THE PHYSIOLOGIST

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Responsibilities of the Mentor to His Ph.D. Student in Physiology

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I am somewhat uncomfortable in this assignment and realize that perhaps few of my senior faculty colleagues will agree in whole or even in part with my assessments. Nevertheless, I shall claim some degree of seniority and presume to proceed. My credentials are nearly 40 years of teaching at the graduate level and having guided some 17 Ph.D., 13 M.S. awardees, and 4 postdoctoral fellows during that time period.

I will assume at the outset that we are discussing a worthy Ph.D. candidate: one who needs no defense on the basis of intellectual capacity, intrinsic creativity, or level of motivation to establish a professional career in physiology. Thus you recognize my elimination of most of the *mentor's* problems without having said anything.

A graduate student rarely arrives at the University fully prepared to articulate the course he wishes to follow in research, and certainly not the precise problem he expects to undertake. Rather, the new student generally comes because he is attracted to a career—research and/or teaching. He needs to learn fundamental principles of physiology, including accumulation of a responsible body of factual information. He must become acquainted with advanced instrumentation, and he must learn what other researchers have done and are currently doing. He must learn to think critically, first about his own approaches to a problem but also in evaluating the experiments and data of others. He must learn to think in terms of a *controlled experiment* and be able to detect violation of this crucial principle whenever and wherever it appears. He will find such violation in his textbooks and in both clinical and scientific journals, he will hear it from his fellow students, and unfortunately he will sometimes hear it from us, his senior faculty. He must learn to carefully weigh what he has come to consider as fact and weigh it against what is being proposed. He must maintain a mind open to new interpretations, new ideas, and new twists and yet not be misled by attractive hypotheses that he wants to believe because they solidify an already preformed bias. Perhaps this primary objective is to develop independence as a thinker, as a laboratory investigator, and as a scholar.

May I now assume I am talking directly to you who are graduate students and about to select your mentor? Most of the requirements to be imposed on you as a Ph.D. candidate will be accomplished within a lifetime, but many of the qualifications need to be accomplished during your first year in graduate school.

You will accomplish much reading, studying, discussing, arguing, assuming extravagant stands, learning to back away when you are wrong, sticking to your guns when you are right, and generally paying careful attention to your most important task during that first year, that of picking your mentor. This probably includes, of course, selecting your major field of specialization. You must select a faculty member who has impressed you as being completely and totally honest, one who is *really* excited about physiology and about his area of research, one who openly seeks critical evaluation of his ideas and his experimental data, one who is not overly concerned that every inquiring colleague is out to steal his stuff. You must assess the potential mentor's personality. Can you reasonably expect to live comfortably with him for the next three to four years, sometimes under extremely close and trying circumstances? Are his research areas and his personality flexible enough to share with you the excitement and reward of *success* as well as the utter scut work, the trial and error, the false starts, and the exhausting repetitiousness required to achieve it? Will he be willing to step aside, out of your way, when you are finally launched on the pathway to independence which you seek? Does he command the respect and admiration of *his* peers? Is his work quoted in the pertinent literature? Is he invited to present his ideas and his data before knowledgeable audiences? Is he asked to review papers submitted to the *American Journal of Physiology, Circulation Research,* and comparable journals in his field? Does he encourage his students to attend APS meetings, and does he introduce them to scientists whose papers they have read and critiqued in your journal clubs, seminars, and colloquies? Does he emphasize a need for you to formulate a hypothesis, collect data, and interpret them in time to meet the deadline for submission for the next APS meeting? Does he harangue you to quit stalling and get the experiments completed and written up, because if you do not, no one will be interested in hiring you? Does he insist that you present the data before a specific meeting because it is there that you will encounter the deepest concentration of experts in the field, where the most critical evaluation of your experiements can be achieved? In spite of the fact that he works you like a dog, do his ideas and his method of ideation excite you? Does he "turn you on" intellectually? Does he make you want to be as good as he is?

When you have found a faculty member about whom the answers to most of these questions seem to be yes, sit down and *think* about him for a long time. It is prob-

Presented by N.R. Alpert for W.C. Randall at Symposium on Career Opportunities: How to Get a Job in Physiology, FASEB Spring Meeting, 20 April 1982, New Orleans, LA.

ably the most important decision you will make once you have selected your graduate school.

I have tried to highlight some of the points of professional expertise that you need to think about—some of the measurable qualities you can place on a balance. But perhaps even more important are the things you *cannot* put on a balance. Is he a nice human being? Does he show real interest in your personal as well as your professional aspirations, your philosophy of life, your down periods, your home life? Almost all of us will let you do anything you wish with the last six hours of the day. If a mentor is worth his salt, he will keep close track of the first eighteen hours. But when one of the kids is really sick, is he there to help? Does he ever admit, especially when he is dressing you down for a technical or theoretical error, that he once "really goofed"? And then elaborate upon that goof so explicitly that you realize clearly that he is really helping you to learn an important lesson. The ideal mentor does this so effectively that you get the point and thoroughly understand the lack of malice behind his bitter criticism. In these thoroughly private sessions, you will come to appreciate the mentor's own self-esteem, his firm philosophic convictions, and his personal sense of intellectual security.

I believe that Ph.D. training absolutely *requires* careful attention to three separate stages of development in a young scientist. The *first* is through his need to acquire essential facts and understanding of fundamental physiology, ability to organize his thoughts and understanding in a defensible and convincing manner (which he does best in journal clubs, seminars, informal student-faculty or student-student discussions), and when he is simultaneously dipping into as many research areas as are possibly available to him. Toward the end of the first year he is ready to *select* his mentor. The *second* stage is spent in working, talking, thinking, dreaming, speculating, criticizing while being criticized, in evaluating his research area with his mentor. He has not yet selected his problem, but he is constantly sampling his ability to generate ideas, to appreciate his mentor's massage of these ideas, and to build a framework for his own plunge into the waters of original and creative research. You will recognize here the mileposts of the Ph.D. dissertation, original and creative research. It goes without saying that the research is original and creative *with the candidate,* not with the mentor. True doctoral credit research is never the result of the candidate's technical manipulation of the mentor's ideas and theories, but rather of his own.

You may be surprised that it does not take long to learn much of what the mentor has to teach in terms of hard facts. You will also know when the mentor is holding nothing back. He will want to make you as good or better than he is and to do so as quickly as possible. He advises you to the very best of his ability. He permits you to make mistakes, and he corrects you in each default. But he does not carp about it either. As student and mentor become a working team at the research bench, the student is continually asking questions. Early on, the mentor can probably answer most of them. But there will be some fascinating ones he *cannot* handle. Each time that happens the student gets a deep and thrilling satisfaction. *Cultivate* that excitement, Dr. Mentor, it's the most valuable medicine you've got. Sometimes the mentor goes out of his way to design a protocol that will provide answers to these questions, but mostly he encourages the student to do so. However, the mentor *never* sits down privately and designs a protocol for his own experiment, apart from the student. Rather, he encourages the student to bring him such a protocol for their mutual evaluation, and frequently enough the two will implement the experiment together. The mentor will insist that you write up these mutually accumulated data and present them as a joint effort.

Eventually you will come up with a question that is sufficiently exciting, comprehensive, deeply penetrating in concept that you both think is worthy of a doctoral dissertation. By this time you are thoroughly familiar with the laboratory, its instrumentation, its supply and equipment budgets, and its capacity to handle the costs of at least a two-year study. You go over each aspect of the question as critically as you can. Dr. Wiggers used to tell us, "Do a few experiments—even *before* you go to the library. You will get the thrill of discovery, and perhaps your approach will be sufficiently unique that you will learn something that previous investigators have missed." As the protocol takes form, if the initial library search is convincing and a few preliminary data appear to be exciting to you and to your immediate colleagues, you may be on your way. You have pride of ownership. It was your idea. You made it work. Surely your mentor evaluated the idea and the initial data as crucially as possible, and he suggested vitally important changes—perhaps even new directions. His grants also probably furnished the financial wherewithal. You will eventually owe his grant appropriate acknowledgement. But with advance of time and achievement, the experiments become more and more stamped with your standards of creativity and individuality. You become increasingly independent, and you begin to bring your questions and concerns to him periodically rather than day to day. And the mentor has sense enough to permit you to schedule the meetings.

You are now in the *third* and final stage of Ph.D. training. From here on, *you* find the literature citations, old and new. *You* bring papers and ideas to the mentor. You suggest new protocols and questions. He begins to ask you questions and about the most recent developments in your competitor's laboratory. He also begins to brag about your work, always going out of his way to credit you, even though you are not present. He does so with detectable pride and even a sense of accomplishment on his part, not for the research, but for your progress. You must concede him this, otherwise he does not have much to show for his effort. But really, your achievements and accomplishments will be the crowning joy and satisfaction of his career. He has already begun to think about where you can best go next after successful defense of your dissertation.

Yes, in these days you will almost certainly want a postdoctoral appointment. You need the advantages of standing on your own reputation and on your own newly acquired stature. You need to be in a milieu which will give you that opportunity as well as to protect you from the initial onslaughts of a cold competitive world. You need to have opportunity to teach without the overwhelming responsibilities of teaching. You need the op-

portunity to expand your ideational abilities and technical skills. You need to learn still more skills. You will want to become associated with still another group of experts and to learn how other authorities apply the scientific method. How do they do a controlled experiment? One of my chiefs told me early on that I must capitalize upon the energy of my youth, the fact that I did not recognize the limitations of the calendar and the clock. He told me flat out that I would make it in my first five years or I would not make it at all.

So now we come to the *"Good Ol' Boy"* syndrome. Some of you have been frustrated by it. You have told me, "I can't get access to the job opening because it was filled before it was advertised." I consider this concept as absolutely essential for every *successful* Ph.D. mentor. If your mentor does not know his peer scientists well enough to pick up the phone and call the one, two, or three scientists of your choice for a postdoctoral experience, you made a wrong choice some three years earlier. He should be so knowledgable and so respected himself that his peers will accept his recommendations. You must recognize that he can only make one or two really bad mistakes in such recommendations. I *will* hold it against him if he tells me you are something you are not. I will not give him a second chance, and he would not give me one. The mentor must be absolutely honest and totally "up front" in presenting your qualifications. But he must also know the best places for you to go, and he should have enough clout to give you at least a fighting chance to land an interview.

Times are tough, and jobs are hard to get—particularly the good ones. According to my scheme of things, a qualified mentor can properly train only one or two candidates at a time if he is to impart everything he has to offer. How the biochemists can manage 20 or 30 graduate students at a time is totally beyond my comprehension. I honestly do not think they can do a proper job under a regimen I consider essential.

Does the mentor's responsibility to the young scientist extend beyond the granting of the degree? Do his responsibilities continue with the Ph.D. into and through his postdoctoral post? The answer is a resounding yes to both questions. I have never found this to be a problem, because I have always become such a close personal friend of my young colleagues that I have maintained close personal contacts extending throughout our lifetimes. Of course I am interested in his total future—he took a vitally choice part of me with him when he left my laboratory.

Career Opportunities in Physiology Committee

Previously published in *The Physiologist:*

Graduate Training in Physiology and Its Relationship to Career Opportunities. Thomas M. Saba. 23(6): 9–12, 1980

Careers for Physiologists in Government. Donald M. MacCanon. 24(3): 48–49, 1981

Symposium: Career Opportunities in Physiology. 24(5): 15–21, 1981

Introduction. Walter C. Randall

Do We Really Need More Physiologists? Theodore Cooper.

Ph.D's in Clinical Departments. Alfred P. Fishman and Paul Jolly.

An Academic Career in a Basic Medical Science Department of Physiology. Thomas M. Saba. 24(6): 16–20, 1981

The Role of the Ph.D. Physiologist in a Clinical Department. James E. Blankenship. 24(6): 20–22, 1981

A Division of Research in an Academic Clinical Department. Richard J. Traystman. 25(2): 63–64, 1982

A Physiology Chairman's Objectives in Selecting Young Faculty Members: Criteria in a Smaller Medical School Setting. James P. Filkins. 25(2): 65–67, 1982

How to Get a Job in Physiology

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Choosing a job might not be as crucial as those two other biggies—selecting the right spouse and picking the right profession—but it's close. In most cases, these choices are made on the basis of personal biases, but better selections can probably be made by being maximally informed of all possible options.

Currently the best source of information about jobs in physiology is the Committee on Career Opportunities in Physiology, a standing committee of the American Physiological Society. This Committee was established for the purposes of evaluating the training processes of physiologists and developing recommendations that will assure a proper balance between this supply and demand. The results of this Committee's studies have been published in a series of articles appearing in *The Physiologist* and presented in symposia at the Society's national meetings. Another source of relevant information, also published in *The Physiologist,* is the analysis of questionnaires prepared by the Association of Chairmen of Departments of Physiology. These analyses deal with departmental staff, training, and salaries. A more general source of information on the subject is in the Scientific Manpower Commission's publication *Manpower Comments,* which is published monthly and may be purchased from the Commission's Editorial Office, 1776 Massachusetts Ave., NW, Washington, DC 20036. Also supplying this type of information is a book by Eleanor Babco, Associate Editor of *Manpower Comments,* entitled *Supply and Demand for Scientists and Engineers,* published in January 1982.

Presented at Symposium on Career Opportunities: How to Get a Job in Physiology, FASEB Spring Meeting, 20 April 1982, New Orleans, LA.

The current article, directed at the candidate in search of a permanent job in physiology, will review the qualifications the candidate must have and list sources of information about the present job market and agencies through which the candidate's availability may be made known.

A candidate must be properly qualified, needless to say. To attract a prospective employer, no amount of information regarding the mechanics of getting a job will circumvent this. Certain specific credentials are basic: first, a good general knowledge of physiology, documented by the academic record.

Most jobs in physiology require teaching; it is important that the candidate be an experienced teacher. This is important for not only the benefit of students but for the physiologist as well; the teaching process in itself is an excellent device for getting a better handle on the subject matter.

The candidate must show evidence of being an effective investigator. Physiology is an investigative science. This means publications. The applicant should have two or more postdoctoral years and should have three or more publications in good journals.

A very important credential is the letters of recommendation that the candidate obtains from his mentor or other senior physiologists.

During the process of application, the presentation of a research seminar is ordinarily required. This must be outstanding. Perhaps the single most convincing evidence of the candidate's qualification is the ability to present an excellent research seminar. In the seminar the candidate should be able to demonstrate why his research is important and that he is excited about continuing this research. It should show that he has had broad training in research.

The candidate must have flexible research plans. It is important for the candidate to realize that he should not place constraints on his opportunities for a job by being too demanding as to the type of research he will carry out. After all, the basic satisfaction from research is problem solving, and in general this satisfaction does not differ in the various fields of physiology.

Beyond the somewhat quantifiable credentials, the candidate should *like* what he or she is doing. It should be evident that the hard work required for a productive career as a physiologist be relished. Unless he or she can speak with manifest genuine enthusiasm about teaching and research in this field, this should be recognized as a time to reconsider choosing physiology as a profession.

Before listing the individual channels through which the candidate may search for a job, it should be of value to consider the characteristics of a usable curriculum vitae the candidate must have current at all times. The curriculum vitae is the standard instrument for advertising credentials. The major categories are *1*) personal information, *2*) education, *3*) work experience, *4*) professional interests, and *5*) publications. This information should be presented in neat well-organized format. The document should be concise. For instance, "professional interests" should be no more than one or two very short paragraphs. Lists of publications should be comprised of separate headings, including *1*) thesis, *2*) full scientific publications, published or in press, *3*) manuscripts submitted, and *4*) abstracts.

Of the specific means available to help the young physiologist find a job, probably the two most important channels are assistance given him by his mentor and facilities available in the FASEB Placement Service. These two resources are described in detail in the accompanying articles.

The following devices should also be used: Want Ads may identify appropriate "Positions Open" and advertise for "Positions Wanted." Most used of the national journals are *Science* (Room 207, 1515 Massachusetts Ave., NW, Washington, DC 20005) and *Federation Proceedings* (FASEB Placement Service, 9650 Rockville Pike, Bethesda, MD 20814). The journal *Clinical Research* will publish listings of positions available for Ph.D.'s in clinical departments. These will be published three times yearly, probably in October, February, and March.

Physiologists are employed by several types of institutions: academic positions in any of the 95 medical schools in this country may be applied for by contacting chairmen of Physiology Departments, whose names may be obtained in the AAMC Directory of American Medical Education (available from AAMC, 1 Dupont Circle NW, Washington, DC 20036, and available in the Dean's office of each medical school).

Letters of inquiry should include a curriculum vitae, letters of recommendation, and a statement of availability. It is valuable for the letter of inquiry to include a specific statement as to why the candidate is interested in that particular medical school.

Whereas there are fewer than 100 medical schools in this country, hundreds of undergraduate colleges employ the service of biologists, offering jobs for which a physiologist qualifies. In these cases letters of inquiry should be directed to the chairman of the Division of Biology, and it would be of value to learn by phoning that college or university the name of this individual.

Many physiologists are currently finding desirable positions in clinical departments, primarily research appointments, although frequently the physiologist may have a joint appointment, including some teaching function, with the Department of Physiology in that medical school.

The requirement for physiologists in industry is expanding. Letters of inquiry may be directed to the personnel officer of any major pharmaceutical house. It is the practice of the personnel department to circulate such letters and supporting documents to the appropriate department heads.

Opportunities for physiologists available in the Government may be explored by submitting Application Form No. 171 to the appropriate government agency such as NIH, the Armed Forces, or NASA.

Finally, three generalizations which may facilitate the finding of an appropriate job:

1. Make yourself known to senior physiologists who are in a position to write letters of recommendation for you.
2. Use all communication channels possible in advertising your availability. The more doors you knock on, the more likely you are to find one that will open for you.
3. Plan ahead. Because of the time involved in the decision making for any appointment, it is desirable to start your search at least one year before you need the position. Failing to plan is planning to fail.

The FASEB Placement Service

BILLY CLEMENT
Federation of American Societies for Experimental Biology
Rockville, Maryland 20814

The Federation of American Societies for Experimental Biology was founded in 1912. According to the 1913 APS Directory of Members, its first President was "Samuel J. Meltzer, M.D., LL.D., Universities of Maryland and St. Andrews, 13 West One Hundred and Twenty-First Street, New York City—Member of the Rockefeller Institute for Medical Research, Head of Department of Physiology and Pharmacology, Member of the National Academy of Science." He was also the President of APS that year. An article in *Federation Proceedings,* September 1951, states that "Dr. Meltzer contributed $300.00 of personal funds to organize a Scientific Information Service"—the beginning of the Placement Service in 1919. At the time of the donation $300.00 was a sizable amount since Dr. Meltzer's beginning annual salary had been only $1,500.00 a few years before.

I cannot talk exclusively on "How To Get a Job in Physiology" because I am also concerned with the biochemists, pharmacologists, pathologists, nutritionists, and immunologists. I have out of necessity added a seventh category, health-related fields, to serve scientists, such as biomedical engineers, scientific writers, and those interested in sales, who do not fit in the six categories of the Federation. I have often had difficulty in identifying the field of some candidates. They may have a degree in physiology, but their specialties by training or experience or the titles of their publications suggest that they may be biochemists, immunologists, etc. Dr. Ray Daggs, APS Executive Officer for many years, told me, "If you can't make up your mind, just put them in physiology; after all we are the father of all biomedical sciences."

Let me now tell you how we try to help candidates find jobs and employers find scientists. For *employers,* the fee is $280.00 for commercial organizations and $140.00 for academic and other nonprofit institutions. This fee includes the February "List of Candidates," which this year contains the applications of 540 candidates, and allows two people from each organization to interview any of the candidates attending the Annual Meeting. For *candidates,* the fee of $10.00 includes publication of applications in the February "List of Candidates," a 50-word resume in the Employment Opportunities section of *Federation Proceedings,* the use of the Placement Service at the Annual Meeting and the ASBC Meeting when they meet separately (whether FASEB manages their meeting or not), and since last year the APS Fall Meeting Placement Service.

The "Positions Desired" section of Employment Opportunities in *Federation Proceedings* is another means of communicating with potential employers. A minimum fee of $15.00 is charged for up to 6 resumes and $2.50 for each additional resume. In 1981, 1,558 candidates' applications were sent to employers through this service. Searches provide still another method. An employer may write or phone supplying key words. I review the file and locate possible candidates. Then after reviewing them by phone with the potential employer, I send only the applications the employer feels may fit the opening. The minimum charge for a search is $30.00 and or $2.50 for each over the minimum. In 1981, 1,380 applications were sent to the employers from these searches.

In 1975, I conducted my first Placement Service for The Endocrine Society. This is a year-round service with monthly "Positions Available" and "Positions Desired" in *Endocrinology* and *the Journal of Clinical Endocrinology and Metabolism.* The Endocrine Candidate's 50-word resume is also published in *Federation Proceedings.* The Society for Neuroscience asked me to form their Placement Service in 1977. This fortunately provided an audience for the neurophysiologist and psychopharmacologist applications in the FASEB file. The Neuroscience Placement Service is a meeting-only service; however, the candidate applications are retained in my office and are available for searches and review by employers. I have also conducted the Placement Service for the American Society of Anesthesiologists since 1975.

Since 1968 the FASEB Placement Service at the Annual Meetings has registered 13,517 candidates, of whom 3,036 were physiologists, served 3,916 employers with 4,280 interviewers, posted 6,344 position descriptions, and scheduled 45,530 interviews. A special survey in 1976 showed that 460 candidates had been hired through our service.

One subject of importance now is job trends. What is happening in the job market? For the past two years there has been a governmental job freeze. In the past it has been felt that industry could take up the slack until a freeze is over. It has not happened this time. I have been told that when an ad from industry appears in *Science* the company is inundated with applications, many not even remotely qualified. However, there are still jobs out there. As of 13 April, we had 223 employers registered. I still do searches every week, and the *Federation Proceedings* requests keep coming in. I will continue to explore every possibility for job openings, but I need help from every one of you to spread the word about the FASEB Placement Service at 9650 Rockville Pike, Bethesda, MD 20814.

Presented at Symposium on Career Opportunities: How to Get a Job in Physiology, FASEB Spring Meeting, 20 April 1982, New Orleans, LA.

New Zip Code 20814

The Society's zip code has been changed from 20014 to **20814**. Be sure to address all materials accordingly. The Post Office will not deliver items mailed to the old zip code.

New Data Show Changing Science Labor Force

(From Scientific Manpower Commission, 1 April 1982)

The Scientific Manpower Commission has published a new compilation of data on the participation of women and minorities in the professional population which shows progress in many areas.

• Women are continuing their rapid progress in obtaining the education required for a professional career. Minority group members also are increasing their share of earned degrees, although at a somewhat slower pace. During the decade of the 1970's, women earned 45.2% of the bachelor's, 44.5% of the master's and 20.7% of the doctorates awarded. By 1980, these proportions had risen to 49.2%, 49.5% and 30.3% respectively. Minorities have increased their share of freshmen enrollment from 7.9% of men and 9.4% of women in 1971 to 10.5% of men and 12.4% of women in 1981. By 1979, their representation among new graduates reached 10.5% of men and 13.5% of women at the bachelor's level, 10.2% and 12.8% at the master's level, and 8.7% and 10.3% at the doctoral level. Black women have higher representation in higher education relative to black men than do white women relative to white men.

• In most of the professions, both women and minorities have made large strides. In medicine, the proportion of women among graduates has risen from 9.2% in 1971 to 24.8% in 1981. Minorities increased their share of medical degrees from .2% in 1970 to 10.6% in 1981.

• In the biological sciences, the proportion of women has risen significantly. By 1980, women earned 42.4% of the bachelor's degrees, 37.1% at the master's level, and 28.0% at the doctorate level.

• Both women and minorities have increased their proportional representation in the professional labor force, although their proportions are still below their representation among recent graduates, particularly in those fields where they were poorly represented prior to 1970. Women are now 38% of biologists, 20% of chemists, 13% of physicians, 25% of pharmacists and economists, and half of all psychologists. Minorities now make up 11% of physicians. Women are 44.3% and minorities 8.9% of all professional and technical workers in the United States.

• The federal government is a major employer of professionals including women and minorities. However, women of all races still lag well behind their male counterparts in grade level and thus in salary. For example, the government employed 8,083 chemists in 1980, of whom 18.3% were women. However, their salary was only 79.1% that of men's.

• Employment of women and minorities in higher education has grown slowly over the 1970's, and women's progress up the academic ladder is still lagging far behind that of men. Women are disproportionately overrepresented among nonfaculty researchers in higher education institutions while men are disproportionately overrepresented in the tenured professoriate. Among scientists and engineers employed full time at colleges and universities in 1981, women are 17.2% of those in doctorate-granting institutions and 23.5% of those in two-year institutions. Seventy percent of all male faculty at all higher education institutions have tenure compared with less than 50% of women.

• Both women and minorities show slight gains in administrative positions in academic institutions. Among all academic administrators in 1980, 63% are white men, 5.8% are minority men, 17.7% are white women, and 2.0% are minority women. However, women and minorities hold few top administrative jobs except at institutions serving principally women or minority students.

These and hundreds of other statistical parameters of the participation of women and miniorities in the professional work force are available in a new 304-page third edition of *Professional Women and Minorities—A Manpower Data Resource Service,* which provides a comprehensive statistical picture of the professional work force in the United States, detailing the participation of women and minorities in the natural and social sciences, engineering, arts, humanities, education and all of the professions. This book includes basic information on affirmative action; manpower data in all fields from more than 200 sources; annotated recruitment resources, both for specialized fields and for general recruitment of professional women and minorities; a detailed bibliography and a comprehensive cross-index of the 350 tables which provide breakouts by sex and/or minority status. Both historical and current data on enrollments, degrees, and on general, academic and federal work force participation of women and minorities are presented by field and subfield.

Professional Women and Minorities—A Manpower Data Resource Service (3rd ed.), by Betty M. Vetter, Eleanor L. Babco and Susan Jensen-Fisher is available for $60.00 from the Scientific Manpower Commission, 1776 Massachusetts Ave. NW, Washington, DC 20036.

Elections to the National Academy of Sciences

The National Academy of Sciences announced the election of sixty new members in recognition of their distinguished and continuing achievements in original research. Four APS members were elected: *James O. Davis,* Professor and Chairman, Department of Physiology, University of Missouri School of Medicine; *Irving T. Diamond,* James B. Duke Professor of Psychology, Duke University; *Daniel Steinberg,* Professor of Medicine, University of California, San Diego; and *Charles F. Stevens,* Professor of Physiology, Yale University School of Medicine.

Laboratory Animal Reform Bill in Congress Includes All Mammals and Birds

An omnibus bill (H.R. 6245) to promote the development of nonanimal methods of research, experimentation, and testing and to assure the humane treatment of all warm-blooded animals used in the laboratory is under consideration by the Congress.

The bill describes warm-blooded animals as all birds and mammals.

The proposal, entitled the "Humane Care and Development of Substitutes for Animals in Research Act," was introduced by Rep. Doug Walgren (D-PA), Chairman of the House Subcommittee on Science, Research, and Technology, and is cosponsored by Rep. Don Fuqua (D-FL), Chairman of the House Committee on Science and Technology.

Other cosponsors of the bill include Rep. Margaret M. Heckler (R-MA), Rep. George E. Brown (D-CA), Rep. Harold C. Hollenbeck (R-NJ), Rep. Robert A. Roe (D-NJ), Rep. Stanley N. Lundine (D-NY), and Rep. Mervyn M. Dymally (D-CA).

The proposed legislation has four basic concepts:

- It places special emphasis on the development of research and testing methods that reduce the numbers of animals used in research and testing or that minimize animal pain and stress.
- It requires stricter standards for accreditation of Federal laboratories and federally funded institutions using animals in research or testing.
- It requires participation of community representatives and veterinarians on animal care committees at research institutions.
- It requires scientific peer review panels to evaluate the importance of proposed scientific findings as related to any animal distress that may be involved in the experiments.

The bill authorizes appropriations totaling $45 million during the next three fiscal years for the research and development of methods to reduce the number of laboratory animals being used and for ways to reduce pain and stress in such animals.

An additional $30 million were authorized to be appropriated in Fiscal Year 1983 for the purpose of assisting research institutions to improve animal care facilities in order to attain compliance with proposed accreditation standards. However, this appropriation was deleted by Walgren prior to the subcommittee's review of his bill.

Accreditation standards are to be determined by a private agency or agencies designated by the Secretary of the U.S. Department of Health and Human Services. Such agencies would be required to conduct accreditation inspections of research institutions at least once every three years. This would be in addition to the inspections by the U.S. Department of Agriculture currently required under the terms of the Animal Welfare Act.

Federal funds for research, experimentation, and testing will be denied to any Federal agency or to any institution receiving Federal support that fails to comply with the accreditation standards.

In addition to the accreditation inspections, federally funded research institutions using animals will be required to have an animal care committee which will be charged to conduct at least two inspections a year to assure that the animals being used are receiving appropriate care and treatment.

The composition of the animal care committee requires that at least one member who is not affiliated with the institution be responsible for representing the community's concerns for the welfare of all animal subjects. Other committee membership requirements are that at least one member be a veterinarian and that not more than three members are from the same administrative unit of the institution.

The bill would also require that the scientific peer review committees approve the justification for anticipated animal suffering in terms of demonstrable benefits of the research; that appropriate assurances are made for the use of tranquilizers, analgesics, and anesthetics in all cases involving surgery or other invasive procedures on animals; and that a veterinarian is employed in the planning of procedures involving the direct use of conscious animals or for chronic long-term invasive procedures.

APS Supports Federal Inspection Role

The American Physiological Society has told both the Senate and House Appropriations Committee that inspection of the nation's research facilities is a legitimate role of the Federal Government which should be continued by the U.S. Department of Agriculture's Animal and Plant Health Inspection Service (APHIS).

Orr E. Reynolds, Ph.D., Executive Secretary of the Society, said in a statement prepared for the two Congressional committees:

"The inspection function by APHIS is of critical importance, especially if federally funded research is to continue to be carried out under standards prescribed by the Government. To transfer this legitimate Federal function of inspections of research facilities to organizations, agencies, or individuals throughout the nation would most assuredly result in the adoption of widely ranging, chaotic, inhomogeneous, and disparate body of regulations to govern federally funded research."

The Society's testimony was in opposition to an Administration proposal to reduce the APHIS budget from $4.9 million to $1.5 million in Fiscal Year 1983. To accomplish this savings the Administration proposes to transfer to states, industry groups, humane societies, and individuals the primary responsibility for the enforcement of the Animal Welfare Act, which includes inspection of all research facilities using laboratory animals.

