write:

Neuroscience Meeting Headquarters
1629 K Street, N.W., Suite 700
Washington, D. C. 20006

The deadline for submission of abstracts is June 1, 1971.

INTRACELLULAR PO_2 IN HEART AND SKELETAL MUSCLE [1, 2]

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Introduction

Earlier work in our laboratory (17, 18) and some results of others (19) indicated to us that O_2 availability might, in some tissues, at least, limit O_2 consumption. We suggested that some of the resting O_2 consumption might simply have the function of generating heat, but that during activity the energy might be utilized for the active process.

One approach to the problem was to measure cell PO_2 in vivo in order to decide whether oxygen tension was low enough to be a limiting factor in cell respiration, as judged by measurements of the critical PO_2 made by others in isolated mitochondria (13) and in vivo (5).

Methods

We early decided to use the amperometric method for the oxygen determinations (2). Basically, the method makes use of the fact that at an applied potential in the range of about 0.5 and 1.0 V the current, in most biological systems, is almost entirely carried by oxygen molecules. The cathode (Fig. 1) should be an inert metal, and the anode as non-polarizable as possible. It will be obvious that since O_2 molecules must diffuse toward the cathode the diffusion coefficient of the conducting medium around the cathode should be the same during calibration as during use in the tissue. Clark (1) circumvented much of this problem by covering both the anode and cathode with an O_2-permeable membrane so that the current flows only in the electrolyte under the membrane. To date it has proven impossible to make the Clark-type electrode with a tip smaller than 5 μ (16). Incidentally, it is comforting to us that measurements of brain PO_2 with this electrode agree with our own measurements in this tissue (21).

We have concentrated on making a small but separate cathode (henceforth "electrode"). The technique for making the electrode has been published (20), but briefly it consists of a metal-filled glass capillary tube drawn out with a long taper. The tip is sharply bevelled on a rotating

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[1]Taken from the introductory remarks given at the session on Peripheral Circulation I at the 1971 Federation Meetings. These studies have been supported in part by grant numbers HE 11906, HE 12703, and FR 5631 from the U.S. Public Health Service and a grant from the NE Ohio Heart Association.

[2]Thanks are given to the many people who helped make these studies possible, particularly Dr. Pankajam Nair.

drum with diamond dust on its surface. (This procedure was not formerly used.) Usable electrodes measure 1-3 μ at the beginning of the bevel. Unless a recess in the tip occurs naturally, the metal in the tip is removed electrolytically to make a recess of 10-30 μ. A layer of gold (2-10 μ) is plated on the metal in the recess and the remainder of the recess is filled with collodion.

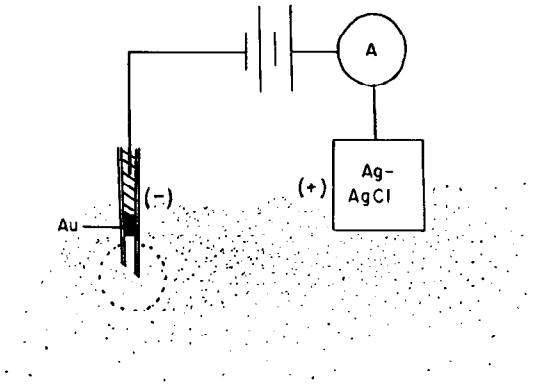

Fig.1. Diagram showing the essential requirements for amperometric determination of oxygen tension. "A" = ammeter. The cathode drawn here is a sketch of the tip of the electrode we use. The circle is to indicate that there is an area of diffusion in the conducting medium.

In the range of interest calibration curves are linear [3], and the oxygen current is not affected by stirring of the medium; the latter is presumably due to the very small diffusion layer which remains stagnant (6). The current measured is of the order of 2×10^{-13} Amps/mm Hg of oxygen tension which means that the O_2 usage is less than 1.5×10^{-6} μ l O_2/min. The response time from PO_2 of O to 150 mm Hg is less than one second (Fig. 2A). The steady state oxygen current is practically independent of the applied voltage in the range used, although there is usually [4] a transient surge of current when the potential is changed (Fig. 2B-upper trace). We have often taken advantage of this transient to determine whether the electrode is in or out of a cell since entry into the cell subtracts 60-100 mV from the applied potential. Of course, as discussed later, during an action potential the applied potentials return to near the original value.

Evidence has previously been provided that the electrode gives an absolute value for PO_2 (7, 8) and does not obstruct blood flow when used in vivo (19).

Recently, Dr. Nair, my associate, has succeeded in making a double-barrelled electrode which measures 2-4 μ at the bevel. The extra barrel

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[3] Above about 350 mm Hg some electrodes show a slightly decreasing slope.

[4] Some of our earlier electrodes (unbevelled) did not show this surge and responded very slowly to a change in applied potential, yet had a rapid response to a step change in PO_2.

is usually filled with KCl[5], and is used to simultaneously measure the cell membrane potential (9). The two circuits are electrically isolated so that interaction is minimal (Fig. 2B), and with some electrodes absent.

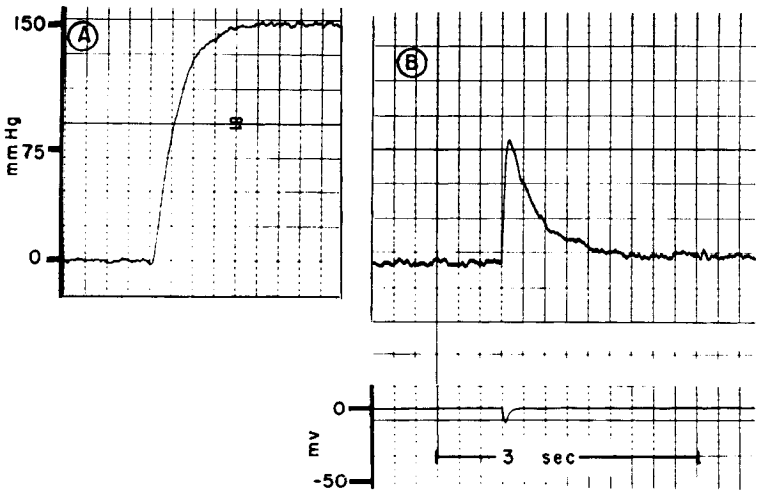

Fig.2. A. Response (slightly damped electronically) of the oxygen electrode to a sudden change in PO_2 of the medium from 0 to 150 mm Hg. B. Response (slightly damped) of the double-barrelled electrode to a step increase in the voltage (from 0.6 to 0.7 V) applied to the oxygen electrode (upper trace). The complete (or nearly complete) return to the original oxygen current level varied with the electrode and the recording conditions but usually required about 1 minute. A small "interaction" artifact was occasionally seen in the voltage circuit (lower trace). Chart speed same in both "A" and "B".

Measurements have been made in the cat gracilis, a white muscle (22), the soleus, a red muscle, and in the beating heart in situ of the cat (23). In the guinea pig we measured cell PO_2 in the gracilis muscle only. Readings were usually taken for several minutes in each location in the muscle. When the PO_2 varied with time the mean PO_2 was determined by planimetrically measuring the area under the trace.

Preparation of the animals was essentially similar in all of our studies. They were anesthetized with urethane and barbiturate, given intraperitoneally; tracheotomized; and kept warm by means of a heating pad. In the studies on the cat, arterial pressure was monitored and arterial blood PO_2 and pH occasionally measured.

For the studies on skeletal muscle the animals breathed spontaneously, usually air, but occasionally oxygen or nitrogen with and without carbon

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[5]The extra barrel has also been used to inject dye for more precise localization of the tip.

dioxide. The nitrogen (or helium) was given to check the zero level of the electrode. More often, however, the blood supply to the muscle was interrupted for this purpose. Additional calibration checks were done in the well made of the skin flaps over the muscle (Fig. 3). The well solution also served to keep moist the cleared area over the muscle.

