TRANSACTIONS OF THE PHILADELPHIA ACADEMY OF SURGERY

Transactions of the

Philadelphia Academy of Surgery

VOLUME XXXIV

1981–1986

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NOTICE

The thirty-fourth volume of the TRANSACTIONS OF THE PHILADELPHIA ACADEMY OF SURGERY covers the six years from 1981–1986 inclusive.

Wallace P. Ritchie, Jr., M.D., Ph.D.
Recorder

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Contents

Constitution

ARTICLE I

The name of the Society shall be "THE PHILADELPHIA ACADEMY OF SURGERY."

ARTICLE II

The objects of the Academy shall be the Cultivation and Improvement of the Science and Art of Surgery, the Elevation of the Medical Profession, the Promotion of the Public Health, and such other matters as may come legitimately within its sphere.

ARTICLE III

Section 1. The Society shall consist of Active, Senior, Nonresident, Government Service, and Honorary and Inactive Fellows.

Section 2. The Active Membership shall be limited to one hundred and fifty (150) Fellows.

Section 3. Active Fellows shall automatically become Senior Fellows of the Academy after they have been members for twenty (20) years or have reached the age of sixty (60). Senior Members shall have all the privileges of Active Fellows.

Section 4. Upon request, any Fellow in good standing, who may remove from the City of Philadelphia, to reside at a distance exceeding thirty (30) miles from the City Hall, may be made a Nonresident Fellow of the Academy, by recommendation of the Council and a two-thirds vote of the Fellows present at any regular meeting of the Academy. Nonresident Fellows shall have all the privileges of Active Fellows.

Section 5. Officers of the Government Services stationed in Philadelphia may be elected as Government Fellows of the Philadelphia Academy of Surgery for the period of their stay in Philadelphia. Such Fellows shall have all the rights and privileges of Active Fellows but shall be ineligible to vote or hold office.

Section 6. Honorary Fellows, to the number of thirty (30), may from time to time be elected. They shall not be eligible for election as Officers.

Section 7. Inactive Fellows. This consists of Active Fellows or Senior Fellows no longer in active practice of Surgery but who wish to participate in the activities of the Philadelphia Academy of Surgery. These Fellows will be subject to reduced dues and will not be subject to assessments.

ARTICLE IV

The Officers of the Academy shall consist of the President, the First Vice-President, the Second Vice-President, the Secretary, the Treasurer, the Recorder, and the Chairman of the Committee on Scientific Business.

ARTICLE V

These Officers shall be elected by a ballot each year, and with the exception of the President shall be eligible for re-election. A Fellow may serve as President for only (two (2) terms) one (1) one-year term.

ARTICLE VI

There shall be a standing Committee on Scientific Business.

The Committee on Scientific Business shall consist of a Chairman, who is an elected Officer of the Society, the Recorder, and one (1) Fellow appointed by the President. The duties of this Committee shall be to organize the Scientific Programs of the Society.

ARTICLE VII

A Council shall be established consisting of the President, the Vice-Presidents, the Secretary, the Treasurer, the Chairman of the Business Committee, and three (3) Fellows-at-large elected by the Society annually, one (1) of whom will whenever possible be a previous President. The President of the Academy shall act as Chairman of the Council. The duties of the Council shall be three:

1. To act as an Executive Committee for the Academy between meetings,
2. To receive all nominations for Fellowship and to report names for election to the Academy after due investigation.
3. To act as a Board of Censors as required by the Academy.

ARTICLE VIII

At the stated meeting in February every fifth year, three (3) Fellows shall be appointed by the President to serve for five (5) years, or until their successors are appointed, as Trustees of the S. D. Gross Prize Fund and Library. It shall be the duty of the Trustees to keep charge of the Fund, to attend to its safe investment, and to submit a report to each annual meeting of the Academy of their work during the year, which shall be entered upon the minutes of the Academy. The Trustees shall have, on behalf of the Academy, charge of the S. D. Gross Library, which is, in accordance with the will of the Testator, in the custody of the College of Physicians of Philadelphia. They shall each year make such additions to the collection of Surgical Books in the Library as may be deemed advisable, and as the funds contributed to the care and support of

the Library may permit. They shall have charge of the distribution of the S. D. Gross Prize. It shall be their duty to publish in the medical journals the conditions on which the Prize is offered, to receive all essays submitted for competition, and upon approval of their decision by the Academy, to make award of the Prize to the successful competitor, who shall present the winning essay at a regular scientific meeting of the Academy. Discussion of this paper is permitted.