"This responsibility," Dr. Reynolds said, "includes the inspection of research facilities to assure that the standards established by the Animal Welfare Act for the humane treatment of laboratory animals are met and

are maintained. These standards . . . include transportation, purchase, sale, handling, housing, feeding, watering, sanitation, ventilation, shelter from extreme weather and temperature, and the reporting of both pain and stress as well as the alleviation of pain and stress in research involving animals.

"The fact of the matter is that the users of laboratory animals have accepted the spirit of the Animal Welfare Act and have adhered to its requirements with commendable success."

Joining with the Society in the testimony before the two committees were the American Institute of Biological Sciences, the Association of American Medical Colleges, the Federation of American Societies for Experimental Biology, and the National Society for Medical Research.

The testimony also was shared with the Senate and House Agricuture Committees. Sen. Jesse A. Helms (R-NC) and Rep. E. (Kika) de la Garza (D-TX), Chairmen of the Senate and House Agriculture Committees, respectively, both wrote letters concurring with the Society's position and saying that their committee would urge for funding of APHIS at the current level.

Society's Public Affairs Committee Restructured

The Public Affairs Committee of the American Physiological Society has been restructured to provide the Society with a more effective mechanism for meeting the increasing pressures from local animal rights activist organizations and to broaden the base for constituency input regarding national issues.

The restructured Committee as approved by Council is now composed of at least one member in each state. This group will serve as the Public Affairs Advisory Committee. Three other Society members will be selected to serve as the Public Affairs Executive Committee. John T. Shepherd, M.D., D.Sc., will continue as the Committee's Chairman.

The members of the Public Affairs Advisory Committee are responsible for identifying and monitoring all legislation and regulation pertaining to biomedical research that is being considered by state, county, or local governments within the member's state and to keep the Society informed of such actions and subsequent developments.

Each member may develop his or her own working group within the state, if desired, to assist in this effort. Also assistance from the Society's national office will be made available, whenever requested, to help in local or state problems.

The Public Affairs Executive Committee is charged with dealing with all matters pertaining to public affairs that affect physiologists and to bring such matters to the attention of the Council and to act upon Council recommendations. The Public Affairs Executive Committee will also coordinate its activities with the Society's Committee on Animal Care and Experimentation and with the Public Affairs Committee of the Federation.

The full Committee will meet twice a year at the Society's spring and fall meetings to share information regarding national, state, and local issues and to develop techniques for meeting local challenges to physiologists.

William Samuels

Fifty-Fifth President of APS

Dr. Walter C. Randall of Loyola University of Chicago has been named President of the American Physiological Society effective July 1, 1982.

Dr. Randall is internationally known for his research on the nervous control of the heart and cardiodynamics. He is Professor of Physiology in the Stritch School of Medicine, Loyola University of Chicago, having served as Chairman of the Department from 1954 to 1975. During that period, he coordinated an interdepartmental program of cardiovascular research. His research also has included experiments on the physiology of circulation and the regulation of sweating and body temperature. He has published over 350 scientific papers on his various research projects.

In 1971, Loyola University recognized Dr. Randall's meritorious service by awarding him the Stritch Medal. The Stritch Medal is awarded each year to a physician or medical researcher who exhibits a high degree of professional competence, resourcefulness, and dedication. It is named after the late Samuel Cardinal Stritch, former Archbishop of Chicago, for whom the Medical School is named.

Dr. Randall was born in Akeley, Pennsylvania. He earned his Bachelor of Arts Degree from Taylor University, Upland, Indiana, in 1938. He earned his M.S. and Ph.D. in Physiology from Purdue University, Indiana, in 1940 and 1942, respectively. He was a Postdoctoral Fellow at Western Reserve University in the Department of Physiology under Dr. Carl J. Wiggers. Dr. Randall was associated with St. Louis University from 1943 to 1954, when he moved to Chicago and became Chairman at Loyola. He has been actively involved in research as a Visiting Scientist at the National Spinal Nerve Injuries Center, Aylesbury, England; National Spinal Injuries Center, VA Hospital, Long Beach, California; and at the University of Washington in Seattle. He served for two terms as a member of the Program-Project Committee of the National Heart, Lung, and Blood Institute and was Circulation Editor of the *American Journal of Physiology* and *Journal of Applied Physiology* for two successive terms. He has been a member of the American Physiological Society since 1943 and has served as a member of Council since 1976. He is deeply interested in problems and concerns of young physiologists and has served as Chairman of the committee on Career Opportunities in Physiology since 1979. He is an Honorary Fellow in the American College of Cardiology and has been actively involved in the research problems of both the American and Chicago Heart Associations, as well as a member of numerous other scientific organizations. Dr. Randall and his family live in Park Ridge, Illinois.

Proposed Amendment to the Bylaws for Amendments

The following amendment to the Bylaws approved by Council will be offered for vote at the Society Business Meeting, Thursday, October 14, 1982.

ARTICLE XIII. *Amendments*

Current Bylaw

SECTION 2. Adoption. These Bylaws may be amended at any Business Meeting of the Society by a two-thirds majority of the regular members present and voting.

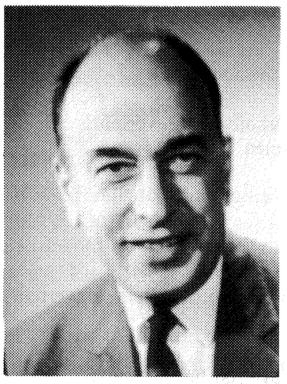
Proposed Bylaw

SECTION 2. *Adoption.* These Bylaws may be amended at any Business Meeting of the Society by a two-thirds majority vote of the regular members present and voting. **If a proposed amendment to the Bylaws receives a majority vote of the regular members present and voting at the Business Meeting but fails of a two-thirds majority necessary for passage, such proposed amendment must be submitted to the regular membership by mail ballot. A two-thirds majority of the regular members responding within thirty (30) days after submission shall result in passage of the amendment.**

APS Election Results

Dr. *A.P. Fishman,* Director, Cardiovascular-Pulmonary Division, Department of Medicine, Hospital of the University of Pennsylvania, Philadelphia, was elected President-Elect and Dr. *F.G. Knox,* Chairman, Department of Physiology and Biophysics, Mayor Medical School, Rochester was elected to Council for a four-year term. Both elections become effective July 1, 1982.

Ray G. Daggs Award



The presentation of the Ray G. Daggs Award to Robert W. Berliner, was made by Dr. Francis J. Haddy, who said, "The Ray G. Daggs awardee is selected from among the members of the Society by a special committee in recognition of distinguished service to the Society and to the science of physiology.

"All of you know or know of Dr. Berliner. He has had an illustrious career which has touched all of us, either directly or indirectly. He eminently satisfies the requirements for the award—he served the Society in many ways including as President and has made many important contributions to the science of renal physiology. The selection committee is responsible for the preparation of a citation which accompanies the award."

It is fitting that Dr. Robert W. Berliner has been selected for the Ray G. Dagg's Award. Dr. Berliner's career exemplifies the ideals and accomplishments in science and service to society which very few physiologists have been fortunate and talented enough to achieve.

His career in science was started at Yale University, where he received a Bachelor of Science Degree in 1936. This was followed by the Degree of Doctor of Medicine, which was awarded in 1939 by Columbia University. Following an internship at Presbyterian Hospital in New York and a residency at the Goldwater Memorial Hospital, Dr. Berliner became first an assistant and then an instructor in Medicine at New York University. From 1947 to 1950 he was Assistant Professor of Medicine at Columbia University. At that point, Dr. Berliner accepted the responsibility of Chief of Laboratory of Kidney and Electrolyte Metabolism at the National Heart Institute, which he held from 1950 to 1962. From 1954 to 1968 Dr. Berliner was Director of Intramural Research at the National Institutes of Health. From 1968 to 1969 he became Director of Laboratories and Clinics, and then from 1969 to 1973 Dr. Berliner served as Deputy Director of Science for the National Institutes of Health. In 1973 he left Washington and returned to New Haven, where he became Professor of Physiology and Medicine and Dean of the School of Medicine at Yale University.

Dr. Berliner's main research interests were concerned with renal function. He and his research associates laid the foundation for our understanding of potassium and hydrogen ion transport by reneal tubules, the control of osmolality, and the factors involved in production of hyper- and hypotonic urine. These pioneering efforts formed the basis of much of our modern understanding of renal function.

Dr. Berliner served on the editorial board of the *Journal of Clinical Investigation and Circulation Research* for 10 years, the *American Journal of Physiology* for 5 years, and *Circulation.*

He is Past President of the American Physiological Society, the American Society of Clinical Investigation, and the American Society of Nephrology and past Vice-President of the American Association for the Advancement of Science. In addition, he is a member of the National Academy of Sciences, the National Institute of Medicine, the Royal Society of Medicine, the American Academy of Arts and Sciences, the Association of the American Physicians, the Society of Experimental Biology and Medicine, the American Society of General Physiologists, and the Harvey Society.

Dr. Berliner received the Doctor of Science Degree from the Medical College of Wisconsin and from Yale University. In addition, he received the distinguished service award from the Department of Health Education and Welfare, the Homer Smith Award, the distinguished Achievement Award in Modern Medicine, and the Research Achievement Award of the American Heart Association.

It is obvious that we honor our society by honoring Dr. Robert Berliner with the Ray G. Dagg's Award.

Dr. Robert W. Berliner responded, "I am really deeply honored to be chosen for the Award, and I do hope my contributions to the Society has come close to balancing what the Society has meant to me.

"I am proud to accept this award in the name of Ray G. Daggs. He was executive officer of the Society for some sixteen years, and, in fact, he was the first full-time executive officer in which capacity he succeeded Milton O. Lee, who was the chief executive officer of both the Federation and APS. Milton was a loyal and imaginative supporter of the goals of the Society, but he naturally had some conflict of interest. Ray was a vigorous supporter of physiology and APS, and there was never any question about whose interests he sought to further. It is a great honor to receive this Award in his name. Thank you very much."

APS Fall Meeting
October 10–15, 1982, San Diego, CA

1982 Bowditch Lecture
Tuesday, October 12, 1982

Electrogenic ion pumps and other determinants of membrane potential in vascular muscle

Kent Hermsmeyer, Professor of Physiology
University of Iowa College of Medicine
Iowa City, IA

Refresher Course on Microcirculation

October 11, 1982

10:00 The microcirculation: old concepts and new facts. B.W. Zweifach
10:30 Blood flow in the microcirculation. H.H. Lipowsky
11:00 Transcapillary water flux. J.N. Diana
11:30 Solute and water exchange in the kidney. C. Bayliff
12:00 Discussion
12:30 Lunch
1:30 Concepts of regulation of the microcirculation. H.J. Granger
2:00 Long-term adaptation of capillary density. N. Banchero
2:30 Pathophysiological mechanisms in the microcirculation. H.G. Bohlen
3:00 Discussion

Schedule of Invited Sessions

Monday, Oct 11, A.M.

Refresher: Microcirculation. Brian Duling
(No other scientific sessions)

Monday, Oct 11, P.M.

Methodology Tutorial Sessions: Recent advances in physiological monitoring. Organized by H. Sandler (No other scientific sessions)

Tuesday, Oct 12, A.M.

Symposium: Man at high altitude. Session I. Chaired by J.B. West
Symposium: Teaching of cardiovascular physiology outside the lecture hall. Organized by W.T. Beraldo, J.A. Michael, A.A. Rovick
Tutorials
New concepts of nephron structure. F.S. Wright
Physiology of gastrointestinal smooth muscle. J.H. Szurszewski
Comparative reproductive physiology. B. Lasley

Tuesday, Oct 12, P.M.

Symposium: Blood-brain barrier. Chaired by D.D. Heistad
Symposium: Regional vascular behavior in the gastrointestinal wall. Chaired by H.G. Bohlen
Tutorials
The role of aldosterone, sodium, chloride and potassium in metabolic alkalosis. N.A. Kurtzman
Hormonal control of the mammalian fetus and infant growth. C.S. Nicoll
Neurochemical mechanisms of thermoregulation. R.D. Myers

Wednesday, Oct 13, A.M.

Symposium: Man at high altitude. Session II. Chaired by S. Lahiri
Symposium: Ionic channels in excitable membranes. Chaired by F. Benzanilla
Special Lecture: Reflexes provoked by cytoglucopenia. C. Timo-Iaria, President, ALACF
Tutorials
Endorphins. F.E. Bloom
Cerebral cortex. R.B. Livingston

Wednesday, Oct 13, P.M.

Symposium: Neurophysiological mechanisms controlling circadian rhythmicity. Chaired by H. Arechiga
Symposium: Differentiation of epithelial cells. Chaired by M. Cereijido
Tutorials
Temperature regulation during exercise. C.V. Gisolfi
Neurotoxins as tools for physiological investigation. L. Friere-Maia

Thursday, Oct 14, A.M.

Symposium: Man at high altitude. Session III. Chaired by R.M. Winslow
Symposium: Anaerobic energy metabolism of invertebrates. Session I. Chaired by C.S. Hammen
Tutorials
Neural integration at the level of autonomic ganglia. D.L. Kreulen
Analysis of physiological systems via mathematical models. J. Hazelrig
Pattern generators in the central nervous system of vertebrates. J.L. Feldman

Thursday, Oct 14, P.M.

Symposium: Is efferent control of arterial baroreceptors important? Chaired by K. Sagawa
Symposium: Temperature effects on fish. Chaired by L.I. Crawshaw and J.R. Hazel
Tutorials
Water channels in red blood cells. R.I. Macey
New concepts in acid-base balance. P.A. Stewart
Membrane transport processes. R.B. Gunn

Friday, Oct 15, A.M.

Symposium: Exchange and compartmentation of calcium in the heart. Session I. Chaired by G.A. Langer
Symposium: Anaerobic energy metabolism of invertebrates. Session II. Chaired by W.R. Ellington
Symposium: Gravitational physiology. Session I. Chaired by H. Bjurstedt

Friday, Oct 15, P.M.

Symposium: Exchange and compartmentation of calcium in the heart. Session II. Chaired by G.A. Langer
Symposium: Gravttational Physiology. Session II. Chaired by A.S. Ushakov

SCUBA Divers Attending the Fall APS Meeting

A weekend trip to San Clemente Island is being organized for APS members (and guests) who are certified SCUBA divers. *For information:* Stephen C. Wood, Ph.D., Department of Physiology, University of New Mexico, School of Medicine, Albuquerque, NM 87131.

Future Meetings

1982	
APS Fall Meeting	Oct 10-15, San Diego
1983	
FASEB Annual Meeting	Apr 10-15, Chicago
APS "Fall" Meeting	Aug 20-24, Honolulu
IUPS Congress	Aug 28-Sep 3, Sydney
1984	
FASEB Annual Meeting	Apr 1-6, St. Louis
*APS "Fall" Meeting	Jul 29-Aug 7, Lexington
1985	
FASEB Annual meeting	Apr 21-26, Anaheim
*APS "Fall" Meeting	Aug 4-9, Buffalo

*Campus meeting

Placement Service

Location: Atlas Ballroom, Town & Country Hotel

Registration:
Tuesday, October 12 ..... 8:30 AM–5:00 PM
Wednesday, October 13 ..... 8:30 AM–5:00 PM

Interviews:
Wednesday, October 13 ..... 8:30 AM–5:00 PM
Thursday, October 14 ..... 8:30 AM–12:30 PM

Interview Facilities:
To use the interview facilities, employers, interviewers, and candidates must register for the APS Fall Meeting as well as for the Placement Service and must be in attendance at the Meeting.

Employers:
Commercial Organizations $150.00
Academic and other nonprofit institutions .... $ 75.00

Fee includes *1*) use of the interviewing facilities at the APS Fall Meeting; *2*) posting position vacancy descriptions at the APS Fall Meeting; and *3*) copy of application of each candidate in attendance at the APS Fall Meeting.

Employers *not registered* with the Placement Service who wish to post a position vacancy notice at the APS Fall Meeting, in the "No Interviews Granted" section of posted positions, may do so for a fee of $25.00 per position posted. *Payment must accompany position description.*

Candidates:
Annual fee $10.00

Annual fee includes *1*) use of the interviewing facilities at the APS Fall Meeting; *2*) publication of a 50-word resume in one issue of *Federation Proceedings; 3*) review of job opportunity listings at the APS Fall Meeting; *4*) inclusion of application in FASEB Placement Service file for review by employers visiting FASEB campus or by FASEB staff conducting searches on behalf of employers; and *5*) publications of application in the 1983 List of Candidates, to be published by FASEB in February.

Candidates registered with the FASEB Placement Service in 1982 are not required to pay additional APS fee.

Candidates who register with the Placement Service at the APS Fall Meeting are automatically registered with the FASEB Placement Service until July, 1983, unless the Placement Service is notified they are no longer available to accept a position.

Advance Registration:
Candidates and employers may register for the Placement Service at the APS Fall Meeting; however, advance registration is strongly recommended. *Deadline for Placement Service Advance Registration is September 17, 1982.*

Forms and instructions for candidate and employer registration for the APS Fall Meeting Placement Service will be mailed upon request.

Direct all correspondence to: Billy M. Clement, Manager, APS Fall Meeting Placement Service, 9650 Rockville Pike, Bethesda, MD 20814; 301/530-7020.

APS 127th Business Meeting

Time: 4:45 P.M., Wednesday, April 21, 1982
Place: New Orleans Hilton Hotel, Grand Ballroom C

I. Call To Order

President, F.J. Haddy called the meeting to order and welcomed the members to the 127th Business Meeting of the Society. The agenda and ballot for the Election of New Members were distributed to the membership.

It was noted by Dr. Haddy that the President-Elect traditionally presents the Membership Report. Unfortunately, W.C. Randall, who had emergency surgery, was not with us. He said he will see us at the next meeting.

II. Report on Membership

The Executive Secretary-Treasurer, O.E. Reynolds, reported on the current status of membership and deaths since the last meeting.

A. Summary of Membership Status. Since the last meeting, the Society membership has increased bringing the total to 6,020. As of this report, there are 4,445 Regular members, 539 Emeritus members, 11 Honorary members, 81 Corresponding members, 702 Associated members, and 242 Student members.

B. Deaths Reported Since the Last Meeting. The names of those members whose deaths have been reported since the previous meeting were read by Dr. Reynolds, who asked the members to stand for a moment of silence in tribute to them (p. 149).

III. Election of Members

A. Appointment of Tellers. Dr. Haddy appointed P.A. Chevalier, J.P. Filkins, R.P. Steffen, and L.B. Hinshaw and asked them to collect the ballots for the Election of New Members.

B. Election of Members. Dr. Reynolds announced that all candidates on the ballot for Election of New Members were elected (p. 146).

IV. Election of Officers

As a result of the Election of Officers by mail ballot, Dr. Reynolds announced that the new President-Elect is A.P. Fishman and the new Councilor to serve a four-year term is F.G. Knox. There were 1,663 ballots received for the office of President-Elect and Councilor. Again, there was a sizable number of invalid ballots with 1,544 valid ballot for President-Elect and 1,428 for Councilor.

V. Ray G. Daggs Award

(See p. 133)

VI. Actions of Council

At the Cincinnati Meeting, reported Dr. Haddy, it was proposed that the Bylaws be changed to provide for amendments by mail ballot. A proposed amendment for this purpose was favorably acted on by Council and will be presented to the Business Meeting in the Fall, after its publication in *The Physiologist* (p. 132).

Council nominated all applicants for Regular membership except eleven, who were recommended for Associate membership. Council also nominated twelve of the thirteen applicants for Corresponding membership.

All except two applicants, whose applications were rejected, were recommended for Associate membership. All applications for Student membership were accepted for nomination by Council. A new Honorary member, Bjorn Folkow, was nominated.

In this age of litigation, Council considered the desirability of liability insurance for individuals carrying on work for the Society, for example, on editorial boards and committees. The Executive Secretary was authorized to investigate the possibility of obtaining such insurance.

A reorganization of the Public Affairs Committee was approved, resulting in an enlargement of the Public Affairs Committee and its division into two parts, a Public Affairs Executive Committee of three and a Public Affairs Advisory Committee consisting of at least one member from every state. The purpose of this committee is to make your Society more effective in dealing with activities at the state and local levels in matters affecting physiologists, particularly those having to do with animal care and experimentation. There will be a first meeting of the members of this new Public Affairs Advisory Committee immediately following the Business Meeting. Mr. William Samuels, who has been appointed as the Society's Public Affairs Consultant, will be addressing the meeting later about his activities. Changes in the structure of this committee and some related changes in the duties of the Animal Care and Experimentation Committee will be published in *The Physiologist.*

Council received a proposal for the establishment of a Section on Water and Electrolyte Homeostasis that was approved in principal. The statement of organization and procedures for the section will appear in *The Physiologist,* after it has been ratified at a meeting of the members of the newly formed section (p. 143).

Council received a report from the Long-Range Planning Task Force, which has been in operation for the last year and a half. Council requested the Task Force to continue its activities for one additional year in order to explore ways in which its recommendations can be implemented. A report of the Task Force's findings and recommendations will be published in *The Physiologist.*

A proposal was received from the Women's Caucus of the Society to establish a standing Committee on Women in Physiology. This was approved by Council.

A report was received from W.F. Ganong, Chairman of the Financial Development Committee, and Council was pleased to note that due to the activities the number of Sustaining Associates (p. 134) has grown from thirteen as of a few years ago to 29 at the present time. To make this relationship with industry meaningful, the Society has established a Liaison Committee with Industry. Membership of this committee was identified at this meeting, and it is hoped that its activities will greatly enhance the development of mutually advantageous programs between the Society and industrial organizations. Also, the individual contributions of members, including Emeritus members, in support of the Society has grown to quite a respectable figure in the last few years because of the efforts of this Committee. The Committee continues to work on new ways in which financial resources of the Society can be developed as a means of restraining the increase in dues in these inflationary times.

The Centennial Committee Chairman, Dr. Chevalier, reported on its many activities, and Council was pleased with the crystalization of a number of plans of the Centennial Celebration in terms of special lectures, the Centennial meeting, and all related publications. The 1987 Centennial Meeting will be held in Washington, DC, in April. The theme will be "A Century of Progress in Physiology." Statements of activities of this committee will be published in *The Physiologist* as they develop (p. 137).

In addition, Council received a number of Committee reports that are available for inspection in the APS office and will appear in *The Physiologist* (pp. 137–143).

Council reacted to a proposal from E.H. Wood, President of FASEB, that the name of the Federation be changed, recommending that the new name include the words "life sciences" and "federation." A motion to this effect was made at the FASEB Board meeting and was defeated.

In another action related to FASEB, Council approved a request from the Society for Experimental Biology and Medicine (SEBM) that the Society serve as sponsor for SEBM membership in the Federation. Subsequently, at the FASEB Board meeting, APS transmitted its sponsorship of SEBM. However, no action was taken for lack of a second sponsor.

VII. Public Affairs Activites

(See p. 130)

A.E. Taylor said that if laws are passed prohibiting the use of pound animals for research, the cost of animals will rise fourfold, which in turn will increase the cost of research. Unfortunately, this is already happening, and the state legislatures should be made aware of this.

Another member stressed the urgent need to continually educate the lay public as is done by the animal welfare groups. Science needs public opinion behind it, particularly at the state level.

VIII. New Business

A. Resolution Against the Use of Nuclear Weapons. A.C. Barger presented the following resolution, which was seconded and passed without a dissenting vote:

"The use of nuclear weapons is intolerable because of the overwhelming and indiscriminate damage they inflict on all living things, and their threat to the survival of civilization.

"The continued pursuit of knowledge and improvement of the human state makes little sense in the event of nuclear war. The increasing probability of this catastrophe already preoccupies the intellect and dampens the spirit.

"The continued escalation of the nuclear arms race seriously subverts resources desperately needed for support of education, science, the arts, and the humanities.

"Even the threatened use of nuclear weapons is totally unacceptable as an answer to international conflicts of interest. The life of the world's innocent populations cannot be held hostage to either the legitimate or misdirected interests of their governments.

"Both the US and USSR have manyfold the number of nuclear weapons required for the total destruction of both countries, thus making the 'balance' of number irrelevant. Attempts to redress 'balance' by either side can only mean uncontrollable escalation.

"This professional society therefore calls upon the President and the Congress of the United States to work with the Soviet Union and other nations to seek an immediate mutual halt to the nuclear build up, with no prior conditions, and to take positive steps toward reduction with the ultimate objective to total elimination of nuclear weapons."

B. Change in the Dates of the 1983 APS Fall Meeting. It was announced by Dr. O.E. Reynolds that the dates of the 1983 APS Fall Meeting have been moved back one day to allow members to participate in the satellite meetings which are being scheduled prior to the Congress of the International Union of Physiological Sciences. The Fall Meeting dates are August 20–24, 1983.

With no other business, the 127th Business Meeting was adjourned at 5:30 P.M., April 21, 1982.

Francis J. Haddy, President

Committee Reports

Animal Care and Experimentation Committee

October 1981–March 1982

Committee members: Mal Hast, R. Hazelwood, L. Ramazzotto, D. Robertshaw, O. Smith, and H. Cecil, Chairman.

Congressional Hearings on Use of Animals

On October 13–14, 1981, Rep. Doug Walgren, Chairman of the Subcommittee on Science and Technology, held public hearings on proposed animal welfare legislation. A summary of the hearings and Ernst Knobil's testimony appeared in *The Physiologist* [24(6) 13–15, 1981]. As an outcome of these hearings the Subcommittee staff is drafting new legislation to fund research on alternative methods and Rep. Pat Schroeder is drafting amendments to the Animal Welfare Act.

Animal Welfare Act Funding

The proposed FY–1983 budget for the U.S. Department of Agriculture designates a 75% reduction in the budget for inspections and enforcement of the Animal Welfare Act and indicates these responsibilities could be transferred to the states, industry, humane societies, and individuals.

First Conference on Scientific Perspectives in Animal Welfare, November 11–13, 1981

The Scientist Center for Animal Welfare held a workshop in Washington, DC, and discussed the institutional, investigator, funding agency, and editorial responsibilities in animal experimentation. Approximately ninety scientists attended, representing industry, academia, government, and animal welfare societies (Canadian Council on Animal Care, Canadian Federation of Humane Societies, and the Institute for the Study of Animal Problems). The most controversial issue was the use of random-source animals (pound dogs) in experimentation. Other issues were: *1*) composition of animal care committee, *2*) review of design of experiments in research protocols using animals, *3*) inspection of research animal facilities (role of USDA and AAALAC), and *4*) effectiveness of editorial review to ensure proper care and use of experimental animals. The proceedings of the conference will be published.

National Society for Medical Research

Business meeting held December 15, 1981. The appointment of four persons to Board of Directors was approved: J.E. Baer, D.H. Cohen, D.R. Thompson, and B.C. Zook. The membership also approved an expanded statement of propose. NSMR has set up a network at the state and local level to keep abreast of ordinances affecting the use of animals in research. NSMR is developing a model law or ordinance to be used by organizations or groups drafting legislation to further the availability of pound animals for research and provide for humane care of pound animals. C. Dennis, Treasurer, reported a 1981–82 budget of $188,000 with $68,000 received as of December 15, 1981.

FASEB Task Force for the Humane Care and Use of Animals in Research

FASEB has established a Task Force to set professional standards of guidelines for the humane care and use of animals in research. It is the desire of the Federation to adopt a statement of ethical guidelines that will serve to demonstrate the concern of scientists for reasonable care and humane structuring of experimental procedures utilizing animals. The Task Force is to explore all aspects of treatment of animals in scientific research and prepare a statement which can be recommended to the Governing Board of FASEB. O.E. Reynolds is Chairman and H. Cecil a member of the Task Force.

AAALAC

Orville Smith attended the meeting of the AAALAC Board of Trustees. The invited NIH representative, Dr. William Gay of the Division of Research Resources, indicated that NIH will *1*) begin spot inspections of the facilities of NIH grant recipients—both selected for cause and randomly selected—directed at reviewing all aspects of animal care, experimentation, etc.; *2*) begin to assess the local institutional review procedures as a part of the regular site visit; *3*) be concerned as to how the public is represented in the institutional review process; and *4*) revise the *Guide for the Use and Care of Laboratory Animals* in 1982 and will start with public hearings. The Guide is used by AAALAC as its source of standards.

Helene Cecil, Chairman

Centennial Celebration Committee

The Centennial Celebration Committee (CCC) Meeting was held at the New Orleans Hilton at 12:00 noon on April 20, 1982, with the following members in attendance: P. Chevalier, Chairman; R. Kollogg, L. Langley, A. Otis, M.C. Shelesnyak, and A. Fishman. Absent were R. Joy, M. Kafka, S. Ochs, and O.E. Reynolds (ex officio).

Centennial (1987) Meeting Site

The Centennial Celebration of the American Physiological Society will be held April 5–10, 1987 in Washington, DC, in conjunction with the FASEB Meetings. The CCC recommended to Council that the overall theme of this meeting be "A Century of Progress in Physiology." This recommendation was presented to Council as a motion (unanimous) from the CCC. Council, after some discussion, approved this recommendation unanimously.

Historical Lecture Series

Beginning with the 1982 Fall APS Meetings, to be held in San Diego, the CCC will sponsor a History of Physiology lecture that will focus on a selected area of Physiology. Kicking off this lecture series will be Dr. Otis speaking on the "History of Respiratory Mechanics." Other speakers are currently being recruited. The CCC would appreciate your suggestions and recommendations.

APS Centennial Celebration Fund

The Centennial Celebration Fund was established to provide financial assistance for the various publications and activities planned for the Centennial Celebration. Recent issues of *The Physiologist* have presented opportunities for members to make a contribution and to receive special jewelry of their choice, bearing the APS logo, in recognition of their contribution. The CCC is pleased with the response to date, but broader support will be needed to provide the necessary funds to make the Centennial meeting a truly commemorative one.

Historical Vignettes

The Historical Section of *The Physiologist* is the medium for publishing vignettes. A number of very interesting vignettes have appeared in recent issues, and the CCC would like to have more published as the APS looks toward its Centennial. Dr. Kellogg is spearheading this effort and would appreciate sugestions of physiologists to prepare historical vignettes. Properly written, these vignettes provide valuable insight and "serendipity" that will be lost forever, if not recorded and published.

Department Histories

In recent issues of *The Physiologist,* several histories of departments of physiology have appeared in response to a letter sent by Dr. Otis to all department chairmen inviting them to prepare a history of their department. Nearly 100 departments have indicated an interest in pursuing this goal. The CCC urges all those interested in documenting the "genesis and evolution" of their department to send your completed manuscripts to Dr. Otis (University of Florida).

Instrumentation Exhibit

Planning for an exhibit of instrumentation used in American Physiology during the past 100 years continues. The exhibit will be housed in the Smithsonian Institution in Washington, DC, and will be available for viewing during the Centennial Meeting in 1987. This exhibit represents the joint efforts of the Smithsonian and the Bakken Library, formerly known as the Bakken Museum of Electricity in Life, located in Minneapolis, Minnesota.