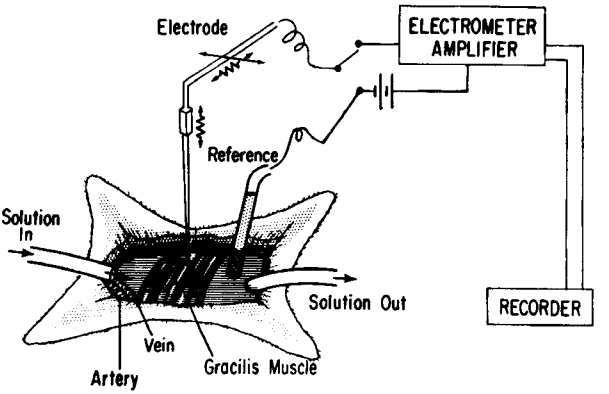

Fig.3. Sketch of the basic experimental set-up.

Under microscopic observation the electrode was lowered into the muscle, taking care to avoid visible blood vessels (> about 40μ). In the guinea pig the resting membrane potential was first measured with the electrode connected to a voltmeter, then the circuits were switched and the PO_2 determined. In later studies we abandoned this procedure because, a) we could not be certain that the tip remained in the cell; b) the tip, in random penetrations, was in a cell 70-90% of the time; and c) it is difficult to penetrate blood vessels (Brian Duling - personal communication) which was what we wanted to avoid. Furthermore, we have lately found with the double-barrelled electrode that when extraneous electrical noise can be eliminated, the oxygen current trace alone can be used to give an indication of cell penetration. An example is shown in Figure 4.

The experimental set up for the cat heart preparation is shown in Figure 5. One of the principle modifications is the rigidly-mounted suction device used to partially stabilize the ventricular wall. The device also contained a small well filled with warmed saline which was used to check the electrode calibration between penetrations. The other major modification consisted of mounting the electrodes (single and double) on a coil of tungsten wire (24) to allow them to "float" in the heart wall. The respiratory pump delivered moistened room air, or, occasionally, oxygen.

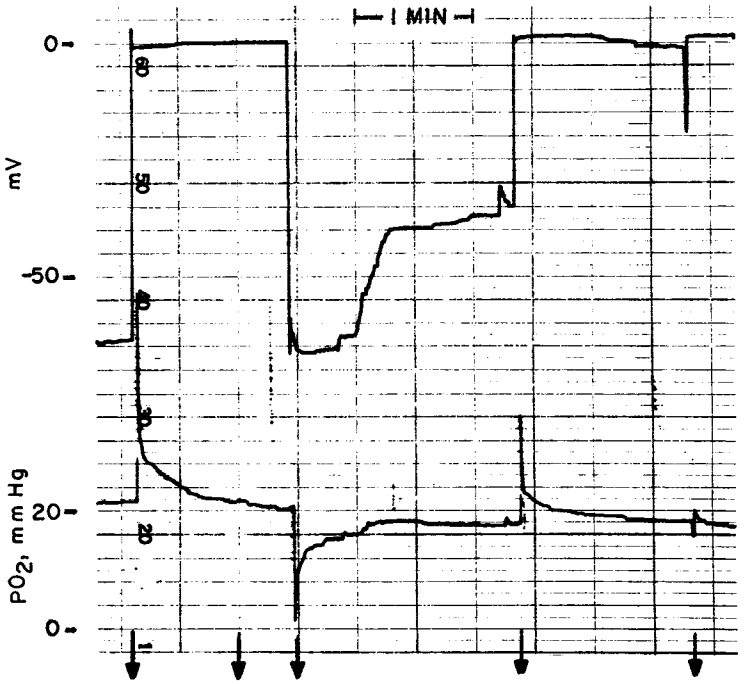

Fig.4. Simultaneous recording with a double-barrelled electrode of the resting cell membrane potential (upper trace) and the oxygen current (lower trace) during stepwise penetrations (arrows) into the cat soleus muscle. The penetrations were made without reference to the depth scale on the micrometer nor to the recorder.

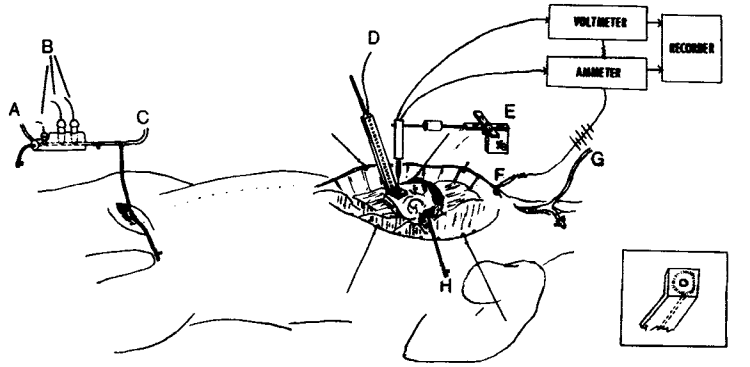

Fig.5. Sketch of in situ cat heart preparation showing electronic circuitry for the double-barrelled electrode, micromanipulator (E), rigid suction device (D), arterial blood sampling system (A,B,C). Insert at lower right is an enlarged sketch of the foot of the suction device.

Results and Discussion

A typical record from the guinea pig gracilis muscle (19) is illustrated in Figure 6. The PO_2 usually fluctuated with time, sometimes irregularly, but most often at a frequency of one or two per minute.

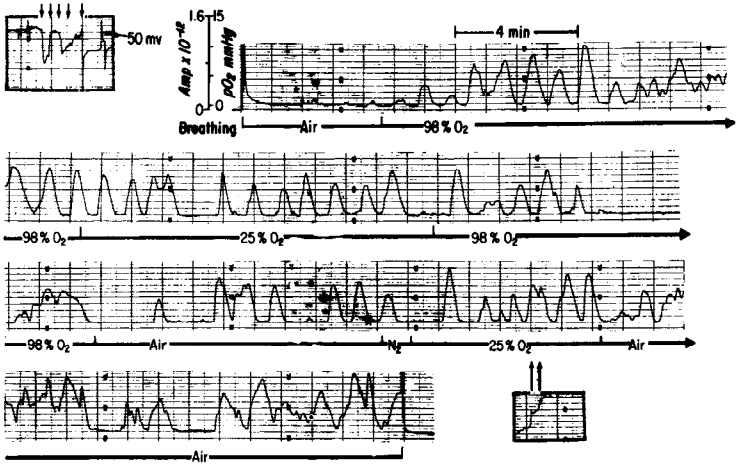

Fig.6. Typical continuous recording of intracellular PO_2 obtained from the guinea pig gracilis muscle. Membrane potentials obtained just prior to the PO_2 measurements are shown at upper left, and upon withdrawal, at lower right. Cell PO_2 (mean 4.7 mm Hg) was independent of the inspired gas. (Previously in Circulation Res. 21: 251-261, 1967).

Intracellular PO_2 varied considerably from one location to another as well as from one animal to another. A frequency distribution of the 184 measurements obtained from 26 guinea pigs is shown in Figure 7. The vast majority of the cells had a PO_2 less than 5 mm Hg. Even when the animal breathed pure O_2 for several minutes (or 98% O_2 - 2% CO_2) there was no increase in PO_2 in most of the 89 cells sampled. This result suggests that the arterial blood was already well-saturated with oxygen during air-breathing and, further, that blood flow is regulated in this muscle to maintain a low level of tissue PO_2.

Cat gracilis muscle gave nearly identical results (22). However, the inter-individual variation was somewhat greater, being from 1.4 to 18 mm Hg. This wide variation was not related to the level of the arterial pressure, pH or PO_2. The mean value of all the locations sampled was about 6 mm Hg. When the animal breathed pure oxygen there was a small but significant increase in tissue PO_2 and a significant decrease when 95% O_2 - 5% CO_2 was breathed. The latter result we assumed to be due to the dominance of the central vasoconstrictor effect of CO_2.

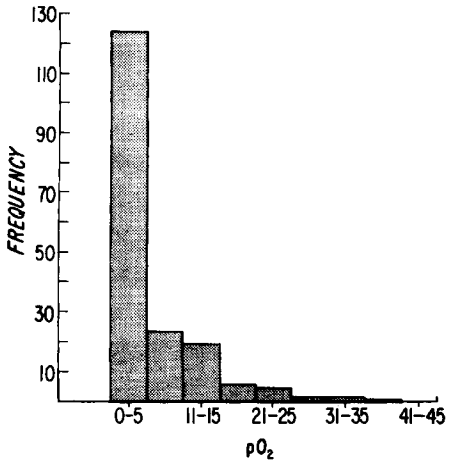

Fig.7. Frequency distribution of the cell PO_2 values found in the gracilis muscle of the guinea pig.