History of the APS, 1887–1987

The History of the society is being prepared under the coeditorship of Drs. Brobeck and Reynolds. Work on this volume is progressing nicely.

Circulation of the Blood: Men and Ideas

This book, edited by Drs. Fishman and Richards, is being reprinted by the APS and will be available late summer. Dr. Fishman has added a preface to his book, and the CCC is pleased that this very readable and informative book will be made available again.

Peter A. Chevalier, Chairman

Education Committee

This report provides an update on selected activities of the committee. Committee members: A. Vander, Chairman, J.E. Randall, J. Michael, J. Spitzer, J. Bassengthwaighte, E. Feigl, P. Timaris, J.B. West, and J.C. Houk.

Credits for Continuing Medical Education

All publications and meetings of the APS are now potentially available for the attaining of CME credits by physicians. The first fruit of our recent accreditation was the obtaining of CME credits by 30 physicians at our APS Fall 1981 Meeting. The major tasks now are devising efficient ways of providing adequate pre- and post-tests for our materials and of publicizing both the published materials and meetings. These tasks are being overseen by J. Michael and J. Spitzer.

Dr. Spitzer has organized an effort to attract physicians to seek our credits at the various FASEB sessions in New Orleans. FASEB announcements bearing information on CME credits were distributed to medical staff offices of all New Orleans area hospitals, LSU and Tulane Medical Schools, affiliated teaching hospitals, and local medical societies. This is a pilot effort to see what sort of response we get; if successful in attracting physicians, we will set up a mechanism for publicizing offerings at all future FASEB and APS Fall Meetings, in addition to listing these offerings in the "Directory of Continuing Education for Physicians" published in the *Journal of the American Medical Association.*

AVMD, the company which markets our audiovisual materials, may take on the responsibility for developing, under the committee's supervision, the pre-and post-tests for these materials. The role of the original authors in generating questions for their own programs is also being explored.

Other ideas for interacting with physician groups have been suggested and are being explored; for example, should APS organize symposia in the nature of "pathophysiology" workshops to be held at medical schools (such workshops might be held, in succession, at several schools).

The Audiovisual Project

The entire series of slide-tapes dealing with "Peripheral Circulation" has been completed and is presently available. This brings to a close the production of new slide-tapes by the APS, at least for the present. We estimate that the deficit accumulated during the production of these programs, approximately $150,000, should be completely paid off within 3 years. AVMD

will bear the cost of producing the pre- and post-test items required for the awarding of CME credits for all the tapes.

Career Brochure

The new version of the careers brochure, supervised by D. Ramsay and P. Timaris, is now complete and should be available for distribution by the end of this year.

Programs on the Teaching of Physiology

The topic to be covered at the 1982 Fall Meeting is "Nonlecture Approaches to Teaching Cardiovascular Physiology," organized by A. Rovick and J. Michael.

The 1983 Refresher Course

The refresher course for the 1982 Fall Meeting (in San Diego) will be "The Microcirculation," organized by B. Duling.

Book Review Section for the *Physiology Teacher*

The committee is attempting to organize a more systematic approach to the publishing of book reviews in the *Physiology Teacher.* The aim of these reviews is to provide information concerning the educational value of books (or review articles) to students and teachers of physiology. Perhaps the most important type of review in this regard is one which covers multiple books in the field, comparing and contrasting their contents and approaches to the field, with particular emphasis on the audience for whom the books are intended and their particular strengths and weaknesses. The first of these reviews has been prepared in the area of cardiovascular physiology by J. Michael and provides an excellent prototype for future efforts. Anyone interested in contributing such a review should contact A. Vander.

It is also hoped that the various sections within the APS will become active in producing lists of reviews appearing in other journals as well as abstracts of review articles particularly valuable for either students or teachers. These list and abstracts will be published in the *Physiology Teacher.*

Arthur Vander, Chairman

Financial Development Committee

The charge to this committee, when it was set up three years ago, was to review the financial base of the Society, seek new sources of support, and introduce flexibility in financing by reducing dependence on income from dues. In pursuit of these goals, a number of changes and new programs have been instituted.

The members of the committee were immpressed that some of our members have to struggle to pay the current fixed dues, whereas others can pay this amount or more with little financial sacrifice. Another relevant fact is that an increasing number of members put their dues on grants or have them paid by the industrial concerns or universities for which they work. One step that has been taken is to provide the opportunity for voluntary contributions by members along with their annual dues, and each year for several years, the President has written a letter that accompanies dues notices encouraging members to contribute. The Society has also instituted early mailing of the dues notices with the request for early payment, because this gives the Society the use of the money and reduces administrative costs. Emeritus members, who receive all the benefits of membership without cost, have been asked for voluntary contributions, and they have given generously to the society. These changes have all worked well, and the voluntary contributions have been appreciable. One unsolved problem is the fact that most of the organizations that pay dues for members will not make contributions, and we have only one category of dues. This year, members were asked to consider a separate voluntary contribution if their dues were paid by institutions. The committee will consider this subject further and discuss the possibility of more than one category of dues for the American Physiological Society.

To encourage bequests to the Society, mechanisms have been set up to receive donations and items about contributions have been published at frequent intervals in *The Physiologist.* Some senior members of the Society have also been recruited to make personal contacts with potential donors, and the head of the Senior Physiologist Committee has agreed to help to the degree that this is possible. An announcement that the American Physiological Society would consider establishing living trust arrangements if there was enough interest was published in *The Physiologist,* and the Society has already been approached about one such arrangement.

Another important goal of the committee has been expansion and revitalization of the Sustaining Associate Program. Sustaining Associate members of the Society, who are usually but not always pharmaceutical concerns, instrument companies, or publishers, make an annual contribution of $500 or more to Society activities. The committee and the officers of the Society have organized an annual reception and meeting with representatives of the Sustaining Associates at the Federation Meeting in order to initiate a dialogue between the Sustaining Associates and the Society. Two meetings of this type have been held, and a third is to be held during the present Federation Meeting. As a result of discussions at these meetings, a standing Liaison Committee with industry has been established. It is hoped that this committee will provide a conduit for complaints, problems, and suggestions in relation to industry. The committee would welcome any ideas about additional approaches and ways to further strengthen the Sustaining Associates Program.

The Society has also initiated contacts with a number of foundations. One result of these inquiries has been the establishment in cooperation with the Kroc Foundation of a program to support the travel of young scientists to international physiological congresses. We are very pleased that this program has developed and are grateful to the Kroc Foundation for their support. However, it seems clear that the best approach to foundations and other potential funding sources is through individuals. The Society cannot afford a salaried development officer, but the Financial Development Committee has argued that a viable and in some ways desirable alternative is many parttime development officers. We urge anyone in the Society who is interested to explore possible sources of financial support. Of course, our main goal is to pick up support for ongoing activities so that the proportion of the budget that must be funded for membership dues is reduced. Many foun-

dations are only interested in new programs in which the Society has not been involved before. We do not exclude programs that increase our activities and hence our overall budget, but our interest is largely limited to sponsored activities for which the extramural support also covers part of the cost presently paid by the Society.

The Financial Development Committee works in consultation with the Finance Committee and Officers of the Society in considering ways to cut the present budget and to increase the efficiency with which currently available funds are being used. However, our primary job is seeking new and different sources of support. In this area, we welcome suggestions, assistance, and new ideas.

William F. Ganong, Chairman

Porter Development Committee

Committee members: E. Ison-Franklin, J.W. Manning, C.E. McCormack, J. Santos-Martinez, S. Solomon, W.N. Stainsby, and A.C. Barger and E.W. Hawthorne, Co-Chairmen.

The Porter Development Committee is now supporting its first Hispanic postdoctoral fellow Dr. Jose E. Garcia-Arraras of Puerto Rico, who received his Ph.D. in the laboratory of Dr. J.R. Pappenheimer, and is now studying in the laboratory of Dr. Nicole Le Dourain at the Institut d'Embryologie in the Centre National de la Recherche Scientific at Nogent-sur-Marne, France. The Committee is also continuing its support of two predoctoral fellows—Mr. Claude Simon working in John Fray's Laboratory at Hunter College and Ms. Cynthia Jackson in Luis G. Navar's laboratory at the University of Alabama School of Medicine. The Committee has also continued funding for the Atlanta and New Orleans consortia, as well a the Native American Summer Research Participation Program at Colorado State University at Fort Collins. We express our appreciation to the Harvard Apparatus Foundation for its continuing support of the Porter Development Program.

A.C. Barger and E.W. Hawthorne, Co-Chairmen

Program Executive Committee

The 1983 Spring Meeting of the Society will be held with the FASEB Meeting, Chicago, Illinois, April 10–15, 1983.

The themes for this meeting are neuroendocrine, calcium, and infectious diseases. The APS assigned neuroendocrine theme sessions will be organized by M. Susan Smith. The following will be proposed to FASEB as the Neuroendocrine Theme Sessions.

Day 1, A.M.

Endocrine Control of Appetite and Obesity (Symposium). Chairman: George Bray

Inhibin and FSH Secretion (Topic for submitted papers; to be programmed as slide or poster session)

Day 1, P.M.

Neuroendocrinology of Behavior (Symposium). Chairman: Donald Pfaff

Enkephalins and Regulations of Adrenal Medulla (Topic for submitted papers; to be programmed as slide or poster session)

Day 2, A.M.

Pituitary Hormone Synthesis, Processing and Secretion I (Symposium). Chairman: Joel Habener

Circadian Rhythms of Hormone Secretion (Topic for submitted papers; to be programmed as slide or poster session)

Day 2, P.M.

Pituitary Hormone Synthesis, Processing and Secretion II (Symposium). Chairman: Harold Gainer

Stress-Induced Hormone Release (Topic for submitted papers; to be programmed as slide or poster session)

Day 3, A.M.

Neuroendocrinology of Aging (Minisymposium). Chairman: Calib Finch

GnRH Receptor Regulation (Topic for submitted papers; to be programmed as slide or poster session)

Day 3, P.M.

Immunocytochemical Identification of Pituitary Cell Types (Minisymposium). Chairman: Gwenn Moriarity (Childs)

Adrenergic Receptors in the Hypothalamo-Pituitary Complex (Topic for submitted papers; to be programmed as slide or poster session)

There will be 19 APS approved Symposia and Special Sessions as recommended by APS Sections. The third in a series of History of Physiology Lectures commemorating the APS Centennial will be presented by A.P. Fishman.

The following sessions were approved by the Program Executive Committee on April 22, 1982 (one-half day session each unless noted).

1. Control of GFR by Contractile Elements in the Glomerulus
2. Cell to Cell Communications
3. The Role of Calcium as Second Messenger (This symposium was furnished to ASPET as an APS suggestion to the FASEB theme on calcium; organization of this session will be contingent on action taken regarding this theme)
4. Transport and Metabolism of H_2O Soluable Vitamins in Intestine and Kidney (AIN cosponsorship has been requested)
5. Aspects of Contraction of Gastrointestinal Smooth Muscle
6. Ion Transport Processes in Apical Membranes of Epithelia
7. Ion Transport Mechanisms in Basolateral Membranes of Epithelia
8. Nutrition and Lactation (AIN cosponsorship has been requested)
9. Information Retrievable from Ventricular Pressure Volume Relationship
10. Automatic Control Electrophysiology
11. Myocardial Hypertrophy
12. Regulation of Vascular Smooth Muscle (This is planned as a Poster-Discussion honoring D. Bohr)
13. Neuroendocrine Control of Insulin Secretion
14. Area Postrema (2 sessions)
15. Body Composition and Exercise
16. Current Problems in Temperature Regulation and Exercise (A special session to be held in the evening in memory of Sid Robinson)
17. Biologically Important Arachadonic Acid Metabolites (3 sessions sponsored by the Clinical Physiology Sub-Committee of the APS Publications Committee)
18. Biological Effects of Nuclear Weapons (It was agreed that APS would forward the proposal to FASEB for intersociety sponsorship in an evening session; the session would be organized by Arthur Vander and Clifford Barger)
19. Electrophysiology of Secretory Cells

The FASEB Theme assigned to APS for the 1984 Spring Meeting is Regulatory Mechanisms and will be organized by Eugene Yates.

The Program Committee welcomes suggestions for future programs. Please forward these suggestions through the members of the Program Advisory Committee.

Cardiovascular: Douglas M. Griggs, Jr.
Cell and General Physiology: Robert B. Gunn
Clinical Physiology: Francois M. Abboud
Comparative Physiology: Donald C. Jackson
Endocrinology and Metabolism: M. Susan Smith
Environmental, Thermal and Exercise: Carl Gisolfi

Gastrointestinal Physiology: Len Lichtenberger
Membrane and Transport: James A. Schafer
Muscle Physiology: M.J. Seigman (tentative)
Nervous System: Richard K. Orkand
Neural Control and Autonomic Regulation: J.W. Manning
Renal Physiology: David G. Warnock
Respiratory Physiology: Albert J. Berger
Water and Electrolyte Homeostatis: Leonard Share

Franklyn G. Knox, Chairman

Public Information Committee

The Public Information Committee of the American Physiological Society held its second meeting on January 18, 1982, at the APS Headquarters. Those attending were Drs. Sever, Cassidy, Moriarty, Reynolds, and Kafka.

Chairperson M. Kafka read the minutes of the first meeting (January 19, 1981).

Old Business

Dr. Kafka congratulated the FASEB Public Affairs Information office on its coverage of the American Physiological Society at the 1981 FASEB Spring Meeting.

Dr. Kafka reported on her discussion with PBS regarding the feasibility of doing programs for television. It was suggested that the committee would need an agent to try to place programs we would make on series such as NOVA, since television time (approximately $100,000 per half hour) is beyond our budget. These expenses would, of course, be in addition to expenditures for producing the programs themselves. The committee agreed that the venture was outside our budget.

It was suggested that we ask chairpersons of Physiology Departments or other departments whose chairpersons are physiologists if they have made videotapes which could be used, in part or in whole, for brief public information spots on television. If so, we might offer copies of them, gratis, to television stations in return for the benefit we receive from their airing. Drs. Cassidy and Kafka will design a questionnaire for these chairpersons to try to assess whether such tapes exist, and if so, where and on what subjects. Dr. Sever will offer his suggestions for the questionnaire to Drs. Cassidy and Kafka.

Dr. Reynolds reported that the Speakers Bureau which the committee had suggested was also suggested by others and that Liaison Committee with Industry has been formed to discuss the formation of a Speakers Bureau for APS. Representatives from other APS committees will enter into these discussions. The committee decided accordingly to wait until the initial liaison is formed and then to offer its help.

At the request of the committee, Dr. Moriarty brought a copy of the *Readers Digest* series "I am Joe's or Jane's Liver, Heart, or Other Organ." The committee asked him to investigate whether *Readers Digest* would give us 1,000 copies of the series to distribute to high school teachers to use in teaching additional periods of physiology in high school science courses. The committee also suggested that an APS member might amplify the series by lecturing to the class on the subject covered or by answering questions that the teacher or students might ask.

Pilot Study Assays

Dr. Sever will draft an announcement suggesting offers of volunteer pilot study assay services under the aupices of the Public Information Committee. The announcement will appear in *The Physiologist* to see if there are responders who offer to do the assays and requesters who want to have them done.

Information Block on Abstract Form

The committee suggests that the APS Abstract Form carry a new block on its front face asking for a sentence indicating whether the study, *1*) has potential as a public information release and *2*) what the basis is for public interest in the study. The purpose of the information in the block is to point out the abstracts that should be reviewed by the Committee on Public Information for potential use in press releases or FASEB Feature Service articles, eliminating reading of all abstracts submitted and at the same time identifying authors who see public information potential in their work and who might be more amenable to doing the lay translation for press releases when requested. The former would help the Public Information Committee in its search for material for the lay media, and the latter would cut down on the approximately 75% nonresponse rate confronting the Public Affairs Office requests for lay translations.

Centennial Celebration

At the committee's request, Dr. Reynolds described the latest plans of the Centennial Celebration Committee. The Public Information Committee has begun to discuss the plans in an effort to see how we can best assist in planning for the Centennial celebration.

Self-Quiz in Physiology

The discussion begun at the first committee meeting of a Question-and-Answer column for newspapers was continued by the committee. Dr. Sever suggested a self-quiz in physiology as a format with explanations at the end of the quiz of each of the multiple choice answers. The committee will send a sample quiz to the Senior Physiologists Committee, requesting that they compose similar quizes on physiological systems. The committee will then send to some papers a group of quizes and ask them if they would like to use them.

Marian Kafka, Chairperson

Publications Committee

1981 saw the continued strengthening of the journals of the American Physiological Society. The reorganization of the journals continues to encourage innovations such as rapid communications and the Modeling Methodology Forum, which appeared for the first time this year. With the smaller journals, each the direct responsibility of an Editor, the journals have improved in many ways and the resolution of any problems that surface occurs more rapidly.

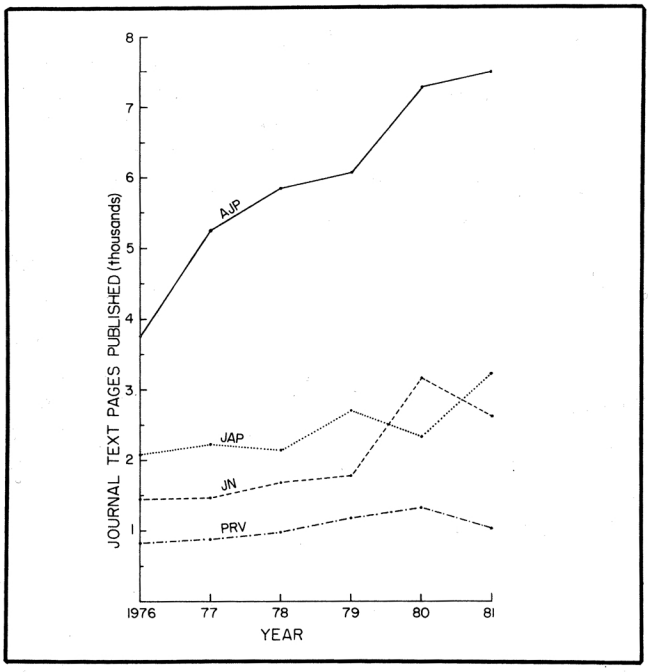
A *Handbook of Physiology* and two volumes in the Clinical Physiology Series were completed in 1981. An extensive series of Handbooks are in preparation. In addition to these traditional projects, the Society's book plans include several exciting new ventures: a book de-

rived from previously published reviews, a reprinting of a classical book, a timely volume on the perception of pain in animals, and several books related to the celebration of the Society's centennial.

The number of new manuscipts received for the journals increased by 6% (175 manuscripts) in 1981, similar to the 6%, 155 manuscript increase in 1980. The number of new manuscripts received for the *Journal of Neurophysiology* increased by 16% despite a new competing journal. The increase in the amount published since the journals were reorganized continues.

	Amount Published	
	Articles	Text Pages
1976	1,031	8,121
1977	1,131	9,825
1978	1,177	10,665
1979	1,287	11,705
1980	1,435	14,138
1981	1,560	14,352

162 manuscripts and 1074 pages (+11% pages) more were published in the consolidated *American Journal of Physiology* and *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology.* However, 37 manuscripts and 860 fewer pages (−19% pages) were published in the *Journal of Neurophysiology* and *Physiological Reviews.* Overall the number of articles increased by 125 (9%) and the number of pages increased by 214 (2%).



In 1981 the journals were operated in the black. Total income (including interest, dividend, royalty, and miscellaneous income) was $2,401,774. Total expenses were $2,394,946.

Books

One *Handbook of Physiology* (*Motor Control*) and two books in the Clinical Physiology Series (*Disturbances in Neurogenic Control of the Circulation* and *New Perspectives on Calcium Antagonists*) were completed in 1981. In addition to an ambitious Handbook program a variety of new book projects have been undertaken.

1. *Excitation and Neural Control of the Heart,* edited by M.N. Levy and M. Vassalle. This book is composed of 13 invited reviews, which have been carefully updated, that appeared in the *American Journal of Physiology: Heart and Circulatory Physiology.*

2. *Pain Perception in Animals,* edited by R.L. Kitchell and H.H. Erickson. It is planned that a symposium, jointly sponsored by AMVA, APS, and ASPET, held at the spring 1982 meeting in New Orleans be developed into a book to be published by the Society.

3. *Circulation of the Blood: Men and Ideas,* edited by A.P. Fishman and D.W. Richards. With the enthusiastic endorsement of the Centennial Celebration Committee the Publications Committee agreed that this book, originally published in 1964 and now out-of-print, should be reprinted and sold at cost.

Handbook of Physiology

The second volume, entitled *Motor Control,* in the section on the nervous system was published in 1981. Revisions of the first three sections of the *Handbook of Physiology* (nervous system, cardiovascular system, and respiratory system) are in progress along with a new volume on skeletal muscle. The preparation of a section on general and cellular physiology was discussed during the year, and a group of consultants met with the Publications Committee to consider these books.

Volume II of Section 1, *The Nervous System, Motor Control,* edited by V.B. Brooks, was published in December. It sells for $245 to nonmembers and $196 to members of the Society.

In 1981, 2,724 copies of the Handbooks were sold, providing an income of $136,796 (1980 income was $224,685). The cost of the series from 1959 through December 1981 totaled $3,272,509; the income was $3,063,091. The total deficit is $209,418. The cost in inventory is $716,374.

Clinical Physiology Series

Two books in the Clinical Physiology Series were published in 1981. *Disturbances in Neurogenic Control of the Circulation,* edited by F.M. Abboud, H.A. Fozzard, J.G. Gilmore, and D.J. Reis, was completed in August. *New Perspectives on Calcium Antagonists,* edited by G.B. Weiss, was completed in October. Each book sells for $38.50 to nonmembers and $31.00 to members of the Society.

A symposium on high-altitude physiology, organized by J.B. West and S. Lahiri, is planned for the fall 1982 meeting in San Diego. It is hoped that the material presented can be developed into the next book in the Clinical Physiology Series.

The Council of the Society reorganized the section on Physiology in Clinical Science into a Subcommittee on Clinical Sciences, a subcommittee of the Publications Committee. F.M. Abboud agreed to stay on as Chairman, along with those members who are involved in organizing symposia in this series.

In 1981, 1,952 copies of the first six books in the series were sold, providing an income of $48,891 (1980 income was $45,856). The cost of the series through December 1981 was $183,516; the income was $186,800.

H.E. Morgan, Chairman

Committee on Senior Physiologists

This year, 1982, marks the first time the Committee has functioned without any of its charter members. Edward Adolph and Hallowell Davis were among the early group that handled the Senior Physiologists. (The others were Bruce Dill, Hy Mayerson, Maurice Visscher, and Philip Bard.) Not only are we operating without Adolph and Davis, we also are rocking along without Horace Davenport, Sid Robinson [who died in January], and Ladd Prosser. We are grateful for their past committee activities and wish them enjoyment of their newfound leisure.

The new committee added names of 49 members born in 1912 to its roster of senior physiologists. We also promoted 21 members to 80-year-old status. The former group receive form letters with room for personal greetings on their seventieth birthdays. The latter group receive Beaumont cards with a personal message.

A guideline of procedures was developed last year, when we had the benefit of the older committee members' experience on the handling among the committee of the distribution of names and of greetings. The workability of these procedures will be assessed in the Fall after the current committee has tried them.

Louise H. Marshall, Chair

APS Representative's Report on the Council of Academic Societies

The 1982 Interim Meeting of the council of Academic Societies was held at the Washington Hilton Hotel on January 19, 1982. The meeting focused on the theme of the research partnership between the federal government and the academic medical center and was highlighted by discussions with Congressional and Executive Branch staff. These staff members were recognized as an effective fourth branch of government inasmuch as they have an outstanding opportunity to effect legislation in its developmental phase. The personal dialogue between members of the council of Academic Societies and the staff was open, frank, and enlightening. The dialogue not only shed light on current issues but also paved the way for continued contact.

A strong message from the Congressional and Executive Branch staffs was that the 1983 NIH Budget is rumored to be increased by 2.7%. Although it is recognized that this will not cover inflation and other costs impacting on research, it is much better than the cuts being suffered by other areas, including those related to health delivery. The main priorities of the Reagan administration, to contain total spending while increasing military spending, are not likely to be shifted on the basis of our input. On the other hand, a resetting of priorities within health and NIH itself would be welcomed by the staff.

It was noted that real leadership in the health area in Congress is lacking. We may wish to assist a Congressional figure in strengthening this leadership.

It was conceded that there is an unrealistic expectation of support from the private sector to fill the gaps left by decreased federal funding. Nonetheless, the administration strongly supports this approach.

In regard to our own lobbying activities, we were instructed to be more open with greater emphasis on self-policing and accountability. We have been clearly hurt by the recent disclosures of fraud and abuse in the scientific community. We have been perceived as insufficiently responsive to these abuses.

Although organizations have a definite role in lobbying for biomedical research, more local grassroots input is necessary. Individual faculty members need to express the consequences of reductions in the funding in specific ways to their Congressional representatives. Perhaps patients who are grateful for the fruits of research might be able to assist in this grassroots input.

The effects of decreased funding on institutions per se seems to fall on deaf ears. The stronger case is the ultimate effects of biomedical research on the welfare of the population as a whole.

The small-business set-aside legislation made a good case in point. The biomedical research community did not fare well in the hearings, since we were perceived as arrogant and inappreciative of the support of biomedical research as well as insensitive to the needs of the small-business community. In regard to the legislative assistants, in many cases the legislative assistant for health affairs in an individual Representative's office was unaware of the impact of the small-business set-aside legislation on the availability of funds in NIH for peer review as well as the erosion of the integrity of the peer review system itself.

It is recognized that the legislative assistants as well as the members of the Council of Academic Societies experience a frequent turnover, and therefore establishing these contacts must be renewed on a continued basis. It was recommended that the legislative assistants visit research institutions and learn first hand of the benefits of biomedical research and of the consequences of decreases in funding. The face-to-face dialogue should facilitate telephone converations of important issues as they arise. Hopefully, this can develop into a two-way street in which these young staffers may come to leaders in the academic community for advice on particular pieces of legislation during the developmental phase.

Franklyn G. Knox

APS Sections

Section on Water and Electrolyte Homeostasis Statement of Organization and Procedures

ARTICLE I. *Name*

The name of this organization is the Section on Water and Electrolyte Homeostasis of the American Physiological Society.

ARTICLE II. *Purpose*

The purpose of this organization is *1*) to advise the American Physiological Society on matters of interest in this area, and *2*) to assist the American Physiological Society in organizing and presenting scientific sessions, symposia, and other programs of interest to physiologists in this area.

ARTICLE III. *Membership*

Regular Membership is open to any member of the American Physiological Society who signs a statement declaring the wish to be a member. To accomplish this, the entire membership of the American Physiological Society will be polled during the first year that this Statement is in effect. Thereafter, only new members of the organization will be polled to ascertain their interest.

Intersociety Membership is open to those with an interest in water and electrolyte homeostasis, but who are not members of the American Physiological Society. Intersociety Members share all of the rights and responsibilities of Regular Members.

Any member of a FASEB Society other than APS may become a member of the Section upon written request. Others with an interest in water and electrolyte homeostasis may become a member of the Section upon written recommendation by two regular members of the Section and approval by the Steering Committee. Documents supporting the nomination for membership should be sent to the Secretary.

ARTICLE IV. *Officers*

SECTION 1. *Steering Committee.* The responsibility for management and supervision of the affairs of the section on Water and Electrolyte Homeostasis shall be vested in the Steering Committee composed of three Councillors.

SECTION 2. *Councillors.* There shall be three Councillors elected to the Steering Committee, each for a term of three years, but with only one being selected in any one year. The Councillor in his/her second year will serve as Secretary, and the Councillor in his/her third year will serve as Chairman.

SECTION 3. *Program Committee Representatives.* A program Advisory Committee Representative (or representatives) shall be elected for a term of three years. The number of such representatives will be as authorized by the APS Council. The representative to the APS Program Advisory Committee shall also be a Councillor. If more than one such representative is authorized, one of these shall be identified as the Councillor.

SECTION 4. *Election of Officers.* One Councillor shall be elected each year, and the Program Committee Representative shall be elected every three years.

Two nominations shall be made annually, as appropriate, by the Nominating Committee to fill forthcoming vacancies. The names of the nominees shall be announced by mail to the members two months in advance of the annual meeting. Additional nominations may be made by three or more members submitting the name of a candidate who has agreed in writing to serve if elected. Nominations must be submitted by February 1 of the election year.

Election of officers shall be by mail ballot sent to all members concurrently with the announcement of the annual meeting.

ARTICLE V. *Standing Committee*

SECTION 1. Nominating Committee. The Chairman, in consultation with the other members of the Steering Committee, will appoint annually two members to serve with a Councillor (serving as Chairman) as the Nominating Committee. The Committee nominates two members as candidates for election to each office.

ARTICLE VI. *Duties of Officers*

SECTION 1. The Program Committee Representative(s) is(are) responsible for performing the functions defined by the Society Operational Guide for members of the Program Advisory Committee. In addition, the Program Committee Representative will have the responsibility for developing a liaison with the Program Committee Representatives of other sections that may have an interest in water and electrolyte homeostasis, for the purpose of avoiding programming conflicts and for developing joint programs that are of mutual interest.

SECTION 2. The Chairman, in consultation with the other members of the Steering Committee, will annually choose topics to be submitted to the APS Program Committee for annual meetings of the Society. They will also choose a person who will organize each approved symposium and be responsible for its presentation.

SECTION 3. The Chairman, in consultation with the Steering Committee, will suggest to the Publications Committee of the American Physiological Society section members for service on the editorial boards of publications of the society which are particularly concerned with water and electrolyte homeostasis, e.g., Endocrinology and Metabolism, Heart and Circulatory Physiology, Regulatory, Integrative and Comparative Physiology, and Renal, Fluid and Electrolyte Physiology.

SECTION 4. The Chairman may appoint committees that are necessary for the proper conduct of the affairs of the Section.

ARTICLE VII. *Dues*

Dues will not be assessed.

ARTICLE VIII. *Meetings*

The Section on Water and Electrolyte Homeostasis will meet at least once a year and at other times determined by the Steering Committee. Members must be notified in writing at least one month before the meeting. Meetings are for transacting the business of the Section on Water and Electrolyte Homeostasis and are governed by Roberts Rules of Order Newly Revised.

ARTICLE IX. *General*

SECTION 1. *Amendments.* Amendments to these procedures must be proposed in writing to the Steering Committee by five members at least two months before the annual meeting. The proposal must then be sent to the members at the time of the announcement of the annual meeting. An amendment requires the approval of two-thirds of the entire membership for adoption.

SECTION 2. *Quorum.* The quorum required for all membership business meetings is no less than 30 percent of the total members of the Section.

ARTICLE X.

Nothing in this Statement of Organizational Procedures shall be construed as contradictory to the Constitution and Bylaws or Operational Guidelines of the American Physiological Society.

Statistics on APS Membership
(As of May 1982)

Total Membership 6,035

Distribution by Employment*

	No.	%
Medical Schools	3,492	65
Physiology Departments	(1,827)	(34)
Other Preclinical Departments	(452)	(08)
Clinical	(1,161)	(22)
Administration	(52)	(01)
Hospitals and Clinics	237	04
Veterinary Schools	104	02
Dental Schools	48	01
Public Health and Graduate Schools	226	04
Undergraduate Schools	416	08
Commercial Companies	105	02
Government	333	06
Institutes and Foundations	221	04
Private Practice	49	01
Other, Emeritus or Inactive	123	02

* 5,354 Respondents.

Distribution by Earned Degree*
(Includes 650 individuals with multiple doctorate degrees)

	No.
Ph.D.	3,525
M.D.	2,087
D.V.M.	140
D.D.S. and other	30

*5,132 Respondents.

Principal Type of Work*

	%
Research	69
Teaching	16
Administration	08
Clinical	06
Other	01

*5,348 Respondents.

Distribution by Primary Speciality*

	%
Cardiovascular	20
Neurophysiology	13
Endocrines	10
Respiration	09
Electrolyte and Water Balance	05
Renal	05
Muscle and Exercise	05
Gastrointestinal, Food and Nutrition	04
Environmental	04
Cellular and Tissue	04
Blood	03
Comparative	02
Energy Metabolism and Temperature Regulation	02
Pharmacology	02
Reproduction	02
All Other Categories (None above 1%)	10

*5,236 Respondents.

Distribution by Age*

70+	497
60-69	785
50-59	1,612
40-49	1,748
30-39	1,133
20-29	121

*Optional personal data (numbers represent total respondents).

Distribution by Sex*

Female	592
Male	5,084

*Optional personal data (numbers represent total respondents).

States in U.S. With More Than 100 Members*

California	647	North Carolina	143
New York	610	Florida	151
Pennsylvania	331	New Jersey	139
Maryland	323	Missouri	131
Texas	314	Connecticut	128
Massachusetts	292	Virginia	126
Illinois	270	Minnesota	113
Ohio	215	Indiana	107
Michigan	179	Washington	104

*50 States plus Puerto Rico and Virgin Islands.

Distribution by Racial Background and Heritage*

American Indian or Alaskan	6
Asian or Pacific Islander	197
Black	29
White	3,838
Hispanic Heritage	81

*Optional personal data (numbers represent total respondents).

APS North American Membership

United States	5,663
Canada	235
Mexico	9

Canadian Provinces With 5 or More Members

Ontario	98
Quebec	67
British Columbia	21
Alberta	20
Manitoba	16
Nova Scotia	7
Saskatchewan	6

Other provinces represented
- New Brunswick
- Newfoundland
- Yukon Territory

APS Membership Outside North America

Countries with 5 or more members

Japan	22
Germany, Federal Republic	21
United Kingdom	20
Switzerland	17
France	10
Israel	9
Italy	9
Australia	7
Denmark	7
Sweden	6
Venezuela	6
Argentina	5
Belgium	5
Norway	5
Spain & Canary Islands	5

Other countries represented

Poland	Hong Kong
Greece	Iceland
Netherlands	India
Peru	Kuwait
South Africa	Lebanon
Hungary	New Guinea
New Zealand	Panama
Nigeria	Paraguay
Austria	Peoples Rep. of China
Brazil	Portugal
Chile	Rhodesia
Dominican Republic	Saudi Arabia
Finland	Taiwan Rep. of China
	USSR

Membership Status

Regular	4,445
Emeritus	539
Honorary	11
Corresponding	81
Associate	702
Student	242
Total	6,020

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FOLKOW, BJORN
GOTEBORG UNIVERSITY
FYSIOLOGISKA INSTITUTE
MEDICINARGATAN 11
S-400 33 GOTEBORG, SWEDEN

Deaths Reported Since the 1981 Fall Meeting

Bach, L.M.N.
Reno, NV
1/05/82

Benson, O.O., Jr.
San Antonio, TX
2/03/82

Bresler, E.H.
New Orleans, LA
5/21/81

Brown, D.E.S.
Woods Hole, MA
6/11/80

Dow, P.
Augusta, GA
12/17/81

Eckstein, G.
Cincinnati, OH
9/23/81

Enns, T.
La Jolla, CA
1/20/82

Fenton, P.F.
Barrington, RI
1/24/82

Fowler, W.S.
Rochester, MN
2/23/82

Gell, C.F.
Bethesda, MD
12/26/80

Glass, G.B.J.
New York, NY
11/81

Grollman, A.
Dallas, TX
1/80

Gross, E.G.
Iowa City, IA
11/19/81

Grossman, M.I.
Los Angeles, CA
5/26/81

Haupt, R.E.
Ames, IA
9/23/81

McCubbin, J.W.
Shaker Heights, OH
1/22/82

Piliero, S.J.
New York, NY
8/81

Read, W.O.
Vermillion, SD
8/12/81

Reissmann, K.R.
Novato, CA
12/21/81

Ricketts, H.T.
Chicago, IL
11/03/81

Robinson, S.
Bloomington, IN
1/08/82

Scott, J.B.
East Lansing, MI
1/05/82

Tipton, S.R.
Knoxville, TN
1/19/82

Werthessen, N.T.
Rehoboth, MA
10/10/81

Wiedeman, M.P.
Philadelphia, PA
4/18/82

N E W S

Edward F. Adolph to Ladd Prosser:

I enjoyed your recall of our work together on the first Education Committee of APS. One of the Committee's projects was your special effort: the setting-up of "workshops on teaching of college physiology." I remember the initial program held in Connecticut in two weeks of 1955. There were demonstrations of laboratory experiments, discussions of teaching materials, evaluations of student responses. Questions were raised about the objectives of physiology courses in colleges. Your selection of participating teachers, both leaders and followers, suited the aims and methods of the planned sessions. Especially prominent was the repeated discovery by teachers that their colleagues in other colleges were meeting identical problems. I found a letter (1955) signed by 29 of the college teachers, expressing their appreciation of the arrangements for the workshop; no doubt you received a similar letter.

In addition to subsequent summer workshops, an outcome of the 1955 project was the arrangement of the "Summer research training program for college teachers" which lasted five years. NIH and NSF were highly responsive to our requests for financial aid on behalf of these projects.

Nowadays, like you I enjoy my daily sojourn in the department and school where colleagues and graduate students are seen and heard. Your alma mater (University of Rochester) has matured.

University of Rochester Medical Center
Rochester, NY 14642

Harold M. Kaplan to Ladd:

Just to update activities as you requested, I am still working in retirement at Southern Illinois University. I am Acting Director of our Vivarium (Central Animal Quarters) until July 1, 1982 and I also give two courses for the medical preparatory program run by our School of Medicine.

Our laboratory was closed in 1977 when I retired, but I have been able to continue occasional publishing in cooperation with other investigators. I am coauthoring a nonclinical textbook of endocrinology which should be put out by the CRC Press in about six months.

Overall, I am currently busier in teaching and administration and less able to control my scheduling than was the case prior to retirement. When these activities cease to be fun, it will obviously be time to retire.

Southern Illinois University at Carbondale
Carbondale, IL 62901

Roy O. Greep to Ladd:

I reached retirement age in '72, was given two additional years at academic full time by the Harvard Medical School, then worked 2½ years full time on a Ford Foundation project. For the past five years I have been on full-time retirement and still feel guilty being out to pasture. Any hazard of dull cares and boredom has thus far been avoided by heading the annual Laurentian Hormone Conference and editing the proceedings, a year-round job; teaching histology (3 weeks) each spring at St. George's University School of Medicine in Grenada (a fun thing teaching the most highly motivated students I have ever encountered); serving one-half day each week as a consultant to the Pediatric Surgery Endocrine Division at the Massachusetts General Hospital; and heading the Finance (fund raising) Committee for the International Society of Endocrinology, a tough assignment these days but *viva le hormones,* they are "in" and the till keeps ringing merrily. That plus lecturing here and there as visiting professor, editing the Reproductive Physiology series of the *International Review of Science* doing book reviews and sporadic evaluation of grant applications, writing letters of recommendation, etc., fill my days to no particular end creatively. Retirement at Harvard workwise is the equivalent of being fired with honor—you're through period!

Harvard Medical School
Boston, MA 02115

Fifty-Year Members and Year of Election

E.F. Adolph, 1921
A.M. Baetjer, 1929
B.O. Barnes, 1932
J.W. Bean, 1932
R.J. Bing, 1922
H. Bourquin, 1925
T.E. Boyd, 1925
E. Bozler, 1932
E.B. Carmichael, 1931
McK. Cattell, 1923
K.K. Chen, 1929
H. Davis, 1925
C.A. Dragstedt, 1928
J. Field, II, 1931
M.H. Friedman, 1929
C.L. Gemmill, 1928
A.S. Gilson, Jr., 1927
P.O. Greeley, 1931
E.M. Greisheimer, 1925
H. Grundfest, 1932
H.K. Hartline, 1929
A. Baird Hastings, 1927
J.M. Hayman, Jr., 1928
R.C. Herrin, 1932
A.B. Hertzman, 1925
M. Hines, 1932
H. Hoagland, 1932
C.B. Huggins, 1932
E. Jacobson, 1929
J.S.R. Johnson, 1925
F.T. Jung, 1930
N. Kleitman, 1923
T. Koppanyi, 1924
E.M. Landis, 1928
A. Lieberman, 1931
H.S. Mayerson, 1928
E.S. Nasset, 1932
H.C. Nicholson, 1932
S.E. Pond, 1924
A.C. Redfield, 1919
S.R.M. Reynolds, 1932
P. Reznikoff, 1927
C.P. Richter, 1924
D.M. Rioch, 1931
J.J. Sampson, 1932
C.F. Schmidt, 1929
F.O. Schmitt, 1930
S. Soskin, 1930
I. Starr, 1929
E.U. Still, 1928
M.L. Tainter, 1929
S.S. Tower, 1932
M.B. Visscher, 1927
J.T. Wearn, 1921
R.W. Whitehead, 1932
L.C. Wyman, 1927

Francis O. Schmitt to Ladd:

Some two years ago, after completion of the work I had been doing on the cerebral cortex, I became impressed with the great desirability of applying to neuroscience the concepts and techniques, particularly those of recombinant DNA technology, to neuroscience. Therefore, in the fall of 1980, with the invaluable aid of a planning committee comprised of leaders in molecular genetics, a week-long conference was organized and from May 2–8, 1981, the conference was held at Woods Hole, MA. Tutorial lectures on the concepts and techniques of molecular genetics were followed by lectures on applications of these concepts that have already been made, to endocrinology and to neuroscience.

Manuscripts representing the proceedings of this conference are in press, having been edited by Drs. F.E. Bloom, S.J. Bird, and myself. The book, entitled *Molecular Genetic Neuroscience,* is expected to appear in the spring of 1982. Meanwhile, I have been considering which of the many exciting aspects of the subject might be appropriate for further study by me.

Neurosciences Research Program
165 Allandale St.
Boston, MA 02130

Wilbor O. Wilson to Ladd:

Thank you for your enquiry concerning my post-retirement activities. I now have time to watch the garden grow. Retirement also permits me to spend more time with my wife. Although our children are grown up, the grandchildren reminds us what it was like to be young, albeit in a different age of over three score years ago.

One of my concerns is to know what to do with mounds of data from experiments the results of which were inconclusive. I hope that some of my former students will be able to solve some of the unanswered questions in the area of environmental physiology, where further research is needed.

Last year in February I was invited to give a paper at the International Symposium on Animal Production in the Tropics. At this meeting held at the University of Gezira in Wad Medani, Sudan, I was able to visit with ASP member, G. Edgar Folk, Jr. of the University of Iowa.

In December I attended a Satellite Symposium on Avian Endocrinology held in Tokyo. This afforded me a chance to visit former students and co-workers. The meeting preceded the 9th International Symposium on Comparative Endocrinology in Hong Kong. Dr. B. Lofts and his committee are to be complimented on doing an excellent job of arranging and being the host of the Symposium. I was especially impressed by Dr. B. Scharrer's lecture on "Neurosecretion—the development of a concept." I would recommend that our younger colleagues and their students read the paper when it becomes available in the *Proceedings* that are due to be published later.

Dept. Avian Sciences
University of California
Davis, CA 95616

S.B. Barker to Arthur Otis:

It was a genuine pleasure to get a personal note from you together with the formal greeting from The American Physiological Society Committee on Senior Physiologists in recognition of my having reached the Biblical goal of seventy. After retiring as first dean of what was then our newly established Graduate School, I have been helping to spare younger members of the Department of Physiology and Biophysics by serving as director of this graduate program, under Dr. Jimmy D. Neill, departmental chairman. I also participate in a variety of university activities, such as our Institutional Review Board for Human Use in Research, our Center for International Programs, and a variety of fine and performing arts activities. I find myself still drawn to the Federation meetings, as well as Thyroid and Endocrinology.

University of Alabama in Birmingham
University Station
Birmingham, AL 35294

Leland C. Wyman to Edward Adolph:

I am learning to be a Dane (including a little of the language). I have not done anything with physiology since I retired in 1962. But I have published five books and a number of papers on my other interest, Navajo Indian ceremonialism and art (sand paintings) during the 21 years of retirement just closing, and I have another book and a 60-page article in the works now, requiring more or less editorial attention. The book, which will be published in a big series on Southwestern Indian arts by the School of American Research in Santa Fe, New Mexico, is called *Southwest Indian Drypainting,* and it will be my swan song and I hope my masterpiece in this field. The article is on the Navajo Ceremonial System and is either now out or coming shortly in the new *Handbook of North American Indians.*

I was much amused by Horace Davenport's remarks about Arturo Rosenblueth in his article on Walter Cannon in the current *Physiologist.* I went through that period, so I can appreciate them. Fred Pratt and I used to go to Cannon's weekly seminar at the Harvard Medical School, before Rosenblueth came and while he was there. How well I remember them all, Dr. Porter, Alex Forbes, Joe Aub, Phil Bard, Hal Davis, Ray Zwemer, Rosenblueth, and of course Walter Cannon, who was a special friend of Fred Pratt (as you know, at that time I was teaching physiology at the Boston University School of Medicine.

Vissingsgade 36
64 Sonderborg, Denmark

William D. Neff to Roy O. Greep:

I am still active in research and graduate teaching. Since I plan to retire during the next year, I am not accepting any new graduate students to work in my laboratory, but I have three who are writing their dissertations and three other students working partime.

Center for Neural Sciences
Indiana University
Bloomington, IN 47401

Broda O. Barnes to Louise Marshall:

Nothing exciting about me. When I lost Mrs. Barnes last October to pancreatic cancer, I retired to confine my time to writing and research. I am now at Rush Medical College doing some writing but hope to soon get some research started on diabetes. Preliminary work has indicated that the complications of diabetes are due to an accompanying thyroid deficiency. I must run this down. Only advice to my young colleagues is, "It is better to wear out than rust out."

2838 W. Elizabeth St.
Fort Collins, CO 80521

Morris B. Bender to Louise:

I am curently practicing and teaching neurology to residents at the Mount Sinai School of Medicine. I am continuing my interests in scientific writing, at the rate of approximately three publications per year. I am enjoying my work, especially discovering correlations between clinical findings and recent physiologic observations. In more specific terms, my long-time interest is in the vestibular-oculomotor system and perception. I retired as the Director and Chief Neurologist of Mount Sinai Hospital and am now the Henry P. and Georgette Goldschmidt Professor of Neurology.

Mount Sinai Medical Center
New York, NY 10029

Nathan R. Brewer to Louise:

I retired in 1969 from my responsibilities as director of the animal quarters at the University of Chicago. I held that position for 24 years, having returned to my alma mater after 8 years as a veterinary practitioner. I was a member of the board of the NRC Institute for Laboratory Animal Science and was instrumental in establishing the certification of animal caretakers through course work and examination. Even though I am now professor emeritus of physiology, I'm still executive secretary of the Illinois Society for Medical Research, and I still do a modest amount of consultation work for laboratory animal facilities.

The last history of the ILAR was published in Laboratory Animal Science, one of a series of articles on the history of the animal care movement. It was a special edition—appeared within the last year. . . .

I have developed an interest in what I call laboratory physiology. I get excited over differences between animals—physiologic differences, that is. I do lots of reading, but when a difference between species is mentioned my heart beats a bit faster and I make a note of the item and try to follow it up. Fortunately it does have *some* value. For instance the forthcoming 4-volume work on the laboratory mouse has a chapter on physiology, and most of it is my work. I also contributed to the chapter on physiology in the 2-volume work on the laboratory rat. . . . The books are published by the American College of Laboratory Animal Medicine (and Academic Press).

There is a group called the American Society of Laboratory Animal Practitioners (I'm a life member of it) that puts out a house organ type release called *Synapse*. I have something in almost every issue of it—mostly physiologic items—interpretations of differences between species.

I don't play as much chess as I'd like to, but I do play postal chess. I also still like to dance. . . .

Illinois Society for Medical Research
5526 S. Blackstone Ave.
Chicago, IL 60637

Frances A. Hellebrandt to Louise:

The APS 80th birthday greetings released a flood of recollections about those distant days in Walter Meek's department at Wisconsin.

I have been living in a good multi-level retirement center for six years and have all the freedom and independence I can manage. It is a perfect milieu in which to study the natural history of the aging process. I spend a great deal of time in the Convalarium where I can observe the ways in which we meet the exigencies of the terminal years of the life cycle. I hold the courtesy title of "Research Associate" and have written three geriatric papers since coming here to live. I have very helpful connections with the Social and Medical Sciences at Ohio State University, primarily in the field of aging. This fall I plan to give four seminars at the Village on "Healthy Living Patterns for the Elderly."

I continue in relatively good health. Percy Dawson taught me that the hardest thing about being a physiologist is to live physiologically. I have never forgotten that—ride my stationary bike for eight miles daily before breakfast, walk regularly and am introspective and analytical about the limitations imposed by a rapidly deteriorating vision and an impaired proprioception. Nobody writes about the deterrents and hazards of exercise in the old-old, but I am living them and monitoring my own physical performance.

A physiologist has a wealth of interesting phenomena to observe in himself as he ages. We are among the lucky ones with the know-how to appreciate the Wisdom of the Body and to get pleasure from its unfolding.

First Community Village
1862-35 Riverside Dr.
Columbus, OH 43212

Virginia M. Fiske to Louise:

For your record, I continue to carry on some research on the mammalian pineal at my lab at Wellesley College. Currently I'm trying to write up for publication results of studies made during the last three years with very considerable help from four Wellesley Honors students, all of whom are now in medical or graduate school. Occasionally I serve as "peer reviewer" and also evaluate papers submitted for publication in a journal. Lack of research funds since my retirement is a continuing problem and may force me to quit the lab.

We are very fortunate in that my husband and I enjoy good health. Gardening, swimming, and tennis keep us moving, while children, grandchildren, and friends give zest to our lives. For your information, I have deposited "archival materials" in the Wellesley College Library. The College asks this, rather firmly, of all its retirees. It is heartening to learn of an institution that has "firm" ideas about the future usefulness of archives.

27 Hollis St.
Sherborn, MA 01770

1983 APS Fall Meeting Honolulu, Aug. 20–24 XXIX IUPS Congress Sydney, Australia, Aug. 28–Sep. 3[1]

Announcement of Travel Plans

A variety of travel plans are being proposed for the 1983 Meetings. Chevy Chase Travel guarantees that you will have the advantage of the lowest existing air fares and the best hotel and tour prices.

To arrange the most economical and attractive travel plans, we would appreciate your completing and returning the postcard attached inside the cover of this booklet.

This is an informational survey and in no way commits you to the plans indicated. Your comments and/or suggestions are welcome and may prove instrumental in the final design of the travel plans. The detailed brochure will be sent to you in September.

Plan 1. Transportation to Honolulu and return for those attending the Fall Meeting only.

Plan 2. Attend the Fall Meeting, then continue to Australia to Heron Island on the Great Barrier Reef for a 3-day interlude before going to Sydney for the Congress.

Plan 3. Fly directly from the West Coast to Sydney for the Congress, or join the 3-day Barrier Reef stay before going on to Sydney.

Plan 4. After the Congress in Sydney, take an exciting 4-day tour to the Outback in the heart of Australia to visit Alice Springs and Ayers Rock.

Plan 5. A Post-Congress 4-day tour to the cities of Canberra and Melbourne, traveling by motorcoach to give you a feel of the countryside.

Plan 6. A 10-day Post-Congress tour to New Zealand, visiting both the North and South Islands, including the beautiful Milford Sound area.

Plan 7. A special Post-Congress Tour to China (limited to 25 persons). From Sydney to Hong Kong for 2 days. By train into China to Canton. From there to the incredible mountains of Kweilin, the cosmopolitan city of Shanghai and then to Peking for 4 days with a visit to the Great Wall. There will be visits to Physiological Institutes and Hospitals. Return via Tokyo to the West Coast.

Please Note: The above plans are flexible. The Fall Meeting in Honolulu can be included with any of the Plans. Also, you are allowed a stopover in Honolulu or Tahiti on the return at no additional air fare.

Individual plans can be arranged to include any of the Satellite Meetings to be announced in the Preliminary Program.

Announcement of Travel Awards

The USA National Committee for the International Union of Physiological Sciences (USANC/IUPS) is cooperating with the American Physiological Society (APS) to administer a travel grant program to benefit American scientists who could not attend the XXIX International Congress of Physiological Sciences in Sydney, Australia, August 28–September 3, 1983, without such assistance. The USANC/IUPS will accept applications for the travel grant program and select the awardees; the APS will raise funds for the program and will issue the travel awards.

A limited amount of funds will be available. Those eligible to apply for awards are qualified scientists who are citizens or permanent residents of any part of North America (Canada, USA, and Mexico) and who plan to participate fully in the Congress. Each applicant will be judged on the merit of his contribution to the Congress in Sydney, considering his training, experience, and potential, and for a portion of the awards priority will be given to young scientists. Grants will be limited to a sum approximately $300 less than the transportation costs based on the lowest scheduled airline fare from airport of departure and return.

Requests for application forms must be made in written form and be addressed to: USA National Committee for IUPS, Attn: June S. Ewing, Staff Officer, Division of Medical Sciences, National Research Council, 2101 Constitution Ave., NW, Washington, DC 20418.

Postmark deadline for completed applications is October 15, 1982. To the degree that it is possible, successful applicants will be notified by December 15, 1982.

[1] For detailed information, refer to *The Physiologist* 24(6): 23–33, 1981.

Third Banff International Hypoxia Symposium

The Third International Hypoxia Symposium will be held January 25–28, 1983 at the Banff Springs Hotel, Banff, Alberta, Canada. This third biennial conference on hypoxia will maintain its original theme and its international scope. Many of the areas of hypoxia will be covered and special emphasis will be placed on the latest research findings and their relation to bedside clinical practice. Topics in the 1983 program include oxygenation during normal and disordered sleep; muscular exertion and fatigue during hypoxia; endocrine responses to oxygen lack; characteristics of high altitude natives; oxygen utilization in animals; and recent observations in extreme altitude mountaineering. Sessions for free communications will be included. Keynote speakers will be invited. *Call for abstracts:* deadline September 30. *Further information:* Third Banff International Hypoxia Symposium, The Arctic Institute of North America, The University of Calgary, 2500 University Drive N.W., Calgary, Alberta, Canada T2N 1N4.

Rhythmic Fluctuation of Systemic Arterial Pressure Symposium

An International Symposium on Rhythmic Fluctuation of Systemic Arterial Pressure will be held in Matsumoto, Japan, October 25–27, 1982, under the sponsorship of the Commission on Cardiovascular Physiology of the IUPS and the Japan Society for the Promotion of Science. Invited lectures and free communications will be presented. *For additional information* contact: Professor K. Miyakawa, The 2nd Department of Physiology, Shinsju University School of Medicine, 3-1-1, Asahi, Matsumoto, 390, Japan.

Announcements

Scipione Caccuri Prize

The Fondazione Clinica del Lavoro has established an International Prize (20,000,000 Italian lire) in memory of Professor Scipione Caccuri, the former Director of the Institute of Occupational Medicine at the University of Naples. The prize will be assigned to a previously unpublished work making an original contribution in the field of Occupational and Industrial Medicine, Physio-Pathology and Hygiene. Texts written in Italian, French, English, German, or Spanish considered meritorious will be published in a special issue of *Giornale Italiano di Medicina del Lavoro. Texts should be addressed to:* Segreteria della Fondazione Clinica del Lavoro, Via Boezio, 26 27100 Pavia, Italy, and should be received by December 31, 1983.

Rogers Heart Foundation's 1982 CME Meetings

Oct 10–15: Rx For a Healthy Heart to be held at Sheraton Sand Key Hotel, Clearwater Beach, FL; director: Henry J.L. Marriott, M.D.; Fee: $200; Credit hours: 24.

Oct 28–Nov 1: 23rd Annual Workshop in Electrocardiography for Nurses & Physicians to be held at Sheraton Sand Key Hotel, Clearwater Beach, FL; conducted by Henry J.L. Marriott, M.D.; Fee: $150; Credit hours: 24.

Nov 29–Dec 4: 20th Annual Seminar in Cardiology to be held at Southampton Princess Hotel, Southampton, Bermuda; director: Henry J.L. Marriott, M.D.; Fee: $300; Credit hours: 24.

For further information contact: Rogers-Heart Foundation, Inc., St. Anthony's Hospital, St. Petersburg, FL 33705.

Annual Postgraduate Clinical Pharmacology Course

The Annual Postgraduate Course in Clinical Pharmacology, Drug Development and Regulation, cosponsored by the University of Rochester School of Medicine and the American Society for Clinical Pharmacology and Therapeutics, will be held at the Marriott Inn Thruway in Rochester, NY, October 25–29, 1982. *For more information contact:* William Wardell, M.D., Ph.D., Pharmacology Department, University of Rochester Medical Center, 601 Elmwood Ave., Rochester, NY 14642. Telephone: Ginny Mastroleo (716) 275-2466.

Arthritis Foundation Research Fellowships

The Arthritis Foundation offers a limited number of research fellowships for physicians and scientists pursuing investigative or clinical teaching careers in the areas of science related to arthritis. Although research projects are involved, these awards are designed for advanced training in research and not as grants-in-aid for specific projects. Applications are due by September 1, 1982. There are also research fellowships and grants available to graduate nonphysician health professionals. These awards are for research training and development of investigative skills. Research proposals on arthritis management and/or comprehensive patient care are appropriate for these programs. Applications for allied health professional fellowships and research grants are due November 1, 1982. *To obtain applications for these awards* (which will commence July 1, 1983) *write to:* Research Department, Arthritis Foundation, 3400 Peachtree Road, N.E., Atlanta, GA 30326.

THE PHYSIOLOGY TEACHER

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A Pulsatile Cardiovascular Computer Model for Teaching Heart-Blood Vessel Interaction

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Because of its mechanical nature, the cardiovascular system lends itself to modeling approaches for teaching some of its fundamental properties. This has been recognized by several workers who have developed computer models of the cardiovascular system for teaching purposes (2–5). These teaching models are unique in providing a mechanism for examining system behavioral changes that result from selected changes in specific system components. Experience gained from observing such simulated behavior gives the student a valuable basis for interpreting integrated aspects of physiological adaptation.

One concept in cardiovascular physiology that is particularly difficult to convey to students is the relationship between systemic circulatory states and cardiac preload and afterload and the relationship between cardiac preload and afterload and cardiac output. This difficulty arises because of three factors: *1*) the terms preload and afterload lose preciseness in their definition as one goes from isolated cardiac muscle fibers to the intact ventricle; *2*) there is inherent complexity in the interactions between system components and the sum of these interactions are responsible for observed events; and *3*) there is a bothersome discrepancy in textbook accounts of the relative roles of the various phenomena that determine ventricular pumping behavior. Most textbooks describe the mechanical behavior of cardiac muscle, in part, by the force-velocity relationship; i.e., myocardial fiber shortening is determined by the afterload against which shortening occurs. This picture of cardiac muscle physiology is in keeping with the many recent experimental findings which show that ventricular emptying, and thus stroke volume, is characteristically sensitive to systolic pressure states (6). Yet cardiac output regulation is routinely described in textbooks and review articles as primarily the result of the interplay between vascular and cardiac factors that determine right atrial pressure, a preload variable. The issue to be resolved for the student is the reconciliation of these seemingly opposing points of view; i.e., ventricular emptying and stroke volume is determined by systolic pressures, but cardiac output is determined by right atrial pressure. A paradigm is needed which includes a pump component that is exquisitely sensitive in its behavior to afterloading variables and yet also, as part of a complete circulatory system, admits to preloading factors as being primarily responsible for cardiac output. This paradigm must extend to include the responsiveness of the system to changes in the pump itself (i.e., changes in inotropic state) and to changes in other parts of the system.

A mathematical model of the mechanical aspects of the cardiovascular system was developed as a vehicle which achieved the above objective. This model was used as an instructional tool in the cardiovascular section of the physiology course taught to students of veterinary medicine and in a separate course to graduate students in physiology. The model differed from others that have been used in the past for instructional purposes in that it predicted hemodynamic events on a time scale commensurate with the pulsatile actions of the heart. This allowed prediction of pulsatile pressure and flow events in both the systemic and pulmonary circulations as well as ventricular pressure-volume loops. This latter information is particularly vital to understanding the mechanisms responsible for bringing about changes in stroke volume and cardiac output that accompany changes in cardiovascular system properties. Thus the model was unique both in being more detailed than those used in the past and in providing the students with the information needed to elucidate the mechanisms responsible for observed behavior changes. The model has proven to be very useful in teaching interaction phenomena in the cardiovascular system with particular emphasis on factors determining pump performance.