The mean value we found for cell PO_2 of 4-6 mm Hg in gracilis muscle is in the range of that calculated by others who used other methods and usually other muscle groups (19, 22). It is considerably lower than the mean of 38 mm Hg Kunze (15) found when he used a somewhat larger, bare-wire cathode in human forearm muscle. However, the use of this type of electrode for quantitative measurements in tissue has been criticized[6] (3).

The shape of the frequency distribution curve for the gracilis muscle suggested that it might characterize a typical capillary domain, i.e., the cylinder of tissue which might be supplied with O_2 by a single capillary. Indeed, this seems to be true (22). In the calculations of the radius of the domain we assumed that the highest value we obtained of 45 mm Hg represented the PO_2 just outside the capillary, and that PO_2 was 0 at the perimeter. Using established constants and several simplifying assumptions (22) the radius of the capillary domain was calculated to be about 100μ [7], which converts to about 25 open capillaries per mm^2. Considering the total number of available capillaries in muscle the ratio of open to closed vessels would be about 1:15. Reports of this ratio by investigators using other methods range from 20:1 down to 1:1 (see 22). Part of the disagreement may be due to the type of muscle studied, since the capillary domain in red muscle may be quite different.

[6] There are theoretical reasons to expect that a bare-ended cathode should perform satisfactorily provided that the exposed metal surface is very small. In our laboratory we have attempted to compare the values obtained with our electrode and the bare-ended type, but without success due to the erratic performance of the latter.

[7] My thanks to Mr. Donald Buerk who made the calculations.

Recently completed studies on the cat soleus muscle (unpublished) have revealed some rather fundamental differences from the gracilis results. Experimental results from 21 cats show that fluctuations in tissue PO_2 occur less frequently and they do not often reach 0 at the nadir of the trace as was seen in gracilis muscle. Individual values ranged from 0 to 65 mm Hg with a mean of 19.7. The shape of the frequency distribution also differs from the gracilis curves (Fig. 8).

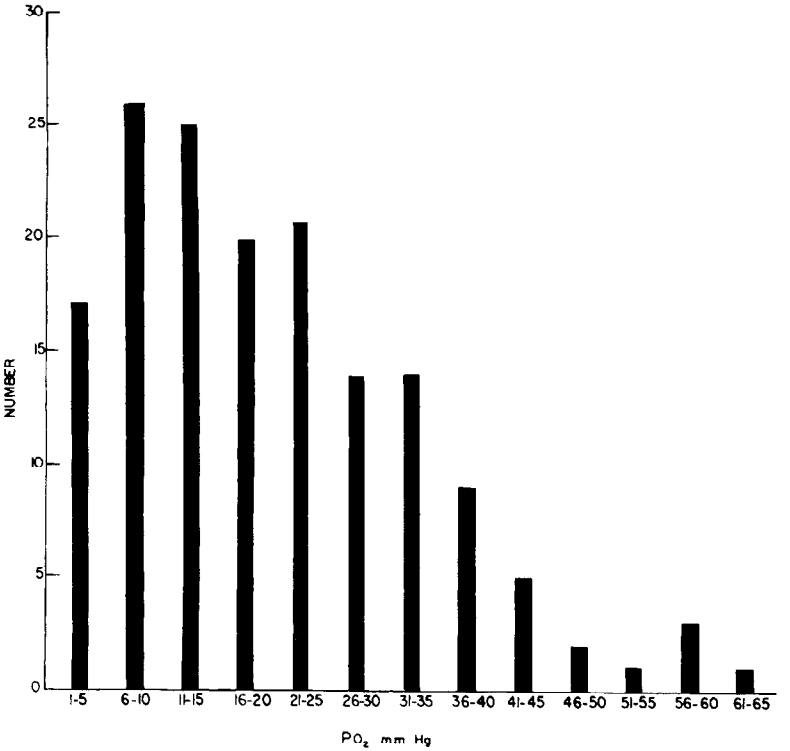

Fig.8. Frequency distribution of individual values for tissue PO_2 in the cat soleus muscle.

Finally, breathing pure O_2 usually resulted in a marked increase in tissue PO_2 (Fig. 9). In 27 trials the tissue PO_2 rose from a control of 18.9 ± 1.8 (S.E.) to 30.9 ± 4.6 mm Hg during O_2 breathing.

Some attempts were made to calculate the capillary domain in the soleus muscle, but the shape of the frequency distribution has discouraged precise calculations. It is only safe to say that it must be considerably smaller than in the gracilis.

In preliminary experiments on the cat gastrocnemius muscle (mixed red and white) the results have been more variable and appear to be

intermediate between the two extremes. Almost certainly, however, the mean PO_2 is significantly higher than in the gracilis.

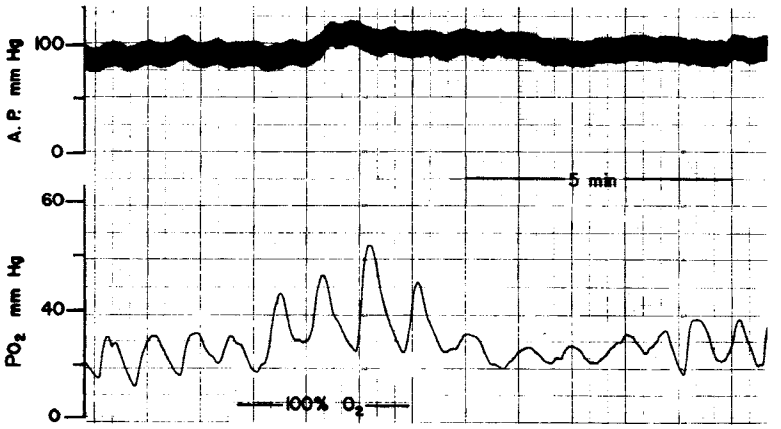

Fig.9. Typical rise in tissue PO_2 in the cat soleus muscle during pure O_2 breathing.

In the light of the results from the gracilis and the calculation of overall mean muscle PO_2 by others (see 19, 22) the markedly higher values for tissue PO_2 in the soleus are not easy to explain. According to Hudlická (12) blood flow in the soleus is unusually high and presumably many more capillaries are open than in the gastrocnemius. On the other hand, the O_2 consumption of the soleus was also very high, in her experiments, so that the ratio of the two was about the same as in the gastrocnemius which had a blood flow of only 10% of that in the soleus. It might reasonably be argued that the higher tissue PO_2 values in soleus are due to the increased possibility that the electrode tip was in a capillary. We think this is not the explanation, partly because of the reasons previously given; and also because in subsequent experiments where we were quite certain that the tip was in a cell, including some experiments using the double-barrelled electrode, the values cluster around a mean of about 20 mm Hg. In contrast, values for the gastrocnemius in the same animal were often zero, sometimes over a rather large area. The explanation may simply be that the soleus is not at all representative, and that the mass of the limb muscles more nearly resemble the gracilis, but the results to date on the gastrocnemius do not support this conclusion.

In any event, if O_2 availability limits O_2 consumption in the soleus the critical PO_2 must be much higher than reported for mitochondrial respiration (13). Even if the in vivo critical PO_2 is as high as 10 mm Hg as reported by Fabel (5) only about one-fourth of the cells would seem to be involved.

Recently, studies on the PO_2 in the beating cat heart have been

completed. In the heart there was little difficulty in deciding whether or not the electrode tip was in a cell after we learned the proper recording conditions. The effect of the intracellular action potential was clearly apparent on the oxygen current trace. Figure 10 shows typical recordings from the double-barrelled electrode. The possibility that these deflections were movement artifacts was considered but rejected for several reasons. Among these are, 1) the deflections obtained with a similar electrode closely resemble the traces often seen in the isolated, isometrically-contracting, almost immobilized, cat papillary muscle(Fig. 11); 2) occasionally, when the electrode was inserted to a new, deeper location, presumably extracellular, the "artifact" almost disappeared and its configuration was different; and 3) electrical activity preceded mechanical movement in the papillary muscle (Fig. 12).

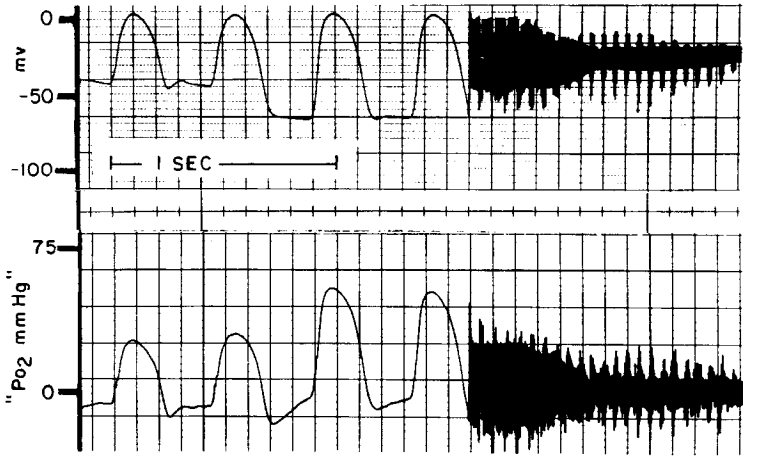

Fig.10. Slightly damped intracellular action potentials (upper trace) seen in the intact heart, and their influence on the O_2 current (lower trace) obtained with a double-barrelled electrode. At right a slow chart speed was used. (Submitted to Science).

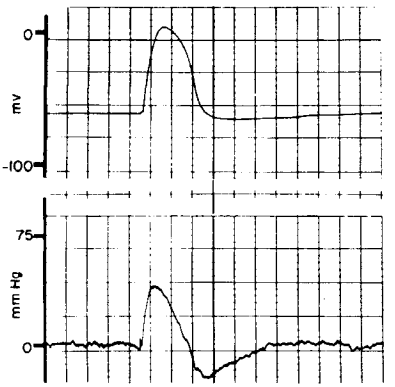

Fig.11.Slightly damped records of the intracellular action potential (upper trace) and the O_2 current obtained with a double-barrelled electrode in the isolated cat papillary muscle (at 26°C) stimulated at a rate which maintained core PO_2 near zero. Chart speed = 4 blocks per second.

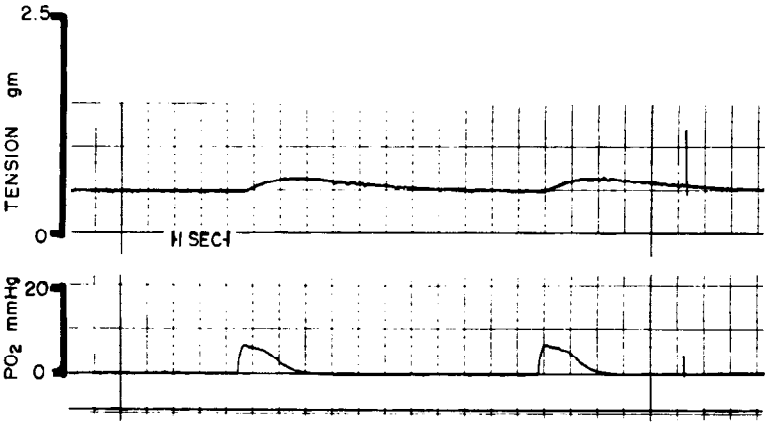

Fig.12. Records of isometric tension (upper trace) and the effect of the action potential on "core PO_2" obtained from a weakly contracting, isolated cat papillary muscle during a prolonged exposure to N_2.

The deflection in the O_2 current is probably the resultant of rapid, nearly simultaneous changes in both the applied potential and the PO_2 during the contraction. In studies on the isolated cat papillary muscle we often observed a sharp fall in PO_2 during the contraction (Fig. 13).

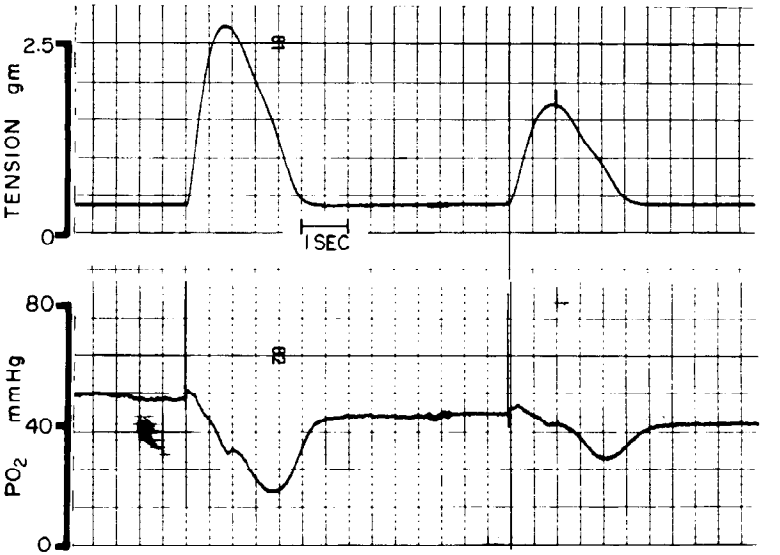

Fig.13. Records from a preparation similar to that in Fig.12, but the muscle has adequate oxygenation. Note, on the O_2 current trace, the "blips" after the stimulus artifact which we believe to be the effect of the intracellular action potential.

The reason no fall was seen in the in situ heart is most likely due to the close proximity of a "generous" O_2 supply, and to the electrical characteristics of the electrode used (see Footnote 4). Since, in this study, our main interest was in the mean level of cell PO_2, the rapid deflections, after identification were electronically damped, and the PO_2 read ($\pm$ 1-2 mm Hg) from the almost smooth trace (not shown).

The cell PO_2, measured in both the right and left ventricular wall was seen to vary somewhat with time as in skeletal muscle, but almost never did we see rhythmic fluctuations. The values (obtained from 8 cats) ranged from 0 to 31 mm Hg with a mean of 6.9 in each ventricle. In both ventricles the cell PO_2 was significantly higher in the outer 1 mm than in deeper layers, as is apparent in the frequency distribution (Fig. 14). Cell PO_2 in the outer layers averaged about 10 mm Hg and the inner layers approximately 5. This difference may be due to a higher blood flow in the superficial layers as found in the dog heart (14).

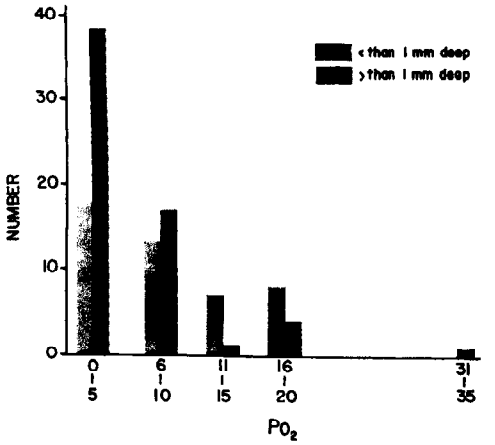

Fig.14. Frequency distribution of cell PO_2 in the in situ, beating heart of the cat. (Submitted to Science.)

It may also be due to a loss of O_2 from the arterial blood vessels as they course inward from the surface. We have reported that even large blood vessels leak O_2 (19) and Duling and Berne (4) found significant O_2 gradients along relatively large arterial vessels in the hamster cheek pouch.

The results from the cat heart suggest that coronary blood flow is regulated at a near minimal level. This is not a surprising finding, however, since signs of heart muscle dysfunction begin very soon after an occlusion of a coronary artery.

Conclusion

Overall, these results indicate that the mean tissue PO_2 in muscle is relatively low, but that there is considerable variation. A few measurements are not sufficient to describe most tissues. The highest

values were seen in the soleus, and one might be tempted to relate this finding to the continuous need for oxygen in this more or less continuously active muscle. However, the same can be said for the heart, in which many low values were found.

These results, reinforce the conclusion that the PO_2 in the venous outflow is much higher than tissue PO_2. If I interpret Dr. Hudlická's experiments correctly (12) venous blood from the gastrocnemius and soleus were both about 50% saturated with O_2. From the dissociation curve of cat blood (11) this corresponds to a PO_2 close to 40 mm Hg. In a few experiments we have measured PO_2 in the venous blood flow from the soleus and found it to be in this range. Most of this venous-blood-tissue difference may be due to A-V shunts or simply a lack of equilibrium between blood and tissue. However, some of this difference must be due to counter-current exchange of O_2 between arterial and venous vessels.