Methods

Model

The cardiovascular system model, which was developed as a teaching tool, is shown in electrical analog form in Fig. 1. It consists of five major components: left heart, systemic arteries, systemic veins, right heart, and pulmonary circulation. The model is similar in general structure to that proposed by Defares et al. (1) but differs in some particulars. Both the right and left ventricles are modeled as time-varying capacitance chambers [C(t)] with internal outflow resistance. The description of time variation for these

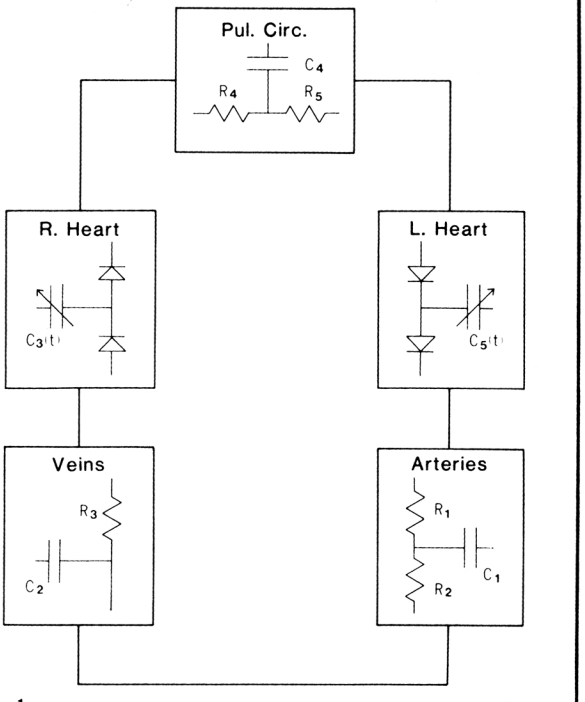


Fig. 1
A cardiovascular model for teaching the mechanical interactions between major components of the system: L Heart, left heart; R Heart, right heart; Pul Circ, pulmonary circulation.

ventricular capacitances is similar to that proposed by Suga and Sagawa (7) for the left ventricle. The actual values of C(t) came from data generated in laboratory experiments. These experiments involved the production of isovolumic left ventricle contractions at known end-diastolic volume (EDV). Calculation of C(t) was according to

$$C(t) = EDV/(P_{iso} - P_0)$$

where P_{iso} is the isovolumic pressure and P_0 is the assumed zero-volume pressure. A plot of this calculation over one cycle is given in Fig. 2. Because the data were in digital format, the calculations resulted in a table of discrete C(t) values at 5-ms intervals. The right ventricle C(t) was scaled so as to undergo one-third the change in value experienced by the left ventricle. Inotropic state is given quantitative definition by the C(t) parameter. Changes in inotropic state could be induced by changing the value of C(t) in a prescribed way.

The systemic arteries were modeled as a modified windkessel (8). The systemic veins were modeled as a single capacitance with an outflow resistance. The

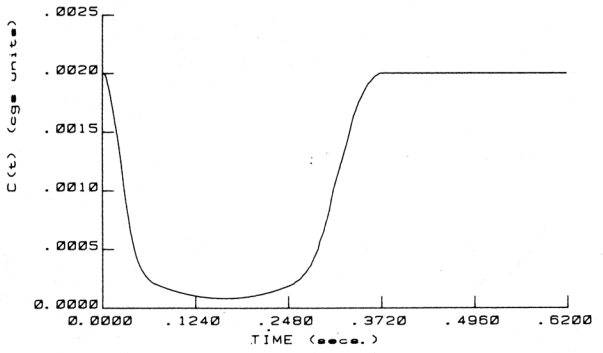


Fig. 2
Time variation of the left ventricle compliance, C(t), over one cardiac cycle. This information was used in the model as a table of numbers representing values at 5-ms intervals.

pulmonary vascular system was modeled as a single capacitance with a lead-in resistance and an outflow resistance. The tricuspid, pulmonary, mitral, and aortic valves were treated as one-way flow devices that opened or closed depending on the sign of the pressure difference across the resistance element either leading into or away from the valve. The capillaries and atria are not explicitly represented in this model.

A set of five first-order differential equations were written to describe the pressure across each capacitance and thus at every node in the model. The method used for forming the differential equations is demonstrated by analyzing pressure and flow events in the arterial component of the model (see Fig. 1). Flow entering the arteries passes through the entry impedance (R_1) and will go to either the arterial capacitor (C_1) or through the arteriolar resistance (R_2) and into the veins. Thus the nodal equation becomes $q_{R_1} = q_{C_1} + q_{R_2}$, where the subscripts indicate the circuit element through or onto which flow passes. Now by definition, $q_{R_1} = (1/R_1)(p_5 - p_1)$, $qC_1 = C_1\, dp_1/dt$, and $q_{R_2} = (1/R_2) \bullet (p_1 - p_2)$, where p_1, p_2, and p_5 are pressures across the arterial, venous, and left ventricle capacitances, respectively. Since q_{R_1} occurs only when the aortic valve is open, we introduce the valve condition V which has the value of either 1 when the valve is open or 0 when the valve is closed and $q_{R_1} = (V/R_1)(p_5 - p_1)$. Substituting the values of the respective flows into the nodal equation and rearranging terms results in $dp_1/dt = -(1/C_1R_2 + V/C_1R_1)p_1 + (1/C_1R_2)p_2 + (V/C_1R_1)p_5$. This is the first member in the matrix differential equation given in the Appendix. By sequentially repeating this analysis for each node in the model, the complete matrix equation is generated. Flow and volume were algebraically related to the node pressures (see Appendix).

The differential equations were programmed to be solved as a simultaneous set using a 5-ms step size and a fourth-order Runge-Kutta integration method. The solution was carried out on a Hewlett-Packard 1000 digital computer. The results of the solution of the dynamic equations allowed calculation of the values of simulated pressure, flow, and volume at every point in the model. Nominal values for all parameters were taken either from data generated in the research laboratory or from data reported in the literature. These parameters are representative of what would be expected in a resting 20-kg dog. This set of model

parameters represent base-line values from which user specified changes could be made. Changes in parameters were brought about by multiplying the base-line value with a user specified factor. Thus steady-state solutions of model eqauations were required for a broad range of parameter values. This created a special problem with regard to supplying initial values for beginning the dynamic solution process. The model contained time constants which ranged over four orders of magnitude, from 140 s for the venous component of the system to 0.025 s for the systolic left ventricle. To represent transient behavior in all regions of the model while simultaneously representing pulsatile behavior would be computationally time consuming and wasteful. Since the intended application involved just steady-state predictions, a procedure was implemented wherein an algebraic model, homologous to the dynamic model, was used to provide starting values for the dynamic equation solution process. A user-initiated change in model parameters would first be acted upon by the algebraic model, which then passed the results of its solution to the dynamic model as starting values. The solution of the dynamic equation was carried out for three complete heart cycles, and the last of these three cycles was used as the prediction of steady-state behavior. Comparison of independent predictions from the algebraic model with average values from the dynamic model showed that over a broad range of parameter values each predicted the results of the other quite closely. This agreement between predictions by the two models validated the procedure of starting the dynamic equation solution process with the results from the algebraic model and running the dynamic solution procedure to steady state over only three cycles. The time involved in carrying out a single solution procedure for any set of parameter values was on the order of 1 s.

Details of the algebraic and dynamic models as well as the base-line parameter values are contained in the Appendix.

Student Interaction Program

A program to supervise the interaction of the student with the model and its output was also written to be run on the Hewlett-Packard 1000. Through prompts on the CRT of a Hewlett-Packard 2647 graphics terminal, each student was able to set any model parameter at any fractional or multiple of the base-line value. Also, the student was able to specify one of several optional forms for the output. Output display was on either or both the terminal CRT and/or an X-Y pen plotter. Output could be in the form of *1*) any variable as a function of time over one cycle, *2*) the left ventricle pressure-volume loop over one cycle, and/or *3*) a table showing parameter values and the cycle average of the various hemodynamic variables which had been calculated. The color of each plot could be specified so that several outputs could be plotted on the same paper and distinguished from one another.

Both the model program and the interaction program were written in FORTRAN. A flow diagram for these programs is presented in the Appendix. The code for solution of the model equations is adaptable to any small computer with FORTRAN capability. The interaction program with graphics input/output is restricted to the Hewlett-Packard system. Code for each program will be provided upon request.

Laboratory Exercises

The laboratory exercise as given to the veterinary students differed in content and format from that given to the graduate students. These differences are described below.

Study I

A laboratory exercise was designed to direct 80 veterinary students through a protocol for achieving specific learning objectives. These students had not previously been exposed to the mathematical and physical details of the model. Only a descriptive explanation in graphical terms had been given prior to the laboratory session. The laboratry was given to 4 groups of 20 students as a demonstration lab. Changes in model parameters were made by the laboratory instructor. The students were informed of all actions and a running discussion explaining what was happening took place throughout the demonstration. Computer output onto the X-Y plotter was videographed and displayed on two television screens for ease of viewing.

The objectives were to develop an appreciation of how hemodynamic variables throughout the heart-blood vessel system depend on individual properties of component parts of that system. Specifically, *1*) to quantify the changes in the hemodynamic variables of left venticular stroke volume, central venous pressure, and arterial pressure that occur with changes in the individual system properties of total peripheral resistance, left ventricular inotropic state, and venous compliance; *2*) to quantify the changes in the above hemodynamic variables when all of the above system properties are changed together as they would change with physiological adaptation; and *3*) to identify, through observation of left ventricular pressure-volume loops, those mechanisms that are responsible for the above observations.

In the laboratory exercise attention was focused on three important system properties [the left ventricular inotropic state (IS), the total peripheral resistance (TPR), and the venous compliance (Cv)] and three often measured hemodynamic variables [left ventricular stroke volume (SV), arterial pressure (Pa), and central venous pressure (Pcv)]. It was stressed that the three selected properties are three which are known to change significantly with physiological adaptation to such conditions as exercise and blood loss and with diseases such as diseases of heart muscle (cardiomyopathy) and the systemic circulation (systemic hypertension). Further, an analysis of the interrelationships between these system properties and variables becomes important to understanding how the cardiovascular system responds to a wide variety of conditions associated with health and disease.

Specific experiments were defined wherein selected parameters were changed by specific amounts and observations were made as to the consequences of these parametric changes.

The data were displayed in two formats. In one, a graph of the instantaneous left ventricular pressure vs. instantaneous left ventricular volume was made by the computer driven X-Y plotter. From this plot, the mechanisms were discerned by which stroke volume changes as a result of system component changes. In the second format, data were displayed in a tabular form from which graphs were constructed by the students.

The table included the values of each of the three variables and the value of the system component that was made to change. These data allowed the assessment of the sensitivity of each hemodynamic variable to a change in a given system component property.

Procedure for Objective 1

The model cardiovascular system predicted hemodynamic behavior with all system properties possessing values expected to be seen in a normal resting 20-kg dog. This was the control state.

The system properties were changed individually from the control state by systematically multiplying each property by the factors 0.5, 1.5, and 2.0. The predicted hemodynamic variables (SV, Pa, Pcv) at each value of the selected property were displayed as described above. The tabular data was used for constructing the following graphs: SV vs. TPR, Pa vs. TPR, Pcv vs. TPR, SV vs. IS, Pa vs. IS, Pcv vs. IS, SV vs. Cv, Pa vs. Cv, Pcv vs. Cv. All data were plotted as percentage change from the control state. Linear regression procedures were used to quantitate the relationship in each case.

Procedure for Objective 2

To simulate a cardiovascular system in a state of excitement, the properties of TPR and IS were simultaneously multiplied by 1.5 while Cv was multiplied by 0.5. The resultant hemodynamic state was predicted and displayed as above. To simulate a cardiovascular system in a state of depression, TPR and IS were multiplied by the factor 0.5 while Cv was multiplied by 1.5. Again, prediction and data displays were as before.

The observations from the above procedures were used as a data base for answering certain questions. The questions were designed to emphasize quantitative aspects of behavior and were as follows.

1) Trace the events of the cardiac cycle on the control pressure-volume loop; identify points that would be associated with valve opening and closure and the points where the P, QRS, and T waves of the ECG could be expected to have taken place.

2) Compare the relative sensitivity of each hemodynamic variable (SV, Pa, Pcv) to changes in each cardiovascular system property (IS, TPR, Cv). Use the slopes of the graphs you have constructed. Sensitivity = % change in variable/% change in system property.

3) Using the pressure-volume loops, estimate how much of the change in stroke volume was due to changes in EDV and to changes in end-systolic volume (ESV) with each of the system changes which were made. Which of the system variables under consideration causes a primary change in EDV and which causes a primary change in ESV?

The questions were answered by groups of four students working together. The question answers were handed in 1 wk after the laboratory period and constituted the laboratory report for that period.

Study II

The model was used in a laboratory exercise for a group of five graduate students enrolled in an advanced course in cardiovascular physiology. The mathematical and physical considerations basic to the model had been explained to these students in considerable detail prior to the laboratory exercise. During the first laboratory session the graduate students went through the same protocol followed by the veterinary students in the above description. However, the graduate students operated the CRT keyboard themselves and personally made the changes in the model parameters. On a second laboratory session, the students were given the opportunity to perform their own experiments with the model. The selection of experiments performed included stepwise changes in blood volume, aortic characteristic impedance, and arterial compliance; differential left- and right-heart inotropic state changes; hemodynamic sensitivity to changes in TPR at various cardiac inotropic states; and simulation of exercise states at different blood volume levels.

Student Acceptance

Questionnaires were distributed to the veterinary students to assess the effectiveness of the laboratory. The students were asked to grade the laboratory exercise with respect to meeting the stated objectives. They were also asked to rate the laboratory exercise vs. lecture and textbook as a means for achieving these objectives.

Graduate students were queried orally as to the usefulness of the laboratory in helping them understand quantitative cardiovascular physiology.

Results

Experimental

Model predicted pressure-volume loops for the procedure in which TPR alone was varied are shown in Fig. 3. The SV, Pa, and Pcv response are shown in the top third of Table 1. The tabular results indicate only a very small change in stroke volume (from 24 to 22.1 ml) with over a fourfold change in TPR and correspondingly large changes in arterial pressure (from 70 to 243 mmHg)! Yet this seeming insensitivity of pump behavior to the load against which flow is delivered is the consequence of rather large system compensations that express themselves internal to the left ventricle. Over the full range of behavior end-systolic volume has changed 18.7 ml while end-diastolic volume has changed 17.5 ml. These changes in ESV and EDV are on the order of the stroke volume itself. Correspondingly, the ejection fraction has also changed dramatically, from 0.54 at rest to 0.40 at the highest TPR to 0.65 at the lowest TPR. Note that the changes in ESV are larger than the changes in EDV.

These observations suggest the following scenario. Elevations of TPR tend to elevate arterial and ventricular pressures which, in turn, affect the end-systolic volume through the ventricular capacitance property. This in itself would reduce stroke volume dramatically. However, the retention of volume within the left ventricle at end systole coupled with nearly undiminished filling via flow delivery from the right heart through the pulmonary circulation results in expansion of EDV. Elevated EDV promotes stroke volume delivery (the Frank-Starling mechanism) and compensates in large for the reduction in SV caused by elevated TPR. It is important to keep in mind that the primary expression of elevated TPR effects is the increase in ESV, whereas the increase in EDV is a secondary effect due to compen-

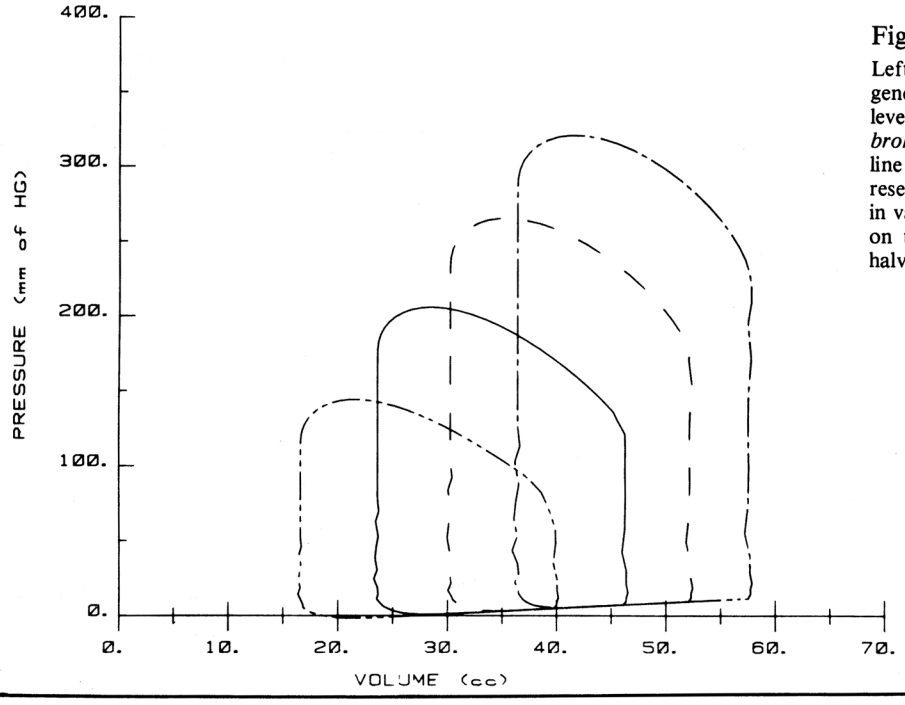


Fig. 3

Left ventricle pressure-volume loops generated by model during 4 different levels of total peripheral resistance. *Unbroken line* represents loop under base-line conditions. Loop on far right represents behavior when TPR was doubled in value over that of base-line state; loop on the far left resulted when TPR was halved.

Table 1

	TPR	IS	Cv	SV	Pa	Pcv
A	1.0	1.0	1.0	23.4	130.0	7.8
	0.5	1.0	1.0	24.0	69.7	8.3
B	1.5	1.0	1.0	22.8	188.1	7.4
	2.0	1.0	1.0	22.1	243.3	6.9
	1.0	0.5	1.0	18.1	103.7	9.9
C	1.0	1.5	1.0	26.0	142.5	6.7
	1.0	2.0	1.0	27.6	149.8	6.0
	1.0	1.0	0.5	32.2	183.0	14.7
D	1.0	1.0	1.5	19.1	104.6	4.8
	1.0	1.0	2.0	16.6	89.6	3.0
E	1.5	1.5	0.5	34.5	285.7	12.3
	0.5	0.5	1.5	15.1	45.3	6.8

Model-predicted stroke volume (SV), means arterial pressure (Pa), and central venous pressure (Pcv) when changes are made in total peripheral resistance (TPR *rows B*), cardiac inotropic state (IS *rows C*), and venous compliance (Cv *rows D*). *Row A* represents basal operating state; *rows E* represent hemodynamic predictions for simulated cardiovascular states of excitation and depression. Values of TPR, IS, and Cv are multiples of basal values. Units of SV are in ml, Pa in mmHg, and Pcv in mmHg.

satory behavior intrinsic to the circulatory system. The primary effect is slightly larger than the compensatory effects and there is a distinct net result that SV tends to decrease with elevated TPR.

The results of this model experiment, the student analysis of the observations, and the explanation through the ensuing scenario are an extremely effective mechanism for conveying to students the concept of internal compensatory mechanisms that result from the manner in which the system is organized. This experiment bridges the gap between the apparent dichotomy of simultaneous pump sensitivity to afterload and system flow insensitivity to changes in cardiac afterload variables.

The results of carrying out the procedures of individually varying venous compliance and myocardial inotropic state are shown in Figs. 4 and 5, respectively, and in the bottom two-thirds of Table 1. Analyses similar to that for changes in TPR were conducted. One result of these analyses was the comparison of the sensitivities of SV changes to each of the system parameters. The results of this analysis are shown in Fig. 6, which shows that the stroke volume is highly sensitive to venous capacitance, moderately sensitive to inotropic state, and only slightly sensitive to TPR.

Several experiments additional to those given to the veterinary students were conducted by the graduate students. Interesting results from one of these experiments were the changes in right atrial pressure and left atrial pressure which accompanied depression of right-heart inotropic state alone and then, separately, depression of left-heart inotropic state alone. This difference in changes of these two pressures vividly demonstrated the backward failure processes of right-and left-heart failure. All of these results from the model are in keeping with the bulk of experimental reports and textbook accounts.

Student Acceptance

To summarize the results of the questionaire, most veterinary students rated the Computer Demonstration/Lab as being either effective or very effective in meeting each of the four specific objectives of the lab. However, with regard to helping them develop an appreciation of how hemodynamic variables throughout the heart-blood system depend on individual properties of component parts of the system, 68% of the students felt that the demonstration was most effective in combination with related lecture material. Only 11% of the students felt that this objective could be met with lectures alone.

The graduate students unanimously endorsed the computer laboratory. They felt that it provided an effective format for synthesizing many conceptually difficult aspects of cardiovascular function.

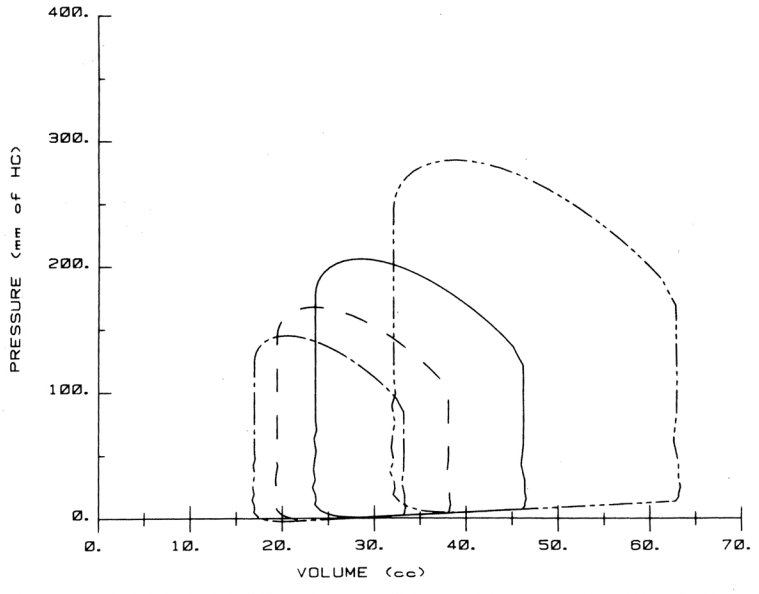


Fig. 4

Left ventricle pressure-volume loops generated by the model with changes in venous compliance (Cv). *Solid line* represents base-line conditions. Loop on the far right occurred when Cv was halved; loop on the far left occurred when Cv was doubled.

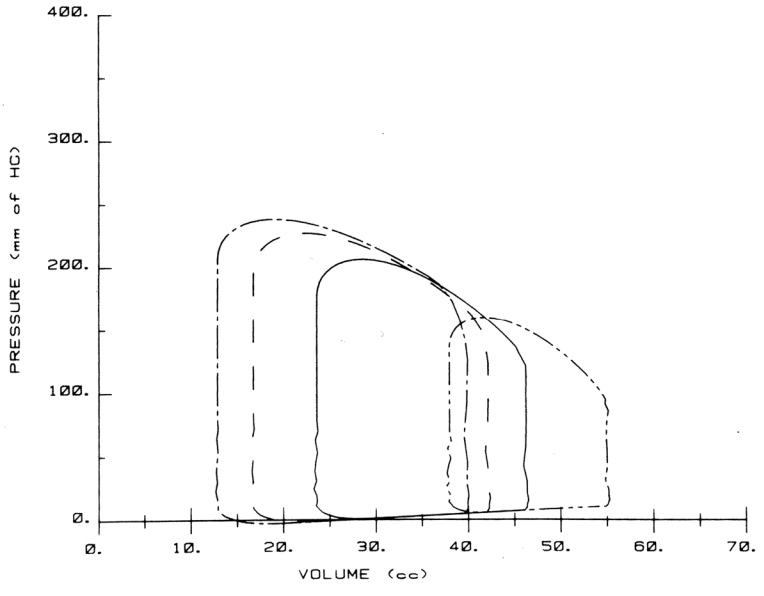


Fig. 5

Left ventricle pressure-volume loops generated by the model with changes in inotropic state (IS). Again, base-line condition is represented by loop with *unbroken line*. Loop on right occurred when IS was halved; loop on left occurred when IS was doubled.

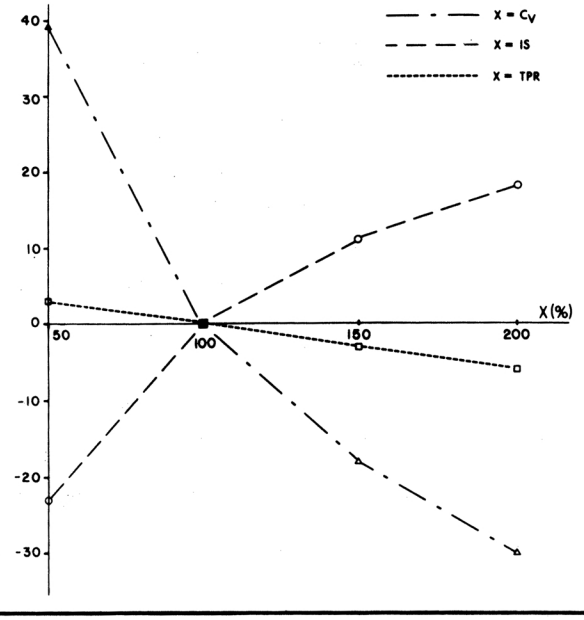


Fig. 6

Comparison of stroke volume sensitivity to changes in TPR (*dotted line*), Cv (*dot-dash*), and IS (*dashed line*). Order of sensitivity magnitude is Cv > IS > TPR.

Conclusion

The model used in these teaching efforts gave realistic predictions of pulsatile pressure, flow, and volume events in the cardiovascular system. Through the observation of predictions that arose from systematic changes in total peripheral resistance, ventricular inotropic state, and central venous compliance it was possible for students to gain an appreciation of how hemodynamic variables throughout the heart-blood vessel system depend on individual properties of component parts of that system. By then conducting his/her own analysis of the model-generated data, the student was able to develop insight into the mechanisms operative within the system which brings about the resultant behavior and its changes. Particularly, it was demonstrated that a ventricular pump which is sensitive to afterload will have its output regulated primarily by preload variables when it becomes part of the intact circulatory system. The computer model is a unique vehicle for demonstrating these interactive phenomena.

Overall, most veterinary students indicated that the computer Demonstration/Lab was an effective means of meeting and teaching the stated objectives. The suggestions of the students for improvements can mainly be summarized as indicating a desire for changing some of the particulars with regard to representation of the material. While the Demonstration/Lab was clearly successful in its present form, moving to either a small group lab or to individualized labs in the future would further improve the effectiveness of the lab. This was demonstrated by the experience of the graduate students who found that hands-on interaction with computer and individually designed experiments resulted in a very rich educational experience.

Appendix

Equations of Dynamic Model

The nodes of the dynamic model (Fig. 1) were numbered sequentially starting at the arterial node and moving through the model in the direction of flow to the left ventricle. The resistance and capacitance elements were numbered according to the node into which they led. The valves were numbered 1-4, starting with the tricuspid valve and finishing with the aortic valve. Letting the X_is stand for the pressure at each node, Q_is the volume on each capacitance, and q_is the flow into each node, the dynamic equations become

$$\dot{x} = \begin{vmatrix} P_{11} & P_{12} & 0 & 0 & P_{15} \\ P_{21} & P_{22} & P_{23} & 0 & 0 \\ 0 & P_{32} & P_{33} & P_{34} & 0 \\ 0 & 0 & P_{43} & P_{44} & P_{45} \\ P_{51} & 0 & 0 & P_{54} & P_{55} \end{vmatrix} x$$

where

$$P_{11} = -1/C_1R_2 - V_1/C_1R_1$$
$$P_{12} = -1/C_1R_2$$
$$P_{15} = V_1/C_1R_1$$
$$P_{21} = 1/R_2C_2$$
$$P_{22} = -1/R_2C_2 - V_2/C_2R_3$$
$$P_{23} = V_2/R_3C_2$$
$$P_{32} = V_2/R_3C_3$$
$$P_{33} = -V_2/R_3C_3 - V_3/R_4C_3 - C_3/C_3$$
$$P_{34} = V_3/R_4C_3$$
$$P_{43} = V_3/R_4C_4$$
$$P_{44} = -V_3/R_4C_4 - V_4/R_5C_4$$
$$P_{45} = V_4/R_5C_4$$
$$P_{51} = V_1/R_1C_5$$
$$P_{54} = V_4/R_5C_5$$
$$P_{55} = -V_1/R_1C_5 - V_4/R_5C_5 - C_5/C_5$$

and

$$Q_1 = C_1X_1;\ Q_2 = C_2X_2;\ Q_3 = C_3X_3;\ Q_4 = C_4X_4$$
$$Q_5 = C_5X_5;\ q_1 = (X_5 - X_1)/R_1;\ q_2 = (X_1 - X_2)/R_2$$
$$q_3 = (X_2 - X_3)/R_3,\ q_4 = (X_3 - X_4)/R_4;\ q_5 = (X_4 - X_5)/R_5$$

and

$$V_1 = 1 \text{ if } X_2 \geq X_3;\ V_2 = 1 \text{ if } X_3 \geq X_4$$
$$V_3 = 1 \text{ if } X_4 \geq X_5;\ V_4 = 1 \text{ if } X_5 \geq X_1$$

otherwise $V_1 = V_2 = V_3 = V_4 = 0$

Equations of Algebraic Model

The single difficulty in bridging the gap between the algebraic and dynamic model is in the description of the ventricles. In its algebraic form the model description of the left ventricle becomes at fixed heart rate.

$$SV = SV_0 + \beta P_f - \alpha P_L$$

where α is the minimum value of $C_5(t)$ from the dynamic model; β is the maximum value of $C_5(t)$ from the dynamic model; and $SV_0 = P_0(\alpha - \beta)$, where P_0 is the zero intercept on the pressure axis in $C_5(t)$.[1]

Likewise for the right ventricle

$$SV = SV_0' + \beta' P_f' - \alpha' P_L'$$

where β' is the minimum $C_3(t)$; α' is the maximim $C_3(t)$; and $SV_0' = -P_0(\alpha' - \beta')$. Conservation of volume requires $Q_1 + Q_2 + Q_{RV} + Q_4 + Q_{LV} = K$.