The results have some bearing on the questions of the possible regulation of local blood flow according to the demand for oxygen (10). In white muscle the regulation may be more effective than in red muscle. Certainly, the set-level appears to be different in the two types of muscle. Also, the sometimes large difference in PO_2 from one population of cells to another in the same muscle seems difficult to explain in terms of the oxygen hypothesis, although there is evidence that O_2 exerts at least part of its constrictor influence on upstream vessels so that a relatively large mass of muscle might be affected (22).

As to the hypothesis that O_2 availability may limit O_2 consumption I can only say that it remains a possibility. Experiments in progress are aimed at answering this question and, hopefully, some others that have been raised here.

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BEHAVIOR AND CARDIOVASCULAR FUNCTION*

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It has long been suspected that behavioral factors might play a role in the pathogenesis of cardiovascular disease. This hypothesis is based on the clinical observation that a great variety of psychological stimuli may provoke a marked rise in mean arterial blood pressure. Many events of normal daily life, physical activity and certain kinds of intellectual activity, may cause elevations of arterial blood pressure (13). Usually these elevations of blood pressure subside quickly but in some individuals they may not. Although the precise sequence of events is unknown, human subjects with frequent episodes of hypertension from any cause have a high likelihood of developing permanent hypertension and all its serious sequelae (5).

All living, conscious subjects perceive a great variety of stimuli from the environment and from within which activate somatomotor, visceral, and endocrine adaptive responses. The efferent responses to many stimuli are highly stereotyped and predictable even on the first encounter. For example, exposures to hypoxia, to hemorrhage, to heavy exercise, or to extremes of environmental temperature provoke adaptive responses which are highly predictable. The magnitude of mechanical, chemical, and neural events forces each subject into common patterns of response. However, the behavioral physiologist is most intrigued by the study of adaptive mechanisms that evolve gradually over time as stimuli are presented repeatedly. Eventually, after repeated exposures to the same afferent stimuli, most subjects develop complex behavior patterns that tend to be repeated whenever the same stimuli are presented. For example, if a monkey occasionally receives noxious stimuli in the presence of a light, and lever-pressing responses turn off the light, the animal will tend to respond by pressing the lever whenever the light is on (12). Thus, complex patterns of responding in the presence of a light can be maintained over long periods of time even though noxious stimuli are only infrequently delivered. Such schedules of intermittent reinforcement that exert persistent control over somatomotor activity also exert predictable and persistent effects upon the cardiovascular system (2,9). Although the response of a normal, healthy subject to severe hemorrhage is predictable on the first occasion, his response to a tone or a set of lights will depend on what happened during previous presentations of the same stimuli.

The subject's emotional response to the hemorrhage or to the tone and lights need not concern us in design of our experiments. If a subject responds to behavioral stimuli with prolonged, severe elevations of arterial blood pressure we may ask what sequence of adaptive responses led to this pattern of behavior and we need not ask how he feels about

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*Taken from the introductory remarks given at the session on Blood Pressure II at the 1971 Federation Meetings.

the situation that provokes hypertension. If a behavioral experiment is properly designed, eventually a subject's response will be predictable on each occasion regardless of his emotional state.

Some of the best known experiments in which behavioral stimuli altered cardiovascular and renal function were presented by Homer Smith and published in the 1939-40 edition of the Harvey Lecture series (14). Figure 6 in that presentation depicts urine volume, systemic arterial blood pressure, heart rate, and renal clearances of inulin and diodrast before, during, and after an episode that alarmed the experimental subject. Heart rate and blood pressure rose and renal blood flow decreased during the alarming episode. Glomerular filtration rate and urine volume did not change during those observations.

Figure 7 in the same presentation shows a similar set of observations on another experimental subject. Again, during an alarming episode, renal blood flow, glomerular filtration and urine volume all decreased and persisted at low values even after the subject had been reassured.

The most complete studies of cardiovascular changes in human subjects during behavioral experiments have been reported by Brod and his collaborators (3). They studied subjects with normal blood pressure and patients with arterial hypertension. Blood pressure was measured directly in the brachial artery, cardiac output was measured by the dye dilution technique, forearm blood flow was measured by venous occlusion plethysmography and renal blood flow was estimated from the clearance of PAH. After control measurements had been made, the subjects were asked to do mental arithmetic. A metronome was set clicking once a second and each subject was asked to subtract a two figure number, like 17, successively starting with a four figure number, like 1194, giving his answers once every two seconds. During a four minute period of calculations, each subject was repeatedly urged to keep up to the pace of the metronome. During the stimulus period, systolic, diastolic, and mean blood pressures rose in parallel fashion. The rise in blood pressure was almost always due to a rise in cardiac output and total peripheral vascular resistance changed very little. However, forearm blood flow increased markedly while other vascular beds had increased resistance. For example, renal clearance of PAH decreased slightly in the presence of increased arterial blood pressure indicating that renal vascular resistance was probably increased.

In all normotensive subjects, mean blood pressure rose from an average of 100 mm Hg to an average of 122 mm Hg. Cardiac output increased an average of 25% and forearm blood flow increased to more than twice the control values. After the stimulus period, blood pressure usually fell to control levels within five minutes although some of the normotensive subjects maintained an increased blood pressure for longer than 10 minutes.

In hypertensive subjects, mean blood pressure rose from an average of 142 mm Hg to an average of 171 mm Hg. Increased cardiac output was less consistent than in normotensive subjects and clearance of PAH

was significantly decreased. After the stimulus period, increased blood pressure persisted longer in the hypertensive than in the normotensive subjects. Instead of returning to control levels within five minutes, blood pressure in hypertensive subjects remained elevated for ten minutes or more. Individual differences in response may have indicated a relative likelihood of developing sustained hypertension or complications of sustained hypertension in each subject but no long-term studies were reported.

Other reports suggest that more common stimuli may also provoke marked changes in cardiovascular function. Ulrych (16) studied cardiac output and arterial blood pressure in a group of normotensive and hypertensive subjects performing mental arithmetic under the same conditions described by Brod and his collaborators. Ulrych also reported elevations of mean arterial blood pressure, heart rate and cardiac output above resting control values. In addition, he found these same subjects responded in a similar way during quiet conversation. These results imply that common, daily, interpersonal activities may be associated with elevations of blood pressure above resting control values.

Perhaps the most interesting event that affects levels of systemic arterial blood pressure is the casual measurement of blood pressure in a physician's office. Under these circumstances, many individuals have marked elevations of arterial blood pressure when examined in a routine way. Then, after a period of quiet rest, arterial blood pressure often declines to normal values. If arterial blood pressure of an individual is frequently elevated above normal levels but occasionally returns to normal, he is said to have "labile hypertension."

Frohlich and his collaborators (6) performed hemodynamic studies in patients with labile uncomplicated hypertension as well as in patients with sustained hypertension and in normal subjects. Patients with labile and uncomplicated hypertension (Class I) had modest elevations of systolic and diastolic blood pressure and modest elevations of heart rate compared to normal subjects. The elevated blood pressures of patients in Class I were due almost entirely to elevations of cardiac output. Total peripheral resistance was the same as in normal subjects and much less than in patients with sustained hypertension.

Sokolow and his collaborators (15) have attempted to quantitate the phenomenon of labile hypertension by continual measurements of blood pressure over long periods of time. By means of portable blood pressure recording units, they obtained measurements of blood pressure in hypertensive subjects repeatedly throughout the day and compared average daily levels with those measured in a physician's examining room. Average systolic and diastolic blood pressures during the day averaged 10 to 20 mm Hg lower than the values obtained by casual checks made as part of a physical examination.

The observation that the circumstances under which blood pressure is measured affect the values obtained has great clinical importance. In 1944, Paul D. White and his collaborators (11) published the results of a retrospective study relating the occurence of transient hypertension

to cardiovascular disease in a large group of U. S. Army officers. These men had yearly physical examinations and were followed closely for many years. Below 30 years of age, about 6% of all subjects examined had transient elevations of blood pressure during physical examinations that returned to normal levels during subsequent examinations. With increasing age, frequency of transient hypertension increased and at age 50 years the frequency of transient hypertension was 18%. Each year, some of the individuals examined had hypertension that persisted during all subsequent examinations. The incidence of sustained hypertension was noted to be four or five times greater in those individuals who previously had transient hypertension. The incidence of death due to cardiovascular and renal diseases was more than twice as great in men who previously had transient hypertension and men with labile hypertension had an increased incidence of death from all causes.

These data suggest that labile hypertension should be treated but they give no insight to pathogenesis of sustained hypertension. Apparently the simple behavioral procedure of measuring an individual's blood pressure can elicit a hypertensive response in a prehypertensive subject. However, these data do not prove that behavioral events cause sustained hypertension. They merely indicate that behavioral procedures may be useful in the early diagnosis of hypertensive cardiovascular disease. Still, at the present time, early diagnosis of cardiovascular disease is a worthwhile objective and I urge the refinement of behavioral techniques to this end.

Even if behavioral events do play an important role in the pathogenesis of sustained arterial hypertension, it seems unlikely that we will have much success treating hypertension by altering the life style of susceptible individuals. However, we might be successful in suppressing hypertensive responses to behavioral events or to any other pathophysiologic stimuli provoking hypertension. The question is where to interfere in the train of events leading to sustained hypertension.

In recent years, Dr. A. C. Guyton and his collaborators have presented theoretical and experimental evidence supporting the hypothesis that sustained hypertension occurs whenever perfusion of peripheral tissues exceeds metabolic demands and renal excretion of fluid and electrolytes fails to reduce blood volume and restore cardiac output to normal values (4, 8). Recently, these investigators have presented experimental data obtained from anephric human subjects that supports their hypothesis (4). Three anephric patients maintained by frequent hemodialysis were allowed to gain extracellular fluid and increase their body weights. As body weight increased, cardiac output and mean arterial blood pressure increased about 25% over a period of several days. Thereafter, peripheral vascular resistance increased by 20%, arterial pressure rose even higher to 137% of control values and cardiac output returned towards previous levels. Thus, arterial blood pressure increased in response to overhydration, initially as the result of elevated cardiac output and later as the result of increased peripheral vascular resistance. When body weight and fluid volumes were reduced by hemodialysis to control values, cardiac output fell below control values and

arterial blood pressure gradually returned to original levels. In normal subjects, any increase in cardiac output and arterial pressure increases renal excretion of fluid and electrolytes but in the anephric subjects, this compensatory mechanism was unavailable and systemic arterial hypertension persisted.

A theoretical analysis of cardiovascular and renal control mechanisms leads Guyton and his collaborators to the conclusion that sustained arterial hypertension occurs only when renal vascular resistance is increased (8). The precise relation between blood volume, cardiac output, and arterial blood pressure depends on the relations between renal vascular resistance, total peripheral resistance, and venous capacity. From theoretical and experimental considerations, it seems that therapeutic interventions directed against hypertension most likely to be successful should increase renal blood flow and renal excretion of fluid and electrolytes.

However, patients with labile hypertension have been reported to have high rates of renal blood flow (10) and intravenous infusions of saline cause a brisk diuresis in hypertensive patients (1, 7). Typical data have been reported by Kioschos and his collaborators (10). These investigators measured renal blood flow in unanesthetized human subjects by injecting indocyanine green dye into one renal artery and measuring the concentration of indicator dye in renal venous blood from the same kidney. Characteristic indicator dilution curves were obtained that could be used to calculate absolute rates of renal blood flow. The highest values for renal blood flow were obtained in patients with essential hypertension who had high cardiac outputs.

Although these data show that patients with essential hypertension had high rates of renal blood flow at rest in the supine position, these same patients may have had low rates of renal blood flow at other times. For example, it is possible that renal blood flow of hypertensive subjects in the upright posture falls below normal values especially if they are exposed to strong behavioral stimuli.

The hemodynamic response of hypertensive subjects to tilting at 50 degrees for five minutes has been studied by Frohlich and his collaborators (6). Cardiac output decreased and total peripheral vascular resistance increased in all subjects who were tilted but the greatest changes were observed in patients with labile and uncomplicated hypertension. Undoubtedly renal blood flow was also decreased during tilting at 50 degrees. However, no measurements of renal blood flow were reported in these experiments.

In summary, there is a great deal of experimental evidence indicating that cardiovascular and renal function are altered during various behavioral events. Although it is not proven that behavioral mechanisms are important in the pathogenesis of arterial hypertension, the data available are consistent with this hypothesis and behavioral events may provoke episodes of labile hypertension in susceptible individuals. Although patients with early uncomplicated hypertension may have elevated renal blood flow and a brisk diuresis in response to infusions of saline,

additional studies of hypertensive patients should be performed under realistic postural and behavioral circumstances.

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FOURTH INTERNATIONAL CONGRESS OF ELECTROMYOGRAPHY

This Congress sponsored by the International Federation of Societies for EEG and clinical neurophysiology will be held in Brussels, Belgium on the 12th to 15th of September 1971. Prof. John E. Desmedt will be the president of the congress which forms part of a quadriennal series (previous EMG congresses in Pavia, Copenhagen and Glasgow).

Five symposia will be held during the Brussels congress:

1. New concepts of the motor unit (chairmen: E. H. Lambert, Rochester and T. Tokizane, Tokyo).

2. Intracellular electromyography (Chairman: A. F. Huxley, London).

3. Pathological conduction in nerve fibres (Chairman: R. W. Gilliatt, London).

4. Electromyography in biochemical studies (Chairmen: S. Bouisset, Lille and B. Jonsson, Göteberg).

5. Human reflexes and motor mechanisms (I and II) (Chairmen: J. C. Eccles, Buffalo and K. E. Hagbarth, Uppsala).

Voluntary communications will also be programmed and a practical EMG course will be organized on September 16th. For further information, write to the secretary: Dr. K. Hainaut, Brain Research Unit, 115, boulevard de Waterloo - Brussels 1000, Belgium.

PERIPATOSIS - THE SCIENTIFIC DISEASE*

O. B. SERVER

All scientists suffer from an incurable disease. The virulence of the disease appears to be directly related to the status of the scientist afflicted. In all groups the disease is characterized by almost identical features, varying in intensity, but encompassing a readily recognizable syndrome.**

The disease usually develops in the young scientist immediately upon entrance into graduate school. In this early stage, the victim has a gradually increasing desire to travel. In the 25-28 age group this is usually manifested by urges in the Spring to attend meetings, especially those in warm climates and at considerable distance. The urge may become so strong that the victim is compelled to produce an abstract, occasionally requiring completely new research in order to obtain travel.

The disease becomes more virulent as the age of the victim increases. The postdoctoral student (Age 28-30) develops an exacerbation of symptoms. In this case the manifestations are usually an uncontrollable desire to spend one or two years away from his usual locus of activity. The strength of the impulse is determined by the availability of funds (usually Federal in nature) and the location of the fellowship. Those fellowships in Western Europe notably England, France and Scandanavia cause much greater responses in the patient than equal sums in Africa, India, or Japan. The disease at this particular stage often causes an outpouring of egocentric biography in order to justify subsequent behavior.

The malady again changes form as the patient ages and develops greater expertise in his chosen field. At age (30-35) the disease takes the form of sudden gatherings of groups of scientists for fixed periods of time and in certain seasons. Masses of scientists accumulate in Atlantic City in the Spring, on Cape Cod in the Summer and on various university campuses in the Fall. These meetings are characterized by rapid language communication between individual and small groups, by increased intake of food and drink, and by occasional, almost accidental gathering of the entire group at sporadic intervals. Such behavior may continue throughout scientific life. It has been noted that the gathering into large groups at these meetings, usually accompanied by individual outpourings of speech at frequent intervals is restricted to the younger group and that as the scientist ages he still feels the compulsion to gather with his peers but he no longer joins the group gatherings, and his intake of food and drink increases.

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* From an old manuscript discovered by J. H. U. Brown, Office of the Administrator, HSMHA, Rockville, Maryland.

** We have named the disease Peripatosis ("peripa" from the word peripatetic meaning to move about and "osis" for the ending signifying an abnormal or diseased condition).