We define

$$Q_{RV} = \frac{\beta'(P_f - P_0) + \alpha'(P_L' - P_0)}{2}$$

and

$$Q_{LV} = \frac{\beta(P_f - P_0) + \alpha(P_L - P_0)}{2}$$

Using the equations for flow across the resistors and pressure across the capicitors, the entire algebraic model becomes

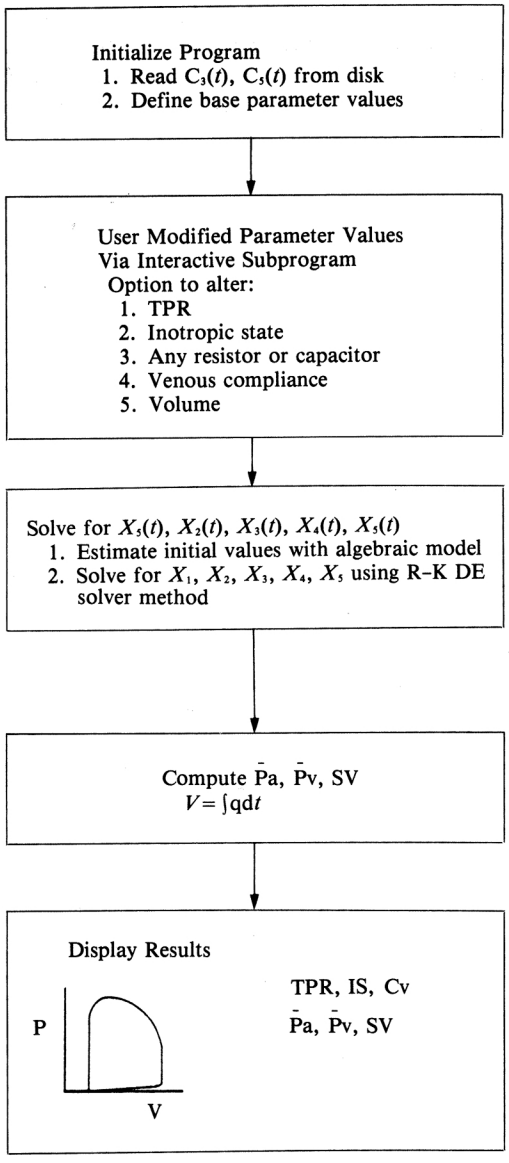
$$\begin{bmatrix} C_1 & C_2 & C_4 & \beta/2 & \alpha/2 & \beta'/2 & \alpha'/2 \\ 0 & 0 & 0 & \beta & -\alpha & -\beta' & \alpha' \\ -1 & 0 & 0 & -\beta/T\,R_1 & (1+\alpha/T\,R_1) & 0 & 0 \\ 1 & -1 & 0 & -\beta/T\,R_2 & \alpha/T\,R_2 & 0 & 0 \\ 0 & 1 & 0 & -\beta/T\,R_3 & \alpha/T\,R_3 & -1 & 0 \\ 0 & 0 & -1 & -\beta/T\,R_4 & \alpha/T\,R_4 & 0 & 1 \\ 0 & 0 & 1 & -(1+\beta/T\,R_4) & \alpha/T\,R_5 & 0 & 0 \end{bmatrix} \begin{bmatrix} P_1 \\ P_2 \\ P_4 \\ P_f \\ P_L \\ P_f' \\ P_L' \end{bmatrix} = \begin{bmatrix} K + [(\beta + \alpha + \beta' + \alpha')/2]P_0 \\ SV_0' - SV_0 \\ R_1/T\,SV_0 \\ R_S/T\,SV_0 \\ R_3/T\,SV_0 \\ R_4/T\,SV_0 \\ R_5/T\,SV_0 \end{bmatrix}$$

[1] See text for more complete description of P_0.

Parameters Values

Base-line parameters used in the model were $R_1 = 250.$, $R_2 = 3{,}800.$, $R_3 = 350.$, $R_4 = 625.$, $R_5 = 200.$, $C_1 = 0.00019$, $C_2 = 0.0326$, $C_4 = 0.0082$. Both C_3 and C_5 are time varying parameters with the changes in C_3 equal to one-third the changes in C_5 over a cardiac cycle (see Fig. 2). The blood volume above dead volume equaled 950 ml. Units of resistance are dyn • s•cm^{-5} and of capacitance are cm^5•dyn^{-1}.

Flow Diagram for Cardiovascular Teaching Program



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Correction

The Physiology Teacher, Book Review Section, 25(1): 60, 1982.

Estrogens and Brain Function: Neural Analysis of a Hormone-Controlled Mammalian Reproductive Behavior. The publisher should be Springer-Verlag. Cost should be $24.90.

Acid-Base Balance in the Dog

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The purpose of this exercise is to illustrate the interrelationships of respiratory and metabolic acidosis and alkalosis.

The student completing this exercise should be able to calculate plasma bicarbonate ion (HCO_3^-) by the Henderson-Hasselbalch equation[1] and predict the respiratory/metabolic compensation for a given acid-base disturbance. The student should also be able to plot and interpret acid-base data on the Siggaard-Andersen curve nomogram.

Overview of Protocol

An anesthetized dog, whose respiratory muscles have been paralyzed by succinylcholine, will be ventilated by a volume ventilator so that the frequency and tidal volume of each breath can be maintained constant at any desired value. The acid-base balance will be studied first during normal pulmonary ventilation and alveolar P_{CO_2} ($P_{A_{CO_2}}$), then during hyperventilation and during elevated $P_{A_{CO_2}}$ to show the changes in arterial pH, P_{CO_2}, and [HCO_3^-] produced by uncompensated respiratory alkalosis and acidosis. These conditions are uncompensated, since the duration of each will be too short for renal compensation to become important. The changes in arterial pH and P_{CO_2} plotted on the Siggaard-Andersen nomogram will define the "CO_2 titration line" of the dog's blood in vivo. Dilute hydrochloric acid will then be injected intravenously to produce a pure metabolic acidosis, while arterial P_{CO_2} (Pa_{CO_2}) and pH are measured to evaluate the results. Hyperventilation and elevated $P_{A_{CO_2}}$ will be repeated during metabolic acidosis to illustrate the altered position of the CO_2 titration line, and if time permits, an equivalent amount of alkali will be injected to return conditions back toward normal.

Materials

1. Dog anesthetized with pentobarbital sodium
2. Recorder with channels for $P_{A_{CO_2}}$, arterial pressure, and electrocardiogram (ECG)
3. Strain-gauge pressure transducer.
4. ECG limb leads
5. Expired CO_2 analyzer (connected to recorder)
6. Blood gas-pH analyzer
7. Variable-speed volume ventilator
8. Infusion pump
9. Glass T tube or cuffed endotracheal tube
10. Source of 95% O_2–5% CO_2
11. 0.9% NaCl solutions with and without heparin
12. 0.1% succinylcholine solution on ice
13. 0.25 N HCl
14. Nylon catheters, stopcocks, syringes (3 ml), heparin (1,000 U/ml), pentobarbital

Procedure

Animal Preparation

A dog that has been anesthetized with pentobarbital sodium (30 mg/kg body wt) will be provided. Place the dog in the supine position and either cannulate the trachea with a glass T tube or insert a cuffed endotracheal tube into the trachea (inflate the cuff to a gas-tight fit). Insert nylon catheters into a femoral vein to infuse drugs and saline and into a femoral artery to monitor blood pressure and collect blood samples. Begin infusing saline into the femoral vein at a rate of about 0.5 ml/min. Attach ECG limb leads and monitor lead II.

The strain-gauge pressure transducer should be calibrated and connected to the femoral artery catheter through *two* three-way stopcocks. The sidearm of the stopcock nearest the pressure transducer will be used for flushing the catheter with heparinized saline and for drawing fresh arterial blood into the catheter before a sample is taken. The arterial sample will be drawn through the stopcock adjacent to the catheter.

Connect the sampling catheter of the CO_2 analyzer to the arm of the T tube that is perpendicular to the trachea (or to a needle in the endotracheal tube) so that the beveled tip of the catheter (or needle) is in the center of the tube facing the expired airflow. Adjust the ventilator (but do not connect yet) to deliver a tidal volume of 15 ml/kg body wt at a frequency of 15/min. The ventilator tubing is connected to each end of a T tube which should be positioned so that it can be connected to the tracheal cannula at a moment's notice.

Equipment Calibration

Check the calibration of the pressure transducer, CO_2 analyzer, and blood gas–pH analyzer at the start of the experiment and about every 0.5 h thereafter.

Paralyzing the Dog's Skeletal Muscles

It is desirable to produce respiratory paralysis when attempting to regulate breathing by a ventilator because an unparalyzed animal can make extra respiratory movements between pump strokes when CO_2 tension is raised high enough. Start infusing succinylcholine solution (1 mg/ml) into the femoral vein at a rate of 0.5 ml/min while recording the arterial pressure and $P_{A_{CO_2}}$ at a slow chart speed. What is the site of action and effect of succinylcholine? Observe the respiratory movements and the $P_{A_{CO_2}}$ and promptly connect the dog's tracheal cannula to the T tube of the ventilator as soon as spontaneous breathing becomes greatly depressed or the $P_{A_{CO_2}}$ rises to about 60 mmHg, whichever happens first. Why might the record of *apparent* $P_{A_{CO_2}}$ actually drop as the succinylcholine takes effect (Fig. 1)? Squeezing the dog's chest to manually expel alveolar gas should verify that the $P_{A_{CO_2}}$ is increased.

[1] The Henderson-Hasselbalch equation in general terms is $pH = pK' + \log_{10}([HCO_3^-]/CO_2)$. This is simplified for measurements in blood by substituting the physical solubility of molecular CO_2 in plasma (0.0301 mmol·l^{-1}· mmHg^{-1}) and the apparent pK' of the HCO_3^-/CO_2 system in plasma at body temperature (6.10). Thus, arterial $pH = 6.10 + \log_{10}$ (arterial plasma $[HCO_3^-]/0.0301 \cdot Pa_{CO_2}$), where Pa_{CO_2} is arterial P_{CO_2} in mmHg. Rearranging to calculate HCO_3^-, arterial plasma [HCO_3^-] = antilog (arterial pH − 6.10) $(0.0301 \cdot Pa_{CO_2})$.

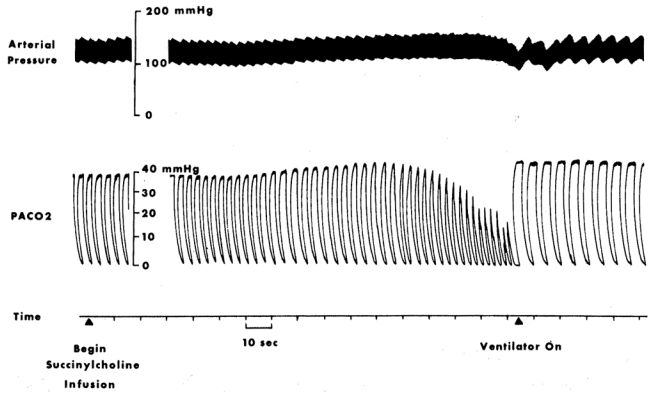

Figure 1
Effect of succinylcholine infusion on PA_{CO_2}. As succinycholine paralyzes the diaphragm, apparent PA_{CO_2} decreases because tidal volume drops and less expired gas reaches the sampling catheter of the CO_2 analyzer. Mechanical ventilation shows that PA_{CO_2} is actually increased.

Because the succinylcholine will also paralyze other muscles besides the diaphragm, the depth of anesthesia can no longer be judged by the usual reflexes. To avoid the possibility that the dog might recover consciousness while paralyzed, inject 30 mg pentobarbital sodium intravenously every 0.5 h by the clock *without fail.* A timer should be used to remind you.

Drawing Arterial Blood Samples Anaerobically

1. Make sure the blood gas–pH analyzer is ready for immediate measurement
2. Connect the sidearm of the three-way stopcock nearest the pressure transducer, via plastic tubing, to a 20-ml syringe containing 5–10 ml heparinized saline (1 U heparin/ml). Fix the syringe point down in a vertical clamp so that when blood is drawn in it will layer below the saline. Draw about 5 ml of blood slowly into the syringe to fill the catheter with fresh arterial blood
3. As this catheter is being filled with blood, someone should be filling the void volume of a 2- to 3-ml syringe with concentrated heparin (1,000 U/ml)
4. The sidearm of the second three-way stopcock is opened just enough to permit blood to drip, and the syringe is attached to the stopcock, being careful not to admit air into the syringe as the attachment is made. The stopcock is then opened and the arterial pressure should fill the syringe with blood
5. Have an assistant force the blood from the 20-ml syringe back into the body, followed by enough heparinized saline to completely clear the catheter
6. Meanwhile, carefully remove the syringe from the stopcock and rotate the syringe a few times to mix the blood and heparin
7. Expel the first 0.2 ml of blood from the syringe and then measure the Pa_{CO_2} and pH. Measurements should be completed within 5 min from the time the blood leaves the artery to avoid changes due to cellular metabolism

Acid-Base Balance Under Normal Conditions

Adjust the frequency of the ventilator until the PA_{CO_2} stabilizes at about 40 mmHg. Record at a paper speed of 1 mm/s. After PA_{CO_2} has remained stable for a minute or two, draw an arterial sample and measure pH and Pa_{CO_2}. As soon as the analyzer is ready again, draw a second blood sample at the same PA_{CO_2} to provide a measure of reproducibility. If the Pa_{CO_2} of two successive samples does not agree within 3 mmHg, then take two more samples and continue taking duplicate samples until your technique provides reproducible results and your animal is stable.

Calculate the plasma $[HCO_3^-]$ from the Henderson-Hasselbalch equation, enter the data in the data table (Table 1), and plot the pH and Pa_{CO_2} on the Siggaard-Andersen coordinates (Fig. 2). Check to see that your $[HCO_3^-]$ lies between the calculated isobicarbonate lines on the nomogram.

Respiratory Alkalosis (Hyperventilation)

Increase the frequency of the ventilator until the PA_{CO_2} is decreased to about 20 mmHg. Record the final frequency. When the PA_{CO_2} has been stable for a minute or two, draw an arterial sample and measure pH and Pa_{CO_2}. Calculate $[HCO_3^-]$ and record the data. What is the relation between the ratio of the ventilator frequencies before and during respiratory alkalosis and the ratio of the corresponding PA_{CO_2} levels? Why must

Table 1
Acid-Base Balance in the Dog

Date Sample; Dog Wt 20 kg; P_B 750 mmHg; Tidal Volume 300 ml

Conditions	Inspired Gas	Time	Ventilator Frequency, min^{-1}	PA_{CO_2}, mmHg	Arterial pH	Pa_{CO_2}, mmHg	$[HCO_3^-]$, meq/l
Normal	Air	1300	15	38	7.41	38	23.4
Normal	Air	1310	15	38	7.40	39	23.4
Respiratory alkalosis	Air	1315	52	17	7.65	17	18.2
Normal	Air	1330	7	42	7.41	40	24.6
Respiratory acidosis	5% CO_2	1345	7	66	7.20	66	25.0
Normal	Air	1400	11	40	7.38	40	22.9
Metabolic acidosis							
Immediate	Air	1415	11	60	7.03	61	15.6
Later	Air	1430	11	40	7.29	40	18.6
Metabolic acidosis and respiratory alkalosis	Air	1445	50	16	7.52	18	14.3
Metabolic acidosis and respiratory acidosis	5% CO_2	1500	6	60	7.13	68	21.9
Normal	Air	1515	12	40	7.30	40	19.1

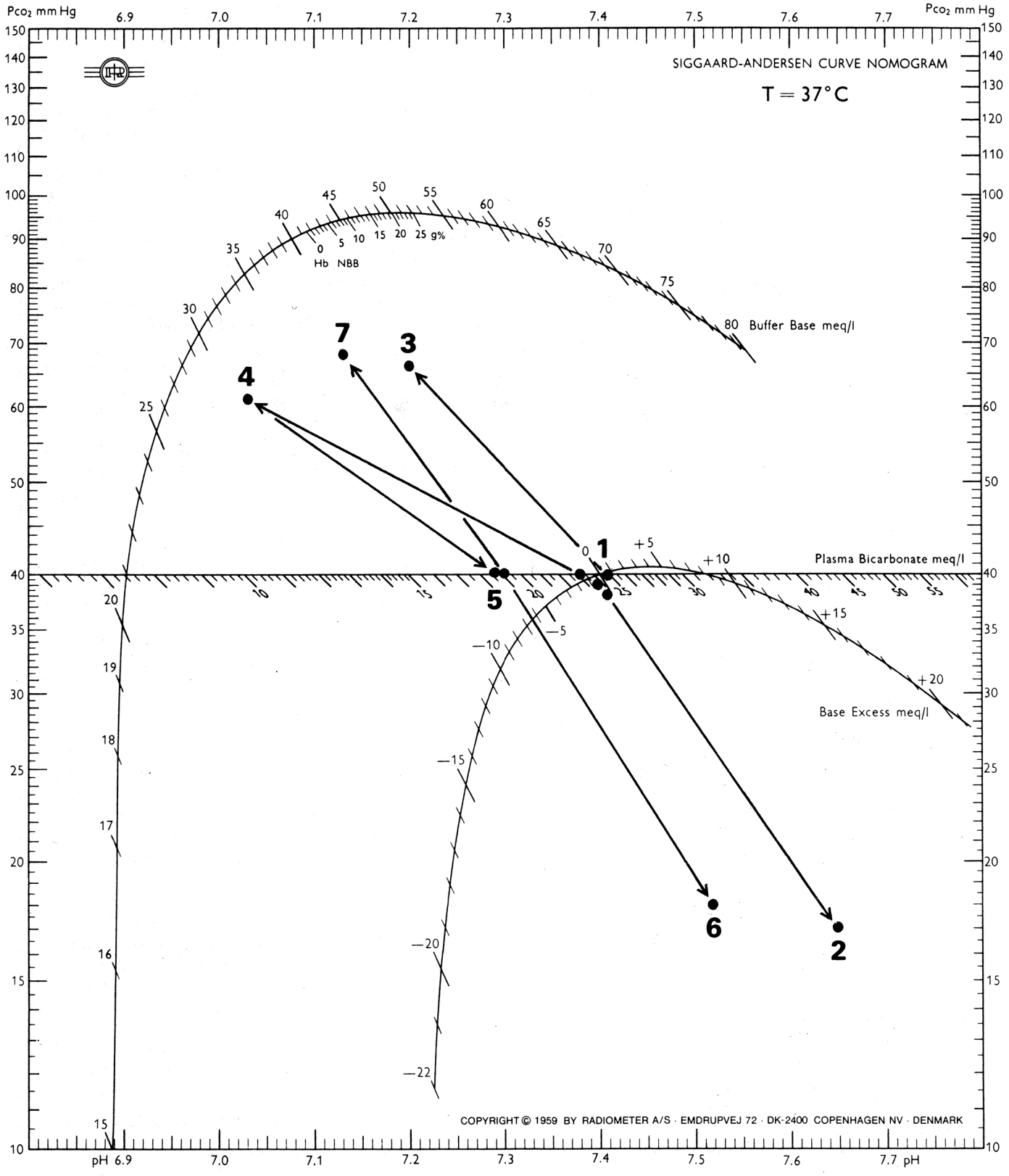

Figure 2

Sample pH and Pa_{CO_2} data (Table 1) plotted on Siggaard-Andersen coordinates. 1, Initial normal conditions; 2, respiratory alkalosis; 3, respiratory acidosis; 4, metabolic acidosis—immediate change; 5, normal conditions after metabolic acidosis (before renal compensation); 6, metabolic acidosis and respiratory alkalosis; 7, metabolic acidosis and respiratory acidosis. Note that "CO_2 titration lines" (2–1–3 and 6–5–7) are nearly parallel before and after acid load and that the slope of these lines differs from isobicarbonate lines because of plasma buffers. (Nomogram reproduced with permission from Radiometer America Inc.)

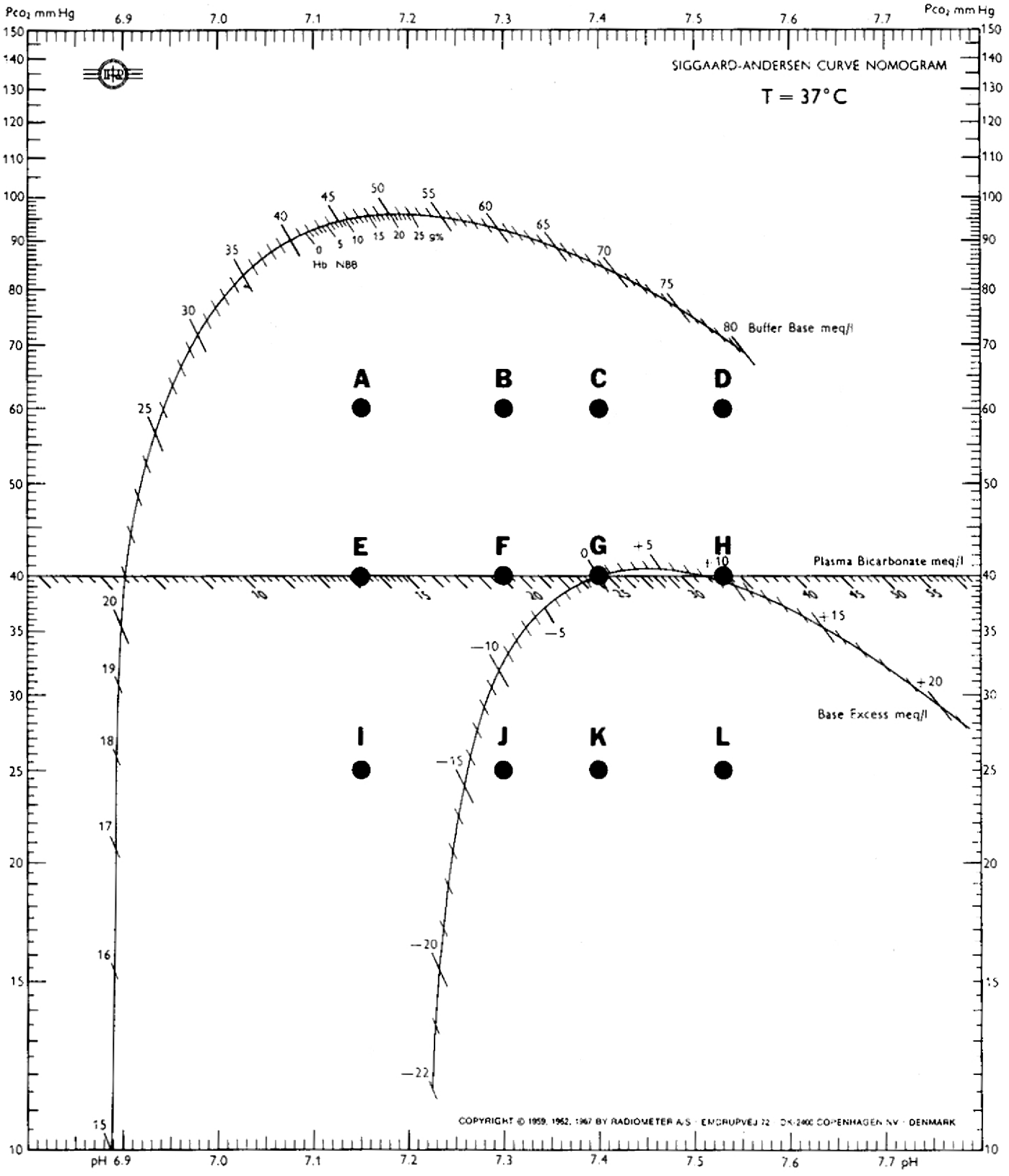

Figure 3
Siggaard-Andersen curve nomogram (reproduced with permission from Radiometer America Inc.) and self-assessment quiz.

the ventilator frequency be more than doubled to halve the $P_{A_{CO_2}}$?

Decrease the ventilator frequency to a level which reestablishes normal $P_{A_{CO_2}}$. Why is the frequency lower now than before respiratory alkalosis? When the $P_{A_{CO_2}}$ stabilizes, draw another arterial sample and measure pH and $P_{a_{CO_2}}$ as before.

Respiratory Acidosis (Hypercapnia)

Start 5% CO_2–95% O_2 flowing into the intake of the ventilator and decrease the ventilator frequency, if necessary, to elevate the $P_{A_{CO_2}}$ to 60 mmHg or higher. Remember that gas sampling will be impaired if frequency is decreased too much. When $P_{A_{CO_2}}$ has been stable for a minute or two, draw an arterial sample.

Return the intake of the ventilator to room air and adjust the frequency to return the $P_{A_{CO_2}}$ to 40 mmHg. Compare the ventilator frequencies before and after acidosis. Also compare the arterial pH and $P_{a_{CO_2}}$ before and after acidosis.

Metabolic Acidosis and Effects of Superimposed Respiratory Changes

Prepare a syringe for an arterial sample. Then while recording the $P_{A_{CO_2}}$, infuse 4.0ml/kg body wt of 0.25 N HCl slowly through the *venous* cathether. As the last 5 ml of the acid are infused, draw an arterial sample and measure pH and $P_{a_{CO_2}}$. Do not change the frequency of the ventilator. After the $P_{A_{CO_2}}$ has been stable for several minutes, draw another arterial sample for analysis. Calculate and plot the results as before, forming additional points on the same graph. How do you explain the transient and subsequent changes in $P_{A_{CO_2}}$, $P_{a_{CO_2}}$, and pH? Would we see the transient increase in $P_{A_{CO_2}}$ after acid infusion in the spontaneously breathing animal? Why? Is compensation to metabolic acidosis complete? How can a rise in $[HCO_3^-]$ be explained when $P_{a_{CO_2}}$ is falling to normal or below?

Repeat the steps to produce respiratory alkalosis and acidosis to see their effects in the presence of metabolic acidosis. After hypercapnia, return the $P_{A_{CO_2}}$ to 40 mmHg and measure pH and $P_{a_{CO_2}}$. Compare the CO_2 titration lines before and after induction of metabolic acidosis (ignoring the transient point during acid infusion). Use these CO_2 titration lines to estimate from the Siggaard-Andersen curve nomogram (Fig. 2) the standard bicarbonate and the base excess of your dog's blood before and after the acid load.

Self-Assessment Quiz

Test your comprehension of the experiment by matching the lettered point or points on Fig. 3 with the following diagnoses (G = normal).

H — Metabolic alkalosis without respiratory compensation

L — Respiratory alkalosis without metabolic compensation

C — Respiratory acidosis with complete metabolic compensation

I, J — Metabolic acidosis with respiratory compensation

A — Metabolic acidosis combined with respiratory acidosis

D — Severe vomiting in a patient with chronically inadequate breathing because of emphysema

B — The effect of inhaling a breath of O_2 and then holding the breath as long as possible

E, F — Metabolic acidosis without any respiratory compensation

K — A normal person who has lived for a year at 17,000 ft above sea level

C — A case of emphysema with a base excess of 10 meq/l

F — A person with a base deficit of 7 meq/l

I — A person with a standard bicarbonate of about 10.5 meq/l

Optional Procedures

Should time be available, the instructor may decide to add one or more of these optional procedures.

Alveolar Ventilation and Respiratory Dead Space

Maintain a given frequency until it is clear that the $P_{A_{CO_2}}$ has stabilized *completely* at a normal value. Quickly change to a different tidal volume and readjust the frequency to bring the $P_{A_{CO_2}}$ back to its former, stable level. From two such successive pairs of tidal volumes and frequencies it is possible to calculate the respiratory dead space, since the effective alveolar ventilation must be the same in the two cases if both produce the same $P_{A_{CO_2}}$ in a true steady state. Calculate the dead space of your dog and apparatus. Check your conclusion by calculating another combination of frequency and tidal volume that should also produce the same $P_{A_{CO_2}}$. One can also investigate the limits of frequency and tidal volume beyond which this relationship fails.

Infusion of Alkali

Infuse 0.25 M $NaHCO_3$ or Na_2CO_3 to neutralize the effect of the previously injected acid or to produce metabolic alkalosis. Try to predict the immediate and long-range effects on the $P_{A_{CO_2}}$. Do not inject NaOH, since this usually kills the animal.

Inhibition of Carbonic Anhydrase

Inject acetazolamide intravenously at a dose of 10 mg/kg body wt and observe the effects on $P_{A_{CO_2}}$, $P_{a_{CO_2}}$, pH, and $[HCO_3^-]$. This should be done as a terminal experiment.

Comment

This exercise is designed to be performed by a group of six students in one 4-h laboratory period. Students should collect arterial blood samples, measure arterial blood gases and pH, record alveolar CO_2 concentrations, and keep data as the instructor conducts the protocol and maintains the stability of the preparation. We have found that this arrangement permits up to three groups of six students each to simultaneously conduct the experiment using one dog, with one instructor to control the preparation and monitor the progress of all groups and several teaching assistants to provide group guidance and answer questions. At the end of the laboratory session, we give the self-assessment quiz and a conference summarizing the important points of the exercise.

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Interactions Between the Renin-Angiotensin System and the Brain

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The brain and the renin-angiotensin system interact in two major ways. On one hand, the brain plays an important role in controlling the release of renin from the kidneys and thus helps to regulate the activity of the renin-angiotensin system. On the other hand angiotensin II, the physiologically active component of the renin-angiotensin system, acts on the brain to produce a variety of cardiovascular, endocrine, and behavioral effects. This second interaction primarily involves circulating angiotensin II formed by the renal renin-angiotensin system, but, in addition, there is some evidence that angiotensin II is also formed locally within the brain by an intrinsic brain renin-angiotensin system. The purpose of this review is to summarize current concepts concerning these interactions between the renin-angiotensin system and the brain.

Neural Control of Renin Secretion

The secretion of renin by the kidneys is controlled by several intrarenal, humoral and neural mechanisms (7). Intrarenal control mechanisms include a renal vascular receptor, which is apparently sensitive to alterations in afferent arteriolar tone, and the macula densa, which is thought to respond to changes in the rate of delivery of sodium and/or chloride to the distal tubule. Humoral factors include angiotensin II, which exerts a tonic inhibitory "short-loop" feedback control on renin secretion, and potassium, which also inhibits renin secretion. Neural control of renin secretion is mediated via the sympathetic nervous system, which influences renin secretion by way of the renal nerves and circulating catecholamines, and via the neurohypophyseal peptide, vasopressin. In this section, the mechanisms by which the renal nerves, circulating catecholamines, and vasopressin influence renin secretion are reviewed. Discussion of other aspects of the neural control of renin secretion can be found elsewhere (7, 35, 65).

The Renal Nerves

Several investigators have reported that reflex increases in renal sympathetic neural activity, or direct electrical stimulation of the renal nerves, increase the rate of secretion (7, 35, 55, 65). These responses are mediated by norepinephrine which in turn increases renin secretion via stimulation of intrarenal α- and β-adrenoceptors.

According to current concepts, the increase in renin secretion produced by α-adrenoceptor stimulation is indirect and probably involves constriction of the afferent arteriole with resultant activation of the renal vascular receptor, together with decreased delivery of sodium and chloride to the macula densa secondary to diminished glomerular filtration rate and increased proximal reabsorption of sodium chloride (7, 65). Under some circumstances, α-adrenoceptor stimulation can actually inhibit the secretion of renin (7). This effect has also been observed in vitro and interpreted as evidence for a direct effect on the juxtaglomerular cell. However, in vivo, α-adrenoceptor stimulation may cause inhibition of the release of norepinephrine from sympathetic nerve endings and thus reduce β-mediated stimulation of renin secretion (7).