At this point (age 35) the disease may take two divergent courses. On the one hand, the victim may continue as outlined above. If, however, he becomes a recognized leader in his field, a secondary phase of the disease develops. The victim finds himself under a compulsion to write and give speeches, to congregate in small groups, and to increase rate of movement. This may take the form of trips to foreign climates, to intercontinental peregrinations. The rate may increase dramatically from a few movements a year to drastic shifts in location. Often such movements are accompanied by goal seeking in the form of prizes. On occasion, the victim of the disease who has obtained his goals appears to have achieved satisfaction and the number of movements decreases rapidly.

Many of the victims in the "recognized" class appear to increase their movement by undertaking additional task oriented responsibilities which may consist of observing and reporting on the quality of activity of fellow victims. In this case, movement is usually to and from a specific mid-Eastern location with occasional sporadic movements toward locations where fellow scientists are congregated for daily living.

At all ages the disease has its most virulent manifestation toward movement across salt water. Here again the virulence is a function of distance with Europe and Japan being optimumly spaced.

The disease has other parameters than age and recognition. It has been clearly demonstrated that the extent of the compulsion and its satisfaction are conditioned by the availability of cash rewards from a central depository. Rewards allowing latitude in the degree of response after movement is completed in terms of food and drink available, distance of movement, etc., produce a greater compulsion than lesser rewards which restrict these responses.

In order to elucidate the syndrome more fully we have made studies of the case history of several victims of the disease.

Case I: I. M. A. Traveller, Ph.D., M.D., first developed symptoms of peripatosis in 1958 after medical school when he entered graduate school more than 1,000 miles away. The first serious twinges appeared when he decided to present the results of a medical school research program at FASEB in Atlantic City and the Department chairman provided $100 for the trip. The following summer the disease increased in intensity and he spent four months at Woods Hole in vain treatment. He returned to Woods Hole the following summer and then the FASEB in Chicago the next Spring. His Ph.D. was awarded in 1960 and the disease returned in full force. 1960-1962 were spent at the Karolinska Institute in Stockholm with small exacerbations of the disease resulting in trips to the continent. In 1962 he returned to a west coast university. Except for one minor trip to Japan the disease remained in remission until 1967. By this time Dr. Traveller had published several papers and a well-received book. The disease returned in full force. He became a consultant to NASA and a member of an NIH Study Section requiring many visits to Washington over the next few years. In 1969 he joined a team on a visit to Russia and in 1970 to Australia. The disease

now appears in full flower.

Case II: Dr. Gottago finished his degree (Ph. D.) in 1940. The disease was apparently absent at this time since he immediately accepted a faculty position in a nearby university. During his early academic years the disease occurred sporadically as demonstrated by applications for travel grants (usually rejected for lack of funds). Dr. Gottago trained several students and managed to conduct a considerable amount of research. The first overt symptoms appeared when he was appointed to several NSF committees and was then elected to NAS. With advent of the second World War he became a consultant to the Air Force of the Navy. His disease became more apparent as he made several trips on carriers. His experience qualified him for scientific liaison and he made regular trips to Europe at the Department of Defense expense. The disease was now developing to a full blown case.

By 1955, he was able to spend 6 months in the Himalayas studying high altitude physiology of the Sherpa and in 1957 he repeated the study in the Andes and in 1959 in Colorado. Speaking engagements came more and more often and by 1962 it was difficult to find him at his home university.

In 1964, a combination of a mild heart attack following a 24-hour flight from Australia and an admonition from the Dean regarding his travel, resulted in a marked remission of the disease.

Dr. Gottago now exhibits almost no symptoms. He is writing a text book on respiratory physiology and beyond wistful comments as colleagues return from far places he may be considered cured.

Case III: One of the most virulent cases on record was that of the cultural anthropologist, Dr. Ura Diggan. Dr. Diggan first developed the disease when he was forced to make a decision on where to conduct research for his thesis on the topic of the effect of slums or ghettos on the indigent population. After much deliberation he decided on the large cities of South America. Since little work had been done on the subject he had no difficulty in obtaining funds from NIMH and other sources.

He discovered early in the disease that he could obtain greater relief by placing his wife on the grant as an assistant and by taking several graduate students with him. There were occasional periods of apparent remission when only a single girl graduate student accompanied him.

The disease really had no serious remissions during a 20-year period. Dr. Diggan spent at least 6 months of every year in South America moving from city to city to demonstrate they were all alike in their sociological makeup. The only apparent decrease in intensity of the disease occurred with the lack of Federal Research funds in the late 1960's and encroaching old age.

The three case histories illustrate only facets of a universal disease. Our observations indicate that the disease is endemic in the scientific population. Almost without exception it appears in the young man, runs

a virulent course through middle years and may wane with increasing age. The infection varies widely in severity. A statistical analysis indicates that the severity is determined by several factors. Weighting of these are presented in Table 1. It is clear that the determining factor is grant support followed closely by scientific reputation.

TABLE 1

The severity of Peripatosis as a function of various ecological factors.

Factor	Severity of disease on a scale of 100
Travel funds	80
Scientific Reputation	60
Location (Harvard = 50, Podunk State = 0)	30
Increasing age past 60	40
Area of research	35
All	100

These two factors are so closely interrelated that it is difficult to separate them. Location may play an important part in enhancing the disease or in decreasing the virulence. It is interesting that the severity of the disease is conditioned by the area of research (linear accelerator physicists have a very low virulence of disease while anthropologists and biochemists appear to have very high infection levels.)

We attempted to duplicate these clinical observations in the laboratory. It became immediately apparent that only man suffers the disease with the possible exception of an aberrant form in the Lemming. Furthermore the disease affects the North American scientific population most strongly followed by Japan, Scandanavia, England, and central Europe. There are sporadic, almost non-virulent cases in Africa, the Middle East (except Israel), and India.

We therefore set up an experimental population of young American male scientists (YAMS).* They were divided into two groups of equal ecological input. One group was given a steady infusion of funds while the second group was denied access to funds. The results were apparent immediately. The funded groups developed the disease immediately as demonstrated by decreased work production, increased journal scanning for meeting notices and increased abstract preparation. The unfunded group continued high work output, and although there were signs of uneasiness, no overt symptoms developed. After one year, the two groups reversed. Immediately the cases appeared in the previously deprived group while the group which had received funds but was now restricted demonstrated a decrease in virulence accompanied by psycho-

*The disease does not occur as frequently in females especially those who are married, but we have noticed a marked increase in the last few years. Conversely it occurs more often in the married male as opposed to the single man.

pathic responses of sensory deprivation and frustration which were not apparent in the originally unfunded group which had never had the disease.

We have been unable to produce a satisfactory treatment for Peripatosis. The disease occurs when funds are available in the proper ecological environment. It is possible to alleviate the symptoms by restricting funds, decreasing meeting frequency, and restricting fellowships. It may not be a wise course of therapy to abolish funds altogether, especially in that group which has already developed the disease even in a mild form. However, it is wise to restrict funds in the group in which the most virulent disease is present to return them to a productive life.

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JOURNAL OF BIOMEDICAL MATERIALS AND ARTIFICIAL ORGANS

A new international quarterly journal called the Journal of Biomedical Materials and Artificial Organs will be published by Marcel Dekker Inc. of New York with the first issue being distributed in March of 1972. This journal will be directed toward basic scientists and clinicians interested in the development of materials for medical applications and the clinical utilization of these materials as for example in artificial kidneys, cardiovascular implants and heart valves, surgical materials, etc.

Manuscripts may be submitted to:

T. F. Yen, Ph.D., Editor
Journal of Biomedical Materials and Artificial Organs
University of Southern California
2025 Zonal Avenue
Los Angeles, California 90033

SENIOR PHYSIOLOGISTS

Belated replies to our inquiry addressed to
"younger" senior physiologists

Louis Nahum wrote to Gene Landis:

"Presently I am indulging in some cardiology practice and am Editor of Connecticut Medicine the scientific organ of the Connecticut State Medical Society. I give an occasional lecture and much advice to younger people. My advice to those who are approaching retirement is to develop a "second career" if they have not begun in its preparation long before this time."