In contrast to the effects of α-adrenoceptor stimulation, the effect of β-adrenoceptor stimulation on renin secretion does not appear to be mediated by the renal vascular receptor or macula densa mechanisms. For example, renin secretion is increased when the renal nerves are stimulated at low frequencies that do not alter renal hemodynamics or sodium chloride excretion: this response can be blocked by β-adrenoceptor blocking drugs (7, 35, 65). β-Adrenoceptor stimulation also increases renin release in vitro. A growing body of evidence suggests that these effects on renin secretion result from a direct action of norepinephrine on β-adrenoceptors located on the juxtaglomerular cells (7, 35, 65). It has further been proposed that this direct β-adrenoceptor stimulation of renin secretion is mediated via the activation of adenylate cyclase and the formation of adenosine 3′,5′-cyclic nonophosphate (7, 35, 65).

Considerable effort has been directed toward characterizing the β-adrenoceptor involved in the control of renin secretion. Conflicting results have been obtained, and arguments for important roles of β_1- and β_2-adrenoceptors have been advanced (35, 38, 52, 65). This conflict probably reflects species differences, the effects of anesthetic agents, and other problems of experimental design (52). Definite conclusions are not possible at the present time, but one working hypothesis is that β_1-receptors mediate renin release in rats, dogs, and humans, whereas in cats and rabbits β_2-receptors are more important (35).

Circulating Catecholamines

Renin secretion can be stimulated by intravenous and intrarenal infusions of catecholamines as well as by maneuvers that increase the release of endogenous catecholamines (7, 35, 65). Under some circumstances, the circulating catecholamines may increase renin secretion by the mechanisms just described for locally released norepinephrine. However, there is now considerable evidence that circulating catecholamines increase renin secretion via stimulation of extrarenal β-adrenoceptors.

The involvement of extrarenal β-adrenoceptors in the control of renin secretion was first demonstrated by Reid and associates (66). They found that low doses of the β-agonist isoproterenol, which failed to increase renin secretion when infused into the renal artery, produced marked stimulation of renin secretion when infused intravenously. The stimulation of renin release by intravenous isoproterenol was not secondary to alterations in renal hemodynamics, electrolyte excretion, or

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other factors known to affect renin secretion and was unaffected by renal denervation.

In a subsequent investigation, Johnson and associates (32) demonstrated that the stimulation of renin secretion by epinephrine is also mediated by an action on extrarenal β-adrenoceptors. Their data further suggest that the stimulation of renin secretion in response to insulin-induced hypoglycemia and hemorrhage may be largely mediated by these extrarenal receptors.

The location of the extrarenal β-adrenoceptors involved in the control of renin secretion and the pathways by which they influence renin secretion are presently unknown. Further studies are required to resolve these important issues.

Vasopressin

The sympathetic nervous system is not the only efferent pathway by which the central nervous system influences renin secretion. It has been known for several years that vasopressin inhibits the secretion of renin in several species including rats, dogs, and humans (7, 35, 65). Vasopressin not only suppresses basal renin secretion, but also inhibits the renin secretory responses to sodium depletion, isoproterenol, diuretics, and ureteral ligation (7, 35, 65).

The concentration of vasopressin required to inhibit renin secretion has been established and shown to be in the range observed during water deprivation and nonhypotensive hemorrhage (42). It is therefore likely that the peptide plays a physiological role in the regulation of renin secretion. This conclusion is supported by the finding that plasma renin activity is suppressed in patients with the syndrome of inappropriate secretion of antidiuretic hormone and elevated in rats with hereditary diabetes insipidus (35). In addition, endogenous vasopressin attenuates the renin secretory responses to isoproterenol administration and vagotomy (65).

The precise mechanism by which vasopressin inhibits renin secretion has not been established. The inhibition does not appear to be secondary to vasoconstriction, since it can be produced by doses of vasopressin that do not affect blood pressure or renal blood flow (7). Furthermore, an analogue of vasopressin that lacks vasoconstrictor activity suppresses renin secretion as effectively as vasopressin (43). It has been reported that an antagonist of the vasoconstrictor action of vasopressin increases renin secretion in water-deprived dogs (68), but this apparently was a reflex response, since it was abolished by propranolol. A direct action on the juxtaglomerular cells seems the most likely possibility at this stage but further studies are required.

Central Actions of Angiotensin II

Actions of angiotensin II on the central nervous system include elevation of arterial blood pressure, stimulation of drinking, increased secretion of ACTH and vasopressin, and inhibition of renin secretion by the kidneys (2, 73). There is also evidence that angiotensin II can affect salt appetite, memory, and the uptake or release of certain neurotransmitters in the central nervous system, but these actions are beynd the scope of this review.

Blood Pressure

Increases in blood pressure can be elicited by injecting angiotensin II into the blood supply to the brain, into the cerebral ventricles, or into specific brain regions (2, 10, 73). These responses can be elicited by low doses of angiotensin, which are ineffective when administered intravenously, indicating that they do not result from recirculation or leakage of the peptide into the systemic circulation.

Considerable information is available concerning the central site(s) of action of angiotensin on blood pressure. The increase in blood pressure produced by intravertebral arterial infusion of angiotensin II in the dog is mediated by the area postrema, a circumventricular organ located in the medulla oblongata. Like the other circumventricular organs, this area lacks a blood-brain barrier and is therefore accessible to circulating angiotensin II. Pressor responses can be elicited by microinjections of angiotensin into the area postrema (79), and lesions of the area abolish the increase in blood pressure produced by intravertebral arterial infusions of angiotensin (20, 21, 33).

However, there is evidence that area postrema is not the only central site of action of angiotensin II on blood pressure. For example, the blood pressure response to injection of angiotensin II into a lateral ventricle in dogs is not abolished by area postrema lesions but is blocked by midbrain transection (21). Evidence has been presented that pressor responses to intraventricular angiotensin II are mediated via the subnucleus medialis of the midbrain (10, 73).

In rats and dogs, pressor responses can also be elicited by intracarotid infusion of angiotensin II (26, 62). Since the carotid arteries do not normally perfuse the medulla oblongata in these species, this response cannot be mediated by the area postrema. Instead, there must be a more rostral site of action. Possible sites include the subfornical organ and the organum vasculosum of the lamina terminalis (OVLT), circumventricular organs which, like the area postrema, are accessible to circulating angiotensin. Direct application of angiotensin II to these structures elicits pressor responses, and ablation of these areas reduces the blood pressure responses to systematically administered angiotensin (3, 44, 45).

Information concerning the efferent pathways that mediate pressor responses to centrally administered angiotensin II is also available. In dogs, the increase in blood pressure is due to a combination of an increase in total peripheral resistance, resulting from increased sympathetic discharge, and an increase in cardiac output, secondary to withdrawal of vagal tone to the heart (10, 70). In rats, increased vasopressin secretion (see below) may also contribute to the increase in blood pressure (22).

The centrally mediated pressor responses to angiotensin are probably physiologically important since they can be elicited by concentrations of the peptide that are within the physiological range (11, 62, 70). In addition, lesions of the area postrema, subfornical organ, and OVLT have all been reported to reduce blood pressure responses to systemically administered angiotensin II (3, 45, 69), and area postrema lesions impair cardiovascular responses to hemorrhage (34). The central pressor effect of angiotensin also appears to be important in renal and other forms of hypertension (3).

Drinking

One of the most recently discovered actions of angiotensin II is stimulation of drinking. This dipsogenic action has been observed when the peptide is injected intravenously, into the cerebral ventricles or into certain brain regions (2). Water intake is also increased in a variety of situations where circulating angiotensin levels are high; in some of these situations, water intake can be reduced by agents that block the formation or actions of angiotensin II (2).

Considerable effort has been directed to determining the site of the dipsogenic action of angiotensin. Since the response can be elicited by blood-borne angiotensin II, attention has been focused on the circumventricular organs. The first of these to be implicated was the subfornical organ, and there is now little doubt that this is an important receptor site for angiotensin-induced drinking (75). The OVLT may also play a role (39). There is no evidence that the area postrema is involved in angiotensin-induced drinking.

The extent to which the renin angiotensin system participates in the physiological control of drinking is not clear. Evidence in favor of a physiological role includes the observation that drinking can be elicited by doses of angiotensin II which produce circulating angiotensin II concentrations within the range seen in hypovolemic states such as sodium deficiency and hemorrhage (12, 62, 78). Additional investigation is required to precisely define the role of the renin-pangiotensin system in the regulation of water intake.

Vasopressin Secretion

There is now general agreement that angiotensin II can increase vasopressin secretion when administered into the cerebral ventricles of dogs and rats (74). Systemically administered angiotensin has also been reported to increase vasopressin secretion, but there is still controversy concerning the effect of circulating angiotensin on vasopressin secretion (6, 25, 37, 57, 62, 74). There have been reports that vasopressin secretion can be stimulated by relatively low doses of angiotensin; but in general high doses are required, and even these are not always effective. These discrepancies may be due in part to the use of anesthetics, differences in the state of hydration, or other differences in experimental design.

The site at which angiotensin acts to increase vasopressin secretion has not been definitely established. Sites that have been implicated include the supraoptic nucleus (51) and posterior pituitary (16), but further studies are required.

At present, it appears unlikely that circulating angiotensin plays an important role in control of vasopressin secretion. As noted above, the doses of angiotensin II required to increase vasopressin secretion are generally quite high, producing plasma concentrations above the range generally considered to be physiological. It could be argued that the increase in blood pressure produced by these infusions exerts an inhibitory effect on vasopressin secretion and that, in hypovolemic states where plasma angiotensin II levels are increased but blood pressure is unchanged or decreased, the renin-angiotensin system may exert a significant effect on vasopressin secretion. Against this, however, is the observation that vasopressin secretion increases during hemorrhage even when changes in renin secretion are prevented (5). Moreover, the increase in vasopressin secretion in dogs subjected to hemorrhagic shock is not prevented by pretreatment with a converting-enzyme inhibitor (50).

ACTH Secretion

As in the case of vasopressin, there are discrepancies concerning the effects of angiotensin on the secretion of ACTH. Intraventricular angiotensin II appears to increase ACTH secretion (46, 59), but increases or decreases have been observed in response to systemically administered angiotensin (13, 57, 58). There is also disagreement concerning the dose required to stimulate ACTH release. In some studies low doses were effective (57); in others, very high doses were required (62). Further studies are required, but in the meantime it appears unlikely that the renin-angiotensin system plays an important role in the control of ACTH secretion.

Proposed sites of action of angiotensin on ACTH secretion include the median eminence (17) and the anterior pituitary (46). Angiotensin may also increase ACTH secretion indirectly by stimulating vasopressin release, since it is known that vasopressin is a corticotropin-releasing factor. The recent observation that the ACTH response to centrally administered angiotensin II is subnormal in vasopressin-deficient Brattleboro rats is consistent with this possibility (18).

Renin Secretion

Renal renin secretion is decreased when angiotensin II is administered into the cerebral ventricles or into the blood supply to the brain (41, 62). The inhibition produced by intraventricular angiotensin appears to be mediated by vasopressin inasmuch as it is absent in hypophysectomized dogs (41) and in Brattleboro rats (18). On the other hand, it is unlikely that the suppression of renin secretion produced by infusion of angiotensin into the carotid or vertebral arteries is mediated by vasopressin (62). This suppression could be a consequence of increased blood pressure (7) or decreased renal neural activity (11), but further studies are required.

The Brain Renin-Angiotensin System

During the past decade, there has been increasing interest in the proposal that brain contains an intrinsic renin-angiotensin system (53, 60, 61). This proposal is based primarily on two sets of observations: *1*) the components required for the formation of angiotensin II—a renin-like enzyme, angiotensinogen (renin substrate), and converting enzyme—are present in the brain; and *2*) as described in the preceding section, angiotensin II can exert a variety of actions on the brain. The purpose of this section is to summarize what is known about the components of the renin-angiotensin system in the brain and to critically review the evidence that they interact in vivo to form a functional brain renin-angiotensin system.

Reninlike Activity in the Brain

An enzyme with reninlike activity was discovered in extracts of brain in 1971 (19). This enzyme was clearly different from renal renin, particularly with regard to pH optimum, but it was not until 1976 that the enzyme was identified as the lysosomal acid protease cathepsin D ((8, 23, 24). It seemed unlikely that cathepsin D would function as an angiotensin-forming enzyme in vivo, and for the first time the existence of a brain renin-angiotensin system was seriously questioned (60, 61).

The cathepsin D problem was partially circumvented in 1978 with the demonstration that cathepsin D is not the only enzyme in the brain with reninlike activity (27). It has now been established that the brain contains several proteases capable of forming angiotensin under the appropriate conditions (9, 28, 47). One of these is a neutral protease with biochemical properties quite similar to those of the renal enzyme; this enzyme can be inhibited by an antibody raised against renal renin. The concentration of the enzyme in brain (approx 250 pg angiotensin I • g tissue^{-1} • h^{-1}) is very low compared with the plasma renin activity (27). The enzyme is widely distributed throughout the central nervous system with highest concentrations occurring in the pineal, choroid plexus, and adenohypophysis (4, 15, 30, 77). In some parts of the brain, the enzyme is present within neurons.

Angiotensinogen

Angiotensinogen is present in cerebrospinal fluid and brain tissue of several species including humans (31, 40, 48, 56, 60, 80). Biochemiclly, brain angiotensinogen is very similar to the plasma substrate, although differences in carbohydrate composition (56, 64) and immunological characteristics have been reported (31, 49). Angiotensinogen is distributed widely throughout the brain, but small regional differences in concentration have been noted. Cell fractionation studies have demonstrated that angiotensinogen is present in the soluble fraction and may be confined to the extracellular fluid (48).

Factors affecting angiotensinogen concentration in the central nervous system have been studied. The angiotensinogen content of certain brain regions of rabbits decreases following adrenalectomy; and this can be reversed by treatment with adrenal cortiocosteroids (80). On the other hand, angiotensinogen concentration in the cerebrospinal fluid of dogs does not change when plasma angioteninogen levels are increased by dexamethasone treatment or nephrectomy (61).

The origin of the angiotensinogen in the central nervous system is still not known. Possibilities that have been considered include local synthesis within the brain and entry from the blood during the process of cerebrospinal fluid formation. Evidence in support of a peripheral origin includes the close similarities between brain and plasma angiotensinogens and the failure to find angiotensinogen in granule fractions of the brain (48). On the other hand, the fact that there are differences between central and peripheral angiotensinogens could be interpreted as evidence for local synthesis. Furthermore, as noted above, cerebrospinal fluid angiotensinogen levels do not change when plasma angiotensinogen levels are increased, and in addition radioiodinated angiotensinogen does not enter the cerebrospinal fluid from the blood (48). Finally, homogenates of rat brain release angiotensinogen when incubated in vitro (72). Thus the bulk of the evidence suggests that angiotensinogen is synthesized by the brain but further investigation is required.

Converting Enzyme

Converting enzyme is present in the central nervous system as well as in every other tissue that has been studied. The levels and distribution of converting enzyme in the brain have usually been studied using biochemical assays. In general, these assays have utilized artificial substrates, and it is important to note that results obtained in these assays may differ from those which utilize angiotensin I as the substrate.

In a recent study in this laboratory (1), converting enzyme was localized in the brain by immunocytochemistry. The enzyme was found in capillary endothelial cells throughout the brain, on the ventricular surface of epithelial cells of the choroid plexus, and in the subfornical organ. With the exception of the subfornical organ, no converting enzyme was detected in neural tissue. Similar results have been reported by Rix et al. (67).

Problems with the Brain Renin-Angiotensin System Hypothesis

It is clear from the preceding section that a reninlike enzyme, renin substrate, and converting enzyme are present in the brain. The question is whether these components interact in vivo to form angiotensin II. Several problems exist, and the most significant of these are discussed next.

First, although some renin activity can be separated from cathepsin D, it is clear that most reninlike activity measured in brain extracts at low pH is due to cathepsin D and that the level of noncathepsin D reninlike activity is very low. It could be argued that this small amount of renin activity might be important if it were localized in discrete areas: however, it has been reported that the enzyme is distributed quite diffusely throughout the central nervous system (77).

Regardless of the concentration and distribution of the reninlike enzyme, there have been no convincing demonstrations that the enzyme functions as an angiotensin-forming enzyme in vivo. Attempts to demonstrate such activity have involved injecting synthetic and natural renin substrates into the brain. Angiotensin formation has been observed following intraventricular injections of the artificial tetradecapeptide renin substrate, but it is now clear that this resulted from an action of converting enzyme rather than of renin (76). Injections of natural renin substrates have yielded unconvincing or negative results (29, 63, 76).

If brain renin does form angiotensin I in vivo, it would presumably do so in or near the neurons where the enzyme has been localized.This presents an additional problem, since as described above brain converting enzyme does not appear to be present in neural tissue but is confined to capillaries and choroid plexus epithelium. How then is angiotensin II formed? It might conceivably be formed by a pathway that does not require converting enzyme, but there is no evidence that this is the case. The angiotensin I might somehow be

transported to the choroid plexus and converted to angiotensin II there, but again this seems unlikely, especially since the concentration of angiotensin I and II in the cerebrospinal fluid is very low (61). It is possible that there is a form of converting enzyme which is not recognized by the pulmonary converting enzyme antibody used in the localization studies: however, this does not appear to be the case, since antisera raised against the pulmonary enzyme effectively inhibit brain converting enzyme activity (54). Finally, it is worth noting that converting enzyme has a broad substrate specificity, and it is possible that the brain enzyme functions in the metabolism of peptides such as the enkephalins, bradykinin, and substance P rather than in conversion of angiotensin I to angiotensin II.

These problems raise serious questions concerning the hypothesis that angiotensin II is formed in the brain. The most direct test of the hypothesis, however, is to determine if angiotensin II is actually present in the central nervous system. Angiotensinlike activity has been measured in brain extracts and cerebrospinal fluid by bioassay and radioimmunoassay techniques (53, 60, 61). The results that have been obtained vary over a very wide range, and there is little or no agreement between the results of bioassays and immunoassays. This lack of agreement apparently results from several problems including nonspecificity of bioassay methods and destruction of tracer by peptidases in radioimmunoassay procedures (61). When steps are taken to avoid these problems, angiotensinlike activity in brain extracts is reduced to low or undetectable levels (61, 80).

Immunocytochemistry has also been used during recent years in attempts to detect and localize angiotensin II in the brain. Staining of cell bodies and nerve fibers has been observed in various brain regions, and this has further stimulated interest in the concept of a brain renin-angiotensin system (1, 14, 36, 53). However, there are problems. For example, although we obtained positive results with the antibodies used by Fuxe et al. (14), we have consistently obtained negative results with another 12 angiotensin II antibodies (1). These antibodies all have high titers and work well in radioimmunoassays. In addition, it must be emphasized that immunocytochemistry alone cannot prove that a substance is present in a tissue; extraction and biochemical characterization is also required. This is particularly true in the case of angiotensin II, since most angiotensin II antibodies cross-react with angiotensin I, angiotensin III, and the tetradecepeptide renin substrate (1).

The results of a recent investigation of the nature of the angiotensin II immunoreactivity in human cerebrospinal fluid and rat brain are therefore significant. Semple and associates (71) studied the angiotensin II immunoreactivity in human cerebrospinal fluid using paper chromatography. They demonstrated that the immunoreactivity was not angiotensin I, angiotensin II, or its fragments and therefore concluded that it is an immunoassay artifact. More recently, Meyer and associates (47a) measured low levels of angiotensin II immunoreactivity in extracts of rat brain using three angiotensin II antibodies, two of which had previously given positive results in immunocytochemistry. Subsequent characterization with high-pressure liquid chromatography revealved that the immunoreactivity was neither angiotensin II nor angiotensin III, and it was therefore concluded that these peptides are not present in the brain.

It is therefore clear that there are problems and difficulties, and the question of whether there is an intrinsic brain renin-angiotensin system remains open for further investigation.

Conclusions

1) The central nervous system regulates renal renin secretion via the renal sympathetic nerves, circulating catecholamines and vasopressin. Norepinephrine released from the renal nerves increases renin secretion by stimulating intrarenal α- and β-adrenoceptors. The increase in renin secretion produced by circulating catecholamines, on the other hand, is primarily mediated via extrarenal β-adrenoceptors. Vasopressin inhibits renin secretion, possibly by a direct action on the juxtaglomerular cells.

2) Angiotensin II formed by the renal renin-angiotensin system can act on the central nervous system to elevate blood pressure, stimulate drinking, increase the secretion of ACTH and vasopressin, and inhibit the secretion of renin by the kidneys. Current evidence indicates that the central pressor and dipsogenic effects of circulating angiotensin II are physiologically important: the significance of the actions on ACTH, vasopressin, and renin secretion is less clear at the present time.

3) The central actions of angiotensin II can also be elicited by intracranial injections of the peptide and could conceivably represent actions of a brain renin-angiotensin system. However, the existence of a brain renin-angiotensin system has not yet been conclusively demonstrated, and conclusions concerning the possible functions of such a system should be made with caution.

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Repeated Cardiac Output Determination in the Experimental Animal

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In physiological and pharmacological experimental animal studies there is a need for an inexpensive, easily applied method for measuring cardiac output as often as every few minutes. The popular thermal dilution method does not lend itself to repeated use in the student laboratory mainly because it uses relatively expensive equipment. Saline, the first indicator to be used for cardiac output, is extremely attractive in that the indicator is inexpensive; the detector (a conductivity cell) is also inexpensive and can be reused. Moreover, the recording equipment (an impedance recorder) is standard in most laboratories. This paper describes the use of a flow-through conductivity cell detector with hypertonic saline as the indicator and an electrical calibration technique that does not require preparation of calibration solutions. The cardiac output determinations in the dog were verified using the direct Fick method. In the Appendix is presented a simple circuit for an impedance recorder.

Methods

Conductivity Cell

At the heart of the method is a flow-through conductivity cell shown in Fig. 1 (5), which is placed in an arteriovenous shunt (Fig. 2) to eliminate the need for using a withdrawal pump. The conductivity cell (Fig. 1) consists of two stainless steel electrodes mounted at the end of a plastic cylindrical chamber 1.0 cm long and 1.13 cm in diameter. Blood enters tangentially at one end of the cell, spirals around the cell, and exists tangentially at the other end, thereby providing a short washout time. The dimensions were chosen to obtain a length-to-area ratio of 1.0, which is the conductivity cell constant. Use of this conductivity cell has been reported by Geddes et al. (5), who compared saline cardiac output with dye cardiac output values. This report describes validation using the Fick method.

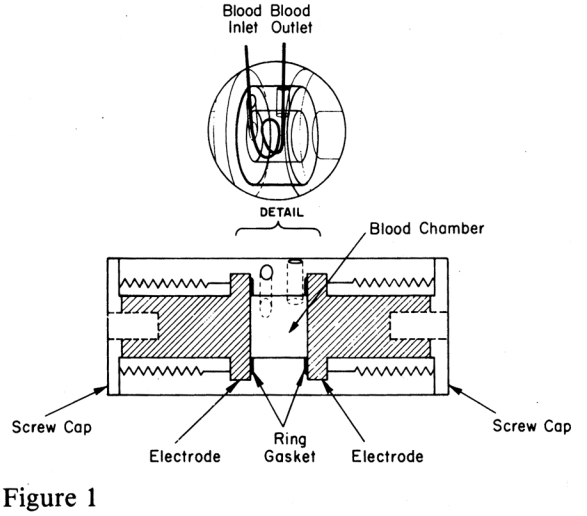

Figure 1
Flow-through conductivity cell.

Electrical Calibration

The theory underlying the electrical method of calibrating a conductivity cell in terms of indicator concentration was presented by Geddes et al. (4). The method consists of recording a calibration signal by momentarily short-circuiting a calibrating resistor in series with the conductivity cell used to inscribe the dilution curve. By knowing the conductivity cell constant (length, L, divided by area, A), and the manner in which blood decreases its resistivity with the addition of sodium chloride ($\Delta\rho/\Delta C$), the concentration change (ΔC) equivalent to the deflection on the recording by short-circuiting the calibrating resistor (ΔR) is given by

$$\Delta C = \frac{\Delta R}{\Delta\rho/\Delta C} \times \frac{A}{L} \qquad (1)$$

where $\Delta\rho/\Delta C$ is the manner in which blood decreases its resistivity ($\Delta\rho$) when sodium chloride is added to produce a change in the concentration (ΔC). In a previous study (Geddes et al., 1974), $\Delta\rho/\Delta C$ was found to be a function of packed cell volume (H) and for dog blood at 37°C is given by the following expression

$$\Delta\rho/\Delta C = 3.77\, e^{0.048H} \qquad (2)$$

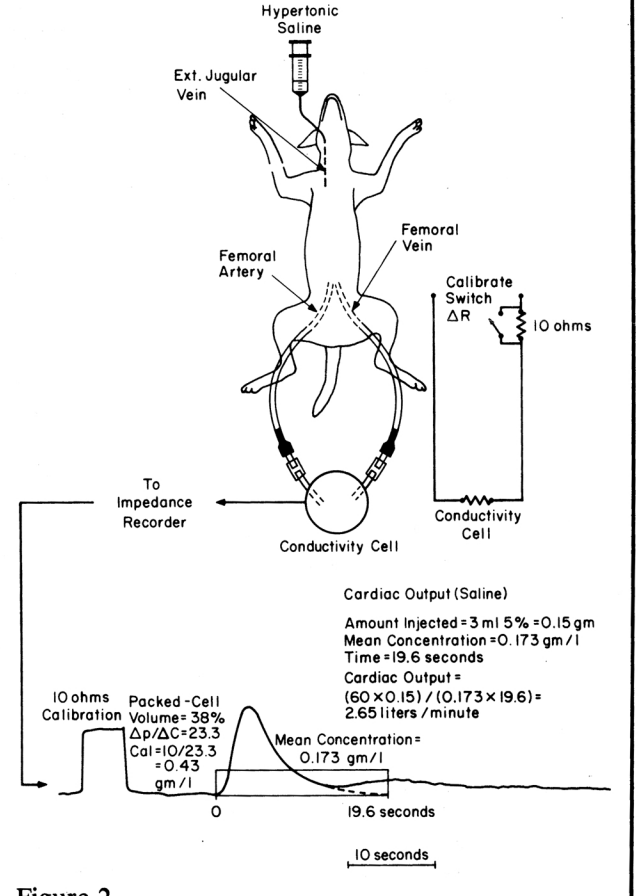

Figure 2
Arrangement of equipment to record dilution curves using saline as indicator and flow-through conductivity cell placed in an arteriovenous shunt. A sample calculation for cardiac output is illustrated below.

Therefore the concentration change (ΔC) equivalent to the deflection obtained by short-circuiting the series resistor (ΔR) is

$$\Delta C = \frac{\Delta R}{3.77\, e^{\,0.048H}} \times \frac{A}{L} \qquad (3)$$

Cardiac output is calculated by first inscribing the dilution curve, correcting it for recirculation by semilogarithmic plotting, and extrapolating to 1% of the maximum height of the dilution curve to find the end of the curve in the manner described by Kinsman et al. (6). Then the area and mean height can be determined and converted to mean concentration ($\bar{C}$) and to duration (t). Cardiac output (CO) is given by the Stewart-Hamilton equation

$$CO = \frac{60\, m}{\bar{C}\, t} \qquad (4)$$

where m is the number of grams of sodium chloride injected as the indicator and $\bar{C}$ is the mean concentration of the corrected dilution curve which has a duration of t seconds.

The value for $\bar{C}$ is obtained by scaling from the electrical calibration deflection, which is converted into concentration change as just described. Figure 2 illustrates a typical dilution curve and use of the electrical calibration method, along with a sample cardiac output calculation.

Fick Cardiac Output

The direct Fick method was used as the standard for comparison. Its use requires measuring the respiratory O_2 uptake ($\dot{V}O_2$) per minute and measuring the arteriovenous (a-v) O_2 difference. The cuffed endotracheal tube was connected to a valved 5-liter spirometer filled with O_2 and containing a CO_2 absorber. The excursions of the bell were recorded graphically to determine the $\dot{V}O_2$ per minute by the diminution of volume in the spirometer. Usually 5 min of $\dot{V}O_2$ were measured. The spirometer recording was calibrated with a 500-ml syringe.

Blood samples for determining the a-v O_2 difference were obtained from a catheter advanced beyond the apex of the right ventricle to the outflow track (venous sample) and a femoral artery catheter (arterial sample). The O_2 content of these samples was measured with the Lex-O_2-Con (Lexington Instruments, Waltham, MA). The gas law was used to correct the volumes to STPD. This procedure was required because the blood O_2 analyzer provided a reading at STPD conditions. The cardiac output was calculated as follows

$$CO = \frac{\dot{V}O_2/\text{min}}{\text{a} - \text{v}\ O_2\ \text{difference}} \qquad (5)$$

Saline Cardiac Output

Eleven mongrel dogs, ranging in weight from 13.5 to 32.5 kg, were used in this study. The animals were anesthetized with pentobarbital sodium (30 mg/kg iv) and intubated with a cuffed endotracheal tube. Cardiac output was increased by an intravenous drip of epinephrine and decreased by controlled hemorrhage. Heparin (2 mg/kg) was given to prevent clotting in the catheter and flow-through conductivity cell.

Two milliliters of 5% saline were used as the indicator and were injected into the right ventricle. Blood resistivity was monitored continuously with an impedance recorder (Narco Bio-Systems, Houston, TX). The Appendix presents a simple circuit for the same purpose. Momentarily shorting a 5-Ω resistor in series with the conductivity cell provided electrical calibration of the dilution curve. The value for $\Delta\varrho/\Delta C$ needed to convert the electrical calibration to a concentration change was determined by first measuring the packed cell volume (H) of an arterial sample using a centrifuge. The value for $\Delta\varrho/\Delta C$ was then calculated from *eq. 2*.

Saline-dilution curves were inscribed before and after determining the $\dot{V}O_2$. The dilution curves were recorded on a graphic recorder. Figure 2 illustrates a typical dilution curve along with sample calculations.

Results

Figure 3 illustrates the rapidity with which dilution curves can be obtained if desired. Figure 4 presents the relationship between saline cardiac output and Fick cardiac output. The dashed line is the line of equal values, and the solid line is the least-squares line for all data points. The saline cardiac output values on the average were about 13% above those obtained by the direct Fick method.

Discussion

A considerable amount of research has been devoted to the use of saline as an indicator. Chinard et al. (1) pointed out that the use of "diffusible" indicators, such as saline, will overestimate cardiac output due to loss of

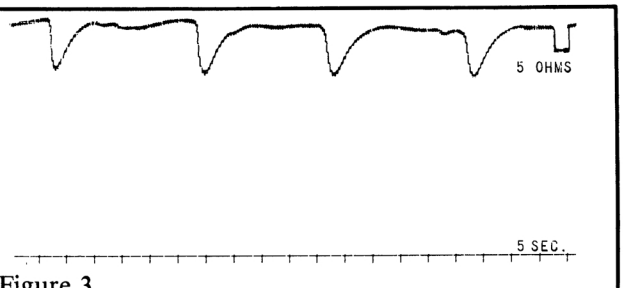

Figure 3
Typical multiple dilution curves taken about 0.5 min apart along with a 5-Ω electrical calibration signal.

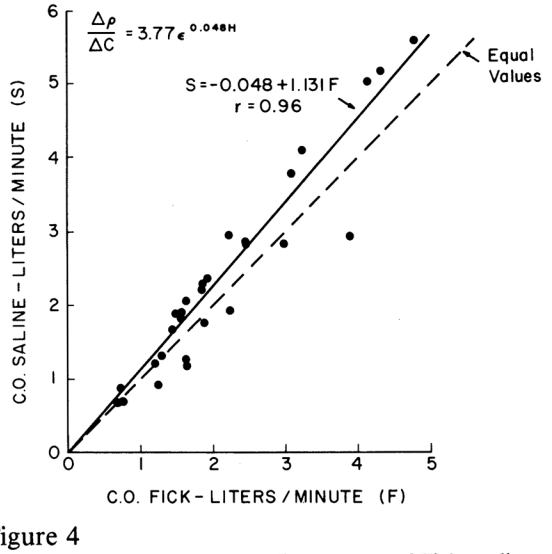

Figure 4
Relationship between saline cardiac output and Fick cardiac output values in the dog.

indicator passing through the pulmonary circuit. Smith et al. (7) summarized this research, which confirmed that the use of saline slightly overestimates the values for cardiac output when compared with values obtained with other nondiffusible indicator methods.

It is useful to note that the use of a diffusible indicator, such as saline, will overestimate cardiac output if there is a capillary bed between the injecting and measuring sites. This fact is unimportant with the thermal dilution method, which employs inection of a diffusible indicator into the right atrium and detection of the dilution curve in the pulmonary artery, there being no intervening capillary bed. Geddes and Babbs (2) have described an analogous technique using saline to measure cardiac output accurately in low-output conditions. The saline indicator was injected into the left ventricle and detected in the abdominal aorta. A good correlation was obtained with the Fick cardiac output values.

In this study the electrically calibrated conductivity method, used with an arteriovenous shunt provides cardiac output values that are only slightly in excess of those measured by the Fick method. The method does not require mixing blood samples with an indicator to calibrate the conductivity cell. By placing the conductivity cell in an arteriovenous shunt, no withdrawal pump is required to record dilution curves that are obtained without blood loss. Therefore dilution curves can be obtained every few minutes if desired. Saline is an inexpensive indicator that needs no special handling. Finally, in collateral studies we have found that the calibration factor ($\Delta\varrho/\Delta C$) is very similar for nine species (baboon, camel, sheep, cow, dog, human, monkey, cat, and goat) and has an average value, $\Delta\varrho/\Delta C = 4.54\ e^{0.036H}$. However, verification studies for cardiac output have not been conducted using this value for $\Delta\varrho/\Delta C$.

Appendix

Impedance Recorder

Figure 5 illustrates a simple 9-V battery-operated impedance recorder which can be used for recording dilution curves as well as a wide variety of physiological events that produce an impedance change (3). The circuit is easily fabricated using standard off-the-shelf components.

The 10-kHz sine-wave oscillator, formed by A_1, is transformer coupled to the conductivity cell via two 100-kΩ resistors, which form the current terminals 1 and 4. For the present application (bipolar measurement), point 1 is joined to 2 and point 3 to 4. Removing the connections 1–2 and 3–4 allows tetrapolar recording if desired. A current of 100 μA flows through the conductivity cell connected between 2 and 3. With both bipolar and tetrapolar connections the same constant current is delivered. To adjust the circuit, a 1,000-Ω resistor. This sets the 100-μA measuring current.

The impedance-dependent voltage appearing across terminals 2 and 3 is amplified differentially by A_3–A'_3 and A_4. The single-sided output provided by A_4 is band-pass filtered at a center frequency of 10 kHz by A_5. Detection of the impedance signal is accomplished by A_6, which feeds A_7, a summing amplifier that provides an output referenced to zero for any basal impedance in the range of 25–1,000 Ω using P_2. Balancing for the basal impedance (referencing) is accomplished by deriving a signal from the oscillator (A_1) via the 200-kΩ balance control, which feeds a signal into A_8, the output of which is detected by A_9. The output of the detector A_9 is subtracted from the detected impedance signal by A_7, which also filters the signal, limiting the rise time (transient response) to 0.01 s. The output is 10 mV for a 1-Ω change in impedance at the input terminals.

The drain from each of the two 9-V transistor batteries is less than 3 mA. These batteries are rated at 325 mA • h. If battery operation is not desired, a ±9-V regulated supply can be used.

The authors express their thanks to D. Santel, undergraduate in Interdisciplinary Engineering, L. Kelly, undergraduate in Chemical Engineering, and C. Wines, undergraduate in Electrical Engineering for their assistance.

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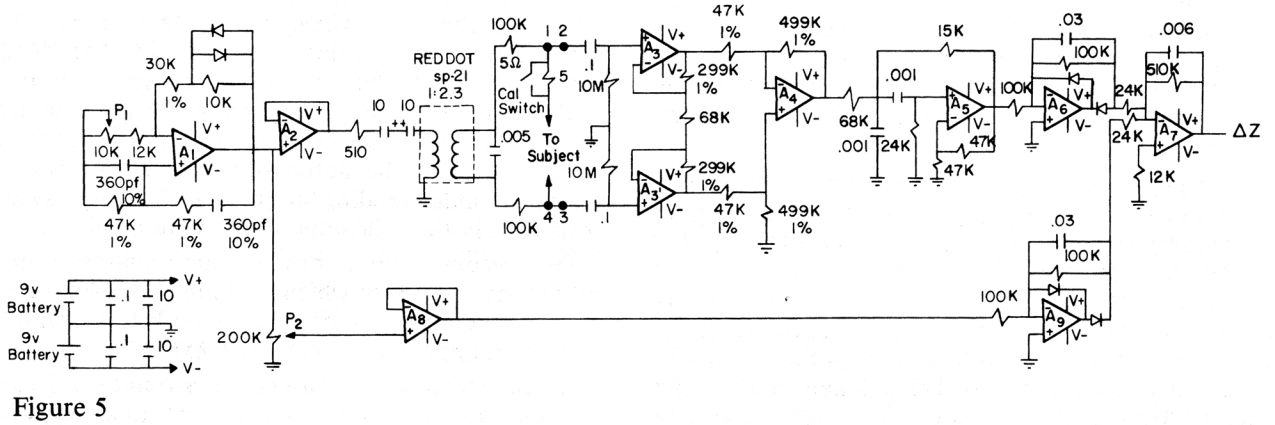

Figure 5

Simple battery-operated impedance recorder. *Circuit specifications: 1*) capacitors are in μF, unless otherwise stated; *2*) resistances are in Ω, 0.25 W, 5%, unless otherwise stated; *3*) operational amplifiers are TL 061's; *4*) 1% resistors are RN600 series; *5*) diodes are IN 4148's.

The Status of Precollege Science Teaching

As a representative of the American Physiological Society, I attended the National Convocation on Precollege Education in Mathematics and Science, held at the National Academy of Sciences in Washington, DC, May 12 and 13, 1982. This national gathering was called to focus on the continuing erosion in precollege teaching of science and mathematics, its causes, and possible remedies. As evidence of serious national concern in this regard, the convocation was addressed by high-ranking members of the Administration, state government, leaders in business, labor, and science, and educators.

The deterioration in science and mathematics education represents a clear and present danger for our defense efforts, jeopardizes our leading position in science and technology, and may result in raising a generation of Americans who are scientifically illiterate and unable to meet the challenges of high technology, computers, integrated circuits, robotics, etc.

In recent years, the decline in achievement scores on student tests and the drop in enrollments in high school science and math courses have been well documented. Of equally critical nature is the fact that there is a signficant decline in the number of high school mathematics and science teachers being trained, and of those with such training, a decreasing number go into teaching, choosing business or industrial employment instead. Half the people teaching mathematics and science at the secondary level this year were unqualified.

There was general agreement that action on this deplorable state of affairs was long overdue. Some factors contributing to the problem were discussed. One factor is the reduction in Federal spending. When President Carter left office in January 1981, he requested $112 million for science and engineering education programs at all levels financed through the National Science Foundation. Mr. Reagan proposes this year to cut the amount to $15 million, most of which would go exclusively for college programs. Another factor discussed is the increased social pressures of the 1960s and 1970s for educational equality leading to a sacrifice of quality and excellence.

Members of the Administration—Edwin L. Harper, White House domestic policy adviser, Defense Secretary Casper W. Weinberger, and Secretary of Education T.H. Bell—offered little hope that a Federal initiative will figure in any major way in correcting the situation. Remedial measures will have to be financed and implemented at the state and local grass-roots level, in partnership with business and industry.

Among specific proposals suggested were the following: expansion of schools that specialize in mathematics and science; establishment of more academically and professionally oriented high schools; encouragement of state and local authorities to increase science requirements; a differential pay scale for science teachers, who are in short supply. Although the problem is a very serious one, some steps are being taken in the right direction towards its alleviation. The North Carolina School of Science and Mathematics in Durham is a residential public high school open to all qualified students at no cost. This new program has already succeeded in identifying and training a pool of academically gifted young people and has produced a substantial number of National Merit Scholarship nominees. As evidence of changing national attitudes, Florida and California, known in the 1960s for their lenient high school graduation requirements, have recently reverted to their former stringent policies. Corporate financial assistance enables the Houston school district to provide supplementary pay—in in the amount of $2,000 per year—to the salaries of qualified science and mathematics teachers.

Much concern was voiced about finding new creative ways to teach scientific concepts to children even before the secondary level so as to sustain their innate curiosity and channel it constructively. As Carl Sagan elaborated on this theme, in recent years we have witnessed an unprecedented explosion of scientific and technological knowledge. We can teach our children to use this knowledge to attain previously unheard of goals for the betterment of all society, or if we ignore the signs of impending danger, we do so at our collective peril.

Judy A. Spitzer

Book Reviews

Introduction to Human Physiology (2nd ed.). M. Griffiths

New York: Macmillan, 1981. 524 pp., illus., bibliographies, index, $19.95

This is one of a number of texts currently available for those studying physiology at a relatively elementary level. It is suitable for students of paramedical subjects and those reading physiology as an ancillary subject or could be used as an introductory text for medical students.

The book opens with a section devoted to the function of cells, commencing with a brief consideration of the constancy of the internal environment, a theme which is maintained. The subject matter has been subdivided, in the traditional manner, according to organs and their function. There is little emphasis on the integrated activity of various systems, although chapters have been included, for example, on growth and aging and on regulation of the composition and volume of extracellular fluids, in which various threads have been brought together. A helpful approach has been to split the discussion on the central nervous system into two sections, a general section appearing early in the book and a more detailed one later. The early section, devoted largely to the autonomic system, provides the necessary understanding of the way in which systems dealt with in the following sections are regulated.

This second edition has an additional section on elementary chemistry which probably adds little to the value of the text. Students might be better advised to consult an appropriate chemistry text.

At the end of each chapter are a number of useful questions on the topic and also a short summary, which could possibly have been placed at the beginning of the chapter to act as a synopsis. The figures are quite good

but are not always the book's strong point. In addition there are a number of helpful tables and a glossary that immediately precedes the index.

In a book of this length there is relatively little room for consideration of experimental proof of a hypotheses. Fortunately some details have been given, as for example methods of recording from excitable tissue. Again there is little opportunity for giving clinical illustrations, but there are some of current interest, as for example a brief consideration of the technique of amniocentesis. Although it cannot be said that this text is greatly superior to any other available, it nevertheless gives a sound introduction to the subject.

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Middlesex Hospital Medical School, London

Locomotion and Energetics in Arthropods. C.F. Herreid and C.R. Fourtner (Editors)

New York: Plenum, 1981. 546 pp., illus., index, $59.50

This book contains the proceedings of a symposium held by the American Society of Zoologists in December 1980. It consists of 19 papers, all but two of which cover some subset of the subject. With the exception of the fine through brief review of the evolutionary and morphological aspects of limb development by Hessler, the papers are physiological in nature.

The grouping in the Table of Contents is somewhat misleading. The largest group deals with central and peripheral nervous control of the various forms of locomotion, as well as other motor activities such as circulation and ventilation, in this notoriously heterogeneous assemblage of animals. Others cover the fluid mechanics of swimming and flying, gas exchange and the intriguing gaps in our understanding of how low muscular activity is fueled, thermoregulation and "energetics," a topic which is (predictably) interpreted variously by three different authors. A novel study on how using the claw muscles during larval development brings about the dimorphic condition of the two appendages in the adult lobster is reported by Govind.

The organizers of the symposium assembled a group of participants with quite different approaches to a common theme. Though the subject is clearly one of considerable research activity, its scope was intentionally restricted to ensure a coherent focus. Therefore the book will be of interest primarily to investigators in the field, or in closely allied fields. Nonetheless the generality of the papers is notably uneven. Among the more useful reviews of traditional topics are the two by Kammer and Rheuben and by Fourtner on neuromuscular control mechanisms in the insects, and also the paper by Kaars on the formidable diversity of the control of spiracle movements among the various insect orders.

Several contributors make a valiant effort to find common ground in, for example, the swimming of a crab and the flight of the bumblebee. This reader, however, was more impressed by the potential support from physiological investigation for the conclusion reached by most modern systematists, viz. that the Arthropoda is a highly polyphyletic taxon.

Charlotte Mangum
College of William and Mary

Going High: The Story of Man and Altitude. C.S. Houston

New York: American Alpine Club, 1980.
209 pp., illus., $4.95

This short soft-cover book tells the story of man's attempts to go to higher and higher altitudes and reviews some of the physiology of high-altitude acclimatization, including aspects of pathophysiology. The author is an outstanding climber of exceptional experience. Ever since he led an expedition that reached the summit of Nanda Devi (7,817 m) in 1936, he has maintained a strong interest in high-altitude physiology and medicine. The present volume covers the history of these topics in an entertaining and readable way and has sections on the physiology of acclimatization and the diseases of high altitude, such as high-altitude pulmonary edema and cerebral edema. Dr. Houston has always been very effective in translating scientific material into language that lay people can understand. This inevitably leads to some superficiality, and the book has some inaccuracies. There are many excellent illustrations, a detailed bibliography, notes on important papers and people in the field, but no index. The book makes an excellent companion for a long airplane flight and can be recommended to medical and graduate students as an entertaining account of this fascinating area of physiology with the proviso that they should try to spot the errors. Certainly the book should stimulate further reading on this colorful topic.

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Respiratory Physiology. A.H. Mines

New York: Haven, 1981. 165 pp., illus., index,
$9.95 (paper); $14.00 (cloth)

Respiratory Physiology by Mines, presently on the physiology faculty of the University of California at San Francisco, is an introductory textbook on the subject intended for students taking medical physiology. The book arose out of the successful instructional approaches the author found in 18 years of teaching. The book contains eight chapters of appropriate topics concluding with one on the regulation of breathing. The text is aptly complemented by a large number of tables and illustrations. In the text the author not only presents equations but also uses them in solving problems. At the end of each chapter are problems and multiple-choice questions as well as answers with detailed explanations to each. Some of the problems have clinical relevance; this should make the material more interesting to medical students. The question-and-answer feature also makes the book useful for individual study for those (such as myself) who have had no formal study of lung physiology but have a desire to understand the basic aspects of the subject. By his presentation, the author seems concerned that the student get the point being made. The references given are mainly to handbooks and other texts on respiratory physiology. This book appears to be a useful complement to those previously published texts.

James F. Collins
University of Texas Health Science Center, San Antonio

Molluscan Nerve Cells: From Biophysics to Behavior.
J.H. Byrne and J. Koester (Editors)
Cold Spring Harbor, NY, 1981. 230 pp., illus., index, $26.00

Neurobiology of the Leech.
K.J. Muller, J.G. Nicholls, and G.S. Stent (Editors)
Cold Spring Harbor, NY, 1981. 320 pp., illus., index, $36.00

Neurobiology is now a mature (if not very old) scientific discipline with its own societies, journals, and degree programs. Its breadth, spanning the range of phenomena from behavior to membrane biophysics, makes mastery of the whole field by any individual almost impossible to contemplate and has already resulted in the proliferation of many subdisciplines. Nevertheless, the multidisciplinary approach to problems that 50 years ago seemed nearly intractable has amply proved its worth.

One of the most significant divisions within the field is between the studies of vertebrate and invertebrate preparations. The recent successes of invertebrate neurobiology in providing at least partial answers to a growing list of significant questions have laid to rest questions of the "relevance" of studying crayfish, snails, or the leech.

The titles of the two books reviewed here suggest that they are both comprehensive and complementary descriptions of the current status of research using two different invertebrate preparations. There is, in fact, minimal similarity between the two volumes, and each offers something quite different to the reader.

Molluscan Nerve Cells is basically a review of recent work on the biophysics of excitable membranes, specifically the properties of ion channels in such membranes; as implied, all studies were carried out on a variety of molluscan nerve cells.

The book begins with two broad review articles. The first by Kandel is short and deals with the historical development of membrane biophysics and the relevance of this area to the broader field of neurobiology; Kandel's thoughts on this topic have now appeared in a number of publications. The second review article is an extremely useful one by Stevens on the techniques with which ion channels and their properties are studied.

With two exceptions the remaining papers deal with particular ion channels found in a variety of particular cells. Most represent brief summary descriptions of our knowledge of the properties of the particular channels at issue, with little or no description of the experiments (or the experimental techniques) from which the data derive. Other than the fact that cells chosen for study were found in invertebrates (because of their size and accessibility in such preparations) few if any conclusions are drawn about the significance of a particular channel being present in the neurons of a particular species.

Finally, there are wo papers that, in closer conformity with the implications of the title, do deal with attempts to explain some aspect of behavior on the basis of the properties of certain ion channels.

The Neurobiology of the Leech offers quite a different selection of topics. As its title implies it represents an attempt to summarize a variety of topics dealing with aspects of the nervous system of the leech. It begins with a discussion of the behavior of the leech in a biological context, continues with an interesting (if perhaps not completely relevant) review of the history of medicinal uses of the leech, and then proceeds to discuss topics ranging from the morphology of the leech and its nervous system, sensory and motor cells and their function, synaptic phenomena, motor activity, the neurochemistry of synaptic transmission, and ends with discussions of development and plasticity.

Here too, the articles are attempts at summarizing the status of work in a particular area, but with perhaps a little more attention given to experimental approaches (conceptually if not from a practical, "how-to-do-it" approach) employed in gathering the data. This volume truly represents an attempt to describe the comprehensive neurobiology of a single group of animals. It also represents the results of years of teaching the leech preparation to graduate and postdoctoral level students.

The differences between these two volumes, and one presumes the differences in the audiences being addressed, require that different criteria be applied in evaluating them.

Molluscan Nerve Cells exhibits the primary liability of symposia review volumes; even if the individual contributions are up to date when originally presented, the simple passage of time (and some considerable time is always lost in the process of preparing such a volume) rapidly outdates the contributions. Further, most articles present such sketchy discussions of a particular topic that significant issues are never raised, even in cases where considerable controversy exists. In experiments where subtle details of the recording electronics can raise serious questions about the interpretation of the results, the almost total lack of attention to the experiments themselves represents a serious problem.

Furthermore, almost none of the articles contain an adequate bibliography and thus cannot serve as adequate introductions to the newcomer to the field; there are relatively few references more recent than 1979, and papers average only 8–10 references each. Finally, most of the papers were prepared for publication by transcription from oral presentations, and this results in a certain uniformity of style that is arguably somewhat better than the worst of scientific papers, but certainly worse than the better efforts one encounters.

Even though this volume may be of some value to workers in the field as a summary statement of the properties of known ion channels in molluscan nerve cell membranes, its utility for a wider audience of neurobiologists seems minimal.

Such is not the case with the *Neurobiology of the Leech.* As its historical origins might suggest, this volume should find a secure place on the reference lists for graduate level (or even advanced undergraduate) courses in neurobiology. Its comprehensive coverage of the range of topics normally considered to constitute neurobiology offers the student a valuable introduction to the kinds of questions that can and should be asked about nervous systems and suggests the conceptual approaches that can be taken to obtain answers to these questions. Each article offers a reasonably complete review of its subject matter, with extensive references generally provided (averaging 40 per paper). While it is

not a laboratory manual (although the Appendix offers three chapters representing a useful approach to the study of the leech preparation), it could most appropriately serve as a companion volume for any such laboratory manual in current use. Though the subject of the volume is the leech, the issues it deals with are independent of the species used, whether they are invertebrates or vertebrates. Even when the details of the experimental results reported here are out of date (which will occur in a year or two given the rate of advance in invertebrate neurobiology), this volume will still be of value in the teaching of neurobiology.

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Cardiovascular Physiology (4th ed.).
R.M. Berne and M.N. Levy
St. Louis, MO: Mosby, 1981. 286 pp., illus., index, $16.95.

Cardiovascular Physiology.
L.J. Heller and D. E. Mohrman
New York: McGraw-Hill, 1981. 169 pp., illus., index, $9.95.

Modern Cardiovascular Physiology.
C.R. Honig
Boston, MA: Little, Brown, 1981. 347 pp., illus., index, $15.95.

Physiology of the Heart and Circulation (2nd ed.). R.C. Little
Chicago, IL: Year Book, 1981. 353 pp., illus., index, $15.95.

The Human Cardiovascular System.
J.T. Shepherd and P.M. Vanhoutte
New York: Raven, 1980. 351 pp., illus., index, $13.50.

Circulatory Physiology—The Essentials.
J.J. Smith and J.P. Kampine
Baltimore, MD: Williams & Wilkins, 1980. 344 pp., illus., index, $15.95.

While the use of comprehensive textbooks of human physiology has certainly not ceased (witness the continued sales of Arthur Guyton's books in their various versions), it is clear that the use of organ system monographs is rapidly growing. The six recently written or revised monographs on cardiovascular physiology to be considered here are examples of the wealth of material currently available. All are aimed at an audience of first-year medical students (and in some cases beginning graduate students). The instructor responsible for the selection of a monograph to be used in a particular course faces a challenge in reviewing the nearly 1800 pages to be found between these covers.

Two general questions must be confronted in evaluating these books: *1*) How well do they serve the needs of their intended audience, first-year medical students? and *2*) How well do they meet the needs of the instructor responsible for the cardiovascular portions of a physiology course?

Students are well served by clarity of explanation, by coverage of the material at a breadth and depth appropriate for their future needs, and, consonant with that, by a measure of conciseness. In addition, there is considerable virtue in providing clear evidence of the applicability of the material being developed to the future goals of the students. It goes without saying that accuracy and currency of the material presented are required.

The instructor is looking for a text that covers the subject matter in appropriate depth, with adequate consideration of as many of the different topic areas as possible; however, no single- or dual-authored text can be expected to deal equally well with every aspect of a topic as broad as cardiovascular physiology. Usually a text is sought either to complement the approach to be taken by the instructor in the lecture hall (strengths and weaknesses of each supplementing the other) or to present the material in a different way so as to provide the student with an equally valid but alternative approach to the material.

With these criteria in mind the strengths and weaknesses of each of the six cardiovascular monographs may be considered.

It is difficult to identify significant features that distinguish the first three monographs considered from one another in a useful way. All are clearly suitable for a first-year medical course in physiology. Each has its strong points and its weaknesses, and an instructor's choice of a text from this group is best based on personal preference arising out of his or her approach to teaching cardiovascular physiology.

Berne and Levy's *Cardiovascular Physiology* (4th edition) offers a comprehensive discussion at an appropriate depth and from a perspective well suited for medical students. Its organization, the sequence of topics, is conventional and well suited for most approaches to teaching cardiovascular physiology. The mathematical description of phenomena is developed rather systematically and with some rigor (although no calculus is used). Their discussion of cardiac cell electrical activity is extensive and is based on current understanding of the molecular mechanisms involved. This is followed by a section on the electrocardiogram, which develops the basic ideas in a particularly appropriate way without bogging down in unnecessary detail about clinical techniques or examples. The chapter on hemodynamics develops all of the needed concepts in an understandable way and occurs early enough to permit the student to apply these ideas to all of the relevant circulatory phenomena; surprisingly, this is not the case in all texts.

The major limitation of this book is its minimal discussion of aspects of integrative cardiovascular function (only exercise and hemorrhage are considered) and pathophysiology (only arrhythmias are considered in the chapter on the ECG). This is a serious lack, not because of the need to prove the "relevance" of this material to the student, but because the opportunity to enlarge on the significance of many important cardiovascular phenomena is thus lost. There is also no systematic discussion of the structures of the cardiovascular system, apparently assuming that the student will have or will be acquiring this information in anatomy. The writing is rather turgid in some places and may be difficult for the student to understand.

Physiology of the Heart and Circulation (2nd edition), by Little, is comparably detailed and rigorous in its development of biophysical concepts and the

mathematical relationships describing them. Its organization is conventional, although it might be preferable if there was at least some introduction to hemodynamics prior to p. 218. The volume contains a very useful chapter on cardiac structure that should provide all students with the requisite background to understand the function of the cardiovascular system; the inclusion of such material is unfortunately rare in monographs of this kind. It is also a pleasant surprise to find a chapter on cardiac energetics, including material on metabolism as well as on cardiac work. The chapter on the electrocardiogram is very complete, with the first part dealing with the basic phenomena and the second part devoted to more clinically relevant topics.

As with Berne and Levy, the major omission in Little's book is the minimal discussion of integrative phenomena (again, only hemorrhage and exercise are considered) and the absence of pathophysiology. In my experience getting students to grapple with the integrated homeostatic response to any of a number of "stresses" or pathophysiological states is an excellent way to ease the student's transition from being a passive memorizer of facts to a more active analyzer of function.

Smith and Kampine *(Circulatory Physiology—The Essentials)* have produced a companion volume to West's by now classic respiratory monograph. It is comprehensive (although not as biophysically oriented or mathematically rigorous as the two preceding volumes), generally well written, and contains considerable relevant pathophysiology; the final four chapters present examples of the integrated responses of the cardiovascular system to disease (ischemic heart disease, congestive heart failure, hypertension, and shock) and various "stresses" (exercise, aging, and hemorrhage).

I am of two minds about *Modern Cardiovascular Physiology* by Honig. It is as comprehensive in its coverage, level, and rigor of development as Berne and Levy or Little. There are a very large number of extremely well-done illustrations that manage to communicate a large amount of complex information. On the other hand, this profusion of illustrations results in many pages with very few words on them, forcing the reader to "flip" many pages to complete a small amount of text. Honig has explicitly organized this text around the themes of cardiac reserve and the systems concept, and this serves to draw together in a didactically useful way many seemingly disparate ideas. Finally, each section concludes with a well-thought-out problem set (with suggested approaches to the answers in the back); this should prove most useful to the student in testing his or her mastery of the material. Thus there is much that I find admirable about this book.

However, this book is rather idiosyncratically organized; the sequence of topics is quite unconventional and the material is arranged in a large number of short chapters grouped together in odd ways. For example, "Section II: Hemodynamics" contains chapters on expected topics such as "pressure gradients and resistance to blood flow" or "blood rheology" but also chapters on "blood volume and its distribution" as well as "cardiac output and regional blood flow." This may make it awkward to assign readings in this book if the organization of one's course differs significantly from Honig's.

The last two monographs are more readily distinguished from the others, since each of them, for different reasons, is obviously unsuited for use in a conventional first-year medical physiology course.

Cardiovascular Physiology by Heller and Mohrman has two significant features that will make it attractive to students. Each chapter begins with a list of objectives that define the facts, concepts, and problems that the student is to master; used appropriately this feature should enable the student to focus his or her attention in a productive manner. There are also a set of problems at the end of the book (with answers and some discussion) to accompany each chapter. The final chapter presents the student with a set of "disturbances to homeostasis" and considers the integrated responses of the cardiovascular system to these states.

The chief drawback to this monograph is its inadequate depth of coverage. While all of the topics usually considered in cardiovascular physiology are presented, many are discussed in a superficial simplistic manner. For example, the biophysics of cardiac cellular membranes receives minimal development with little or no reference to our growing knowledge of the ionic (membrane) mechanisms involved. Were this the only topic slighted in this way the text would be quite adequate, but unfortunately one can cite a number of similar examples (cardiac mechanics and capillary exchange are two other topics that readily come to mind).

The monograph by Shepherd and Vanhoutte (*The Human Cardiovascular System*) suffers from a quite different problem. Its content clearly suggests that rather than being aimed at first-year medical students, its audience is either second- or even third-year students studying the pathophysiology of the cardiovascular system or students in an integrated organ system course. Only the first half of the text deals with topics normally encountered in cardiovascular *physiology,* and of necessity then, the coverage of these topics is superficial and probably more suitable for review than for attempting the initial mastery of this material. The remainder of the book contains an extensive discussion of pathophysiology and more clinically relevant topics.

The instructor responsible for selecting the text for the usual first-year medical physiology course faces a difficult choice; any of the first three monographs discussed would seem to be suitable. My personal preference would be to recommend Smith and Kampine; it is comprehensive, is sufficiently quantitative without being "too mathematical," and seems to me to truly present the "essentials" in this obviously important area. For a course more oriented toward graduate students I would probably select Honig; for this audience the greater biophysical and mathematical "rigor" is not only appropriate but essential. The problem sets to be found here are particularly good at guiding the student into effective ways of analyzing cardiovascular function, again, a particularly desirable feature for a graduate student audience. Hopefully, the idiosyncracies that will probably make this book difficult for medical students will pose less of a problem to graduate students. Both Heller and Mohrman and Shepherd and Vanhoutte should be considered for audiences other than first-year medical or graduate students.

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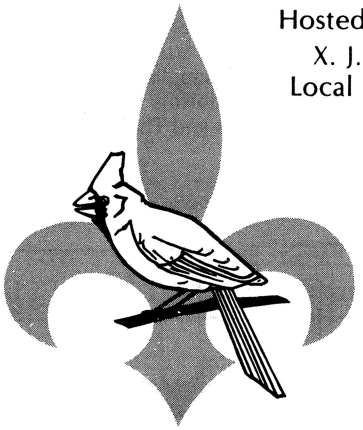

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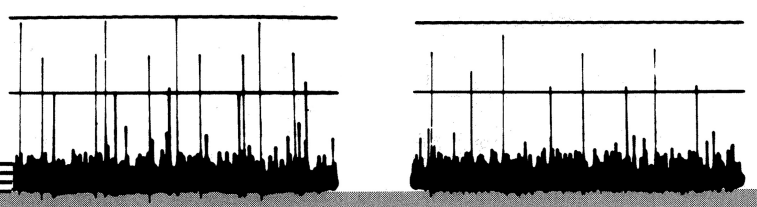

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