R. W. Dougherty wrote Gene Landis from the National Animal Disease Laboratory, Ames, Iowa:

"I am continuing my research work on the physiology of digestion in the ruminant. Administrative duties, unfortunately, for the past few years have reduced my research activities. This is a Federal laboratory that was activated during the Fall of 1961. It employs about 475 people and is one of the best equipped laboratories of its kind in the world. In this laboratory I am head of the Physiopathology Section. We have six physiologists (besides myself). We have five technicians and have excellent equipment. Our research work includes physiological studies on normal animals. At other times we follow the physiopathological changes in animals suffering from experimentally induced infectious diseases as well as noninfectious diseases. In my position retirement is mandatory at age 70; I may retire in a year or so. I would be interested in continuing research and teaching activities after my retirement and would be free to move to another area."

Robert W. Lackey wrote Gene Landis from the Medical School at Dallas:

"I am continuing in a fairly active teaching role on a voluntary basis. This I enjoy very much. As a member of the original faculty of this school I am watching with great interest the beginning of a very ambitious program of expansion here."

Orville Walters wrote from Urbana to Hal Davis:

"Thank you for your letter representing the Committee on Senior Physiologists. I appreciate being so remembered. I returned March 1 from a first semester sabbatical leave spent in Taiwan and India. During that time, I gathered material from Chinese and Indian students for a study comparing the emotional disorders of Asian students with those of American students. I have one additional year before reaching the age of mandatory retirement in the University of Illinois. I resigned as Director of Health Services in 1968 after 10 years in that position, but continue as research professor in health science and lecturer in psychiatry."

Marie Hinrichs wrote Hi Essex:

"At 78, I'm working as consultant in Medicine at AMA Department of Health Education, and running a column in Harvest Years (monthly magazine) - also contribute newspaper articles on health subjects."

Edgar Poth wrote Hi Essex that he is continuing his scientific activities. His advice is, "Keep busy." He is organizing a Senior Study Group in his Medical School at Galveston.

Maurice Friedman wrote to Hi Essex from the Hotel des Trois Couronnes, Vevey, Switzerland:

"I was delighted to receive your letter of greetings, and so be reminded of my advancing years. To answer some of your questions: I am conducting rather careful and discriminating experiments on the effect of Swiss food on body weight (not entirely with the precision of the experiments of Harold Holck). Moreover I have been granted privileges at the nearby Nestle Library, where I have been reviewing the literature in connection with the myth that the intake of fat, and level of blood cholesterol are important factors in the genesis of coronary disease. Before starting to write any kind of a review on this subject, I intend to journey to Geneva in order to use the library of World Health Organization on matters regarding the accuracy of mortality statistics, the ratio of physicians to population, and the consumption of cigarettes in such advanced communities as Ceylon, Thailand and Formosa. If my concurrent experiments with Swiss food do not interfere, I hope to summarize my findings, if not publish them, before next Christmas. I realize that if I do publish anything on this subject, it might reflect rather adversely on one of the fundamentals of American medicine; namely, IF IT TASTES GOOD - SPIT IT OUT . . . .I put high priority on one phase of activity I started some years ago, namely the management of the financial affairs of some University professors, whose pensions and saving would not permit them to live comfortable otherwise. Although these persons are still in the United States, and their funds also are there, I find it possible, tho a bit more difficult, to continue this activity from my new home in Switzerland.... Some persons cannot retire and be happy. Indeed it is likely that some cannot retire and survive for long. Generally, however, most persons fear boredom in retirement. In most cases this fear is not justified, as shown by the subsequent histories of persons who harbored such fears. Even some persons who had never developed any hobbies prior to retirement, found that life was very full and satisfying when they stopped working. I am not joking when I say that one of the greatest problems of the retired man is that of learning how to get along with his wife. During his working years he may not have seen very much of her, and then all of a sudden, he is at home most of the time. As one insurance executive recently said, 'I married my wife for better or for worse but not for lunch."

Julius Sendroy wrote Bruce Dill:

"Currently, I am Science Advisor at the Naval Medical Research

Institute in Bethesda, and Special Scientific Assistant at the Research Division of the Bureau of Medicine and Surgery, Navy Department in Washington. I am soon to retire (June 30) from the first position, to take up full time work in the latter. I expect to stay at the Bureau until Fall, when I will transfer residence and activities to the West Coast, probably in California. There I hope to be able to continue work on a part-time basis as a research administrator, advisor, consultant, or laboratory worker in connection with a clinic, hospital, or university."

Al Behnke wrote to Bruce Dill:

"Since retirement I have been available for voluntary activity pertaining to National Welfare within my sphere of cognizance. Currently, the matter of a simple but nutritionally adequate diet for school children has been of concern to me. I believe the post-war nutritional observations may have some application. A second matter for consideration is that of establishing a modest Senior Fellowship (stipend of $2,500 - $5,000) for nominally retired physiologists who spend more than half of their time expediting research and advising younger investigators. I find myself currently with three hyperbaric chambers which I could 'maintain in operation' with modest support."

Max Kleiber wrote Bruce Dill from Davis:

"Now I am going to postpone other activities until I have written to you. It seems ridiculous for a retired professor to say he does not find the time for everything he likes to do. But there is a reason for this; advancing age increases the time required to accomplish a given task, and reduces the time available for work per day when one has to choose between a midday siesta and an afternoon fight against sleep at the desk. Coffee might help in emergencies but not as a continuous provider of effective working time. The Davis Tracer team has extended its research goals with refined techniques and operates a highly automatized respiration apparatus with so much electronic equipment that I am unable to diagnose sources of trouble as I used to do with the operation of our earlier apparatuses. My participation in the activity now is limited to occasional discussion of problems and results and even in these discussions I cannot keep up with the developments of biochemistry especially enzymology, sufficiently for any creative leadership in the pioneer phase of the experimental research. What I still can do and like to do is teaching, clarification of concepts, simplification of ideas, detection of inconsistencies in deductions and terminology. With this type of activity I feel that I can continue to be useful as a lecturer, a writer and as an occasional reviewer for editors, evaluating papers presented for publication. After my returning from Hawaii to California in the Fall 1963 I was rehired part time to teach my course on animal energetics which since 1961 had become a major part of "The Fire of Life" published by Wiley, New York. The course consisted of 36 lectures, two midterm and one final exam to nine upper division and twelve graduate students. In Spring, 1964 I started the translation of "The Fire of Life" into German, acting in the triple role of original author, translator, and typist. (The Polish translation by Piotr Poczopko appeared in 1968). In March, 1966, I flew back to Honolulu, this time for only

three weeks of Hawaiian interlude with a series of lectures in the Department of Nutrition. The topics were: The principles of energetics; metabolic rate as a function of body size and age; starvation and population; and, life against the degradation of energy. During the Fall semester of 1966 I taught a 25 lecture course on metabolism and energy utilization to eight students of veterinary medicine at Davis. As a visiting professor in the Department of Animal Science of the University of Minnesota at St. Paul I conducted a 24 lecture course on the relation of heat; work and energy; animal temperature regulation and metabolism; and, feed utilization; 18 students took the course for credit. From October to December, 1968 I was entrusted with leading a graduate seminar in Nutrition Science at the University of California at Berkeley with 22 students, 13 taking the seminar for credit, presenting reports for discussion on recent developments in the field of animal energetics. The topics discussed were starvation; hibernation; regulation of food intake; obesity; life and entropy; metabolic effects of gravity; usefulness and limitations of linear regression equations; temperature regulation; calorigenic effects of food intake; and, population and food supply. In my interjected "Words of Wisdom" special attention was given to errors in reasoning and terminology. On February 1, 1969 my wife and I flew to Fairbanks, Alaska where until the middle of May I was visiting professor of zoophysiology at the Institute of Arctic Biology. I presented a series of 15 two-hour lectures on bioenergetics to seven graduate students and nine faculty members. I started to grow a beard and enjoyed leisurely skiing to my office. Since my retirement rather more time than before was spent in shorter engagements, say less than a week at various places lecturing on various topics."

Jerzy Kaulberz wrote to Bruce Dill from Cracow, Poland. He is continuing his scientific activities on a reduced level. He is emeritus at the Medical Faculty of the University although continuing research on the physiology of digestion in collaboration with his successor. He continues as professor of physiology at the Higher School of Physical Education. He attended the Physiological Congress in Washington, and the World Congress of Sports Medicine in Mexico City at the time of the Olympic Games.