ARTICLE IX

To become a Fellow of the Academy, a man must be a Doctor of Medicine who has graduated from a reputable School of Medicine at least ten (10) years before he is proposed. He must be proposed by at least three (3) Fellows of the Academy, who shall write letters to the Secretary in support of the proposal. No officer or other Council member may be a proposer or Seconder of a candidate for Fellowship. The candidate for Fellowship must receive the approval of the Council at three (3) Council meetings before his name may be presented to the Academy as a candidate for election. He must meet such other requirements as are, from time to time, stipulated in the By-Laws, and must be elected by the Fellows in accordance with the By-Laws.

ARTICLE X

Any Fellow having complied with the requirements of the Constitution and By-Laws may resign his Fellowship by presenting at a stated meeting a communication to that effect, with the Treasurer's certificate that he is not indebted to the Academy, and such resignation shall become valid on acceptance by the Academy.

Any violations of the regulations of the Academy, and of the Code of Medical Ethics adopted by it, shall be punished by reprimand, suspension, or expulsion after a full hearing by the Council of the Academy or upon the request of the Fellow in question by the Academy itself.

ARTICLE XI

This Constitution may be amended by a two-thirds vote of the Fellows, after such amendment has been presented in writing to the Secretary and read at the two previous meetings of the Academy, and circulated with the call to the meeting at which action is to be taken.

By-Laws

SECTION I

MEETINGS

The stated meetings of the Academy shall be held at eight-fifteen o'clock P.M., on the first Monday of each month, except June, July, August and September. The date of any stated meeting may be changed at the discretion of

the Council by giving notice to the Fellows at least two (2) weeks before the meeting.

The annual meeting shall be the first meeting of the new year (January), at which time election of officers will occur and reports are to be given.

SECTION II

SPECIAL MEETINGS

A special meeting may be called at any time by the President, and it shall be his duty to do so upon the requisition, in writing, of any ten (10) Fellows.

SECTION III

QUORUM

For the transaction of ordinary business any number of Fellows shall, at any meeting, constitute a quorum. For all elections, changes in the Constitution and By-Laws, for ordering assessments, or for the appropriation or expenditure of any sum of money exceeding one hundred dollars ($100.00), or for any other business affecting the interests of the Academy, or of its individual Fellows, fifteen (15) shall be required to be present.

SECTION IV

DUTIES OF OFFICERS—PRESIDENT AND VICE-PRESIDENTS

The President shall preside at the meetings, regulate debates, sign Certificates of Fellowship, appoint committees not otherwise provided for, announce the results of elections, and perform all other duties pertaining to his office. The Vice-Presidents shall assist the President in the discharge of his functions, and in his absence preside in the order of seniority.

SECTION V

SECRETARY

The Secretary shall keep the minutes of the meetings of the Academy, one copy of which he shall send to the Recorder. He shall notify the Fellows of the meetings, announcing on the notices the business to be transacted, with the names of candidates for Fellowship to be balloted upon by the Academy, attest all official acts requiring certificates in connection with, or independently of, the President, notify the Officers and Fellows of their election, acquaint newly elected Fellows with the requirements of the By-Laws concerning admission, receive the signatures of newly elected Fellows, take charge of papers not otherwise provided for, shall keep in his custody the seal of the Academy, and affix it to any documents or papers that the Academy may direct.

SECTION VI

TREASURER

It shall be the duty of the Treasurer to receive all moneys and funds belonging to the Academy, unless otherwise provided for; he shall pay bills for all expenses properly incurred by the Academy; collect all dues and assessments as promptly as possible, and present an annual account for audit. Two auditors shall be appointed by the President at the December Meeting to audit these accounts with a report at the January meeting.

At the December meeting, the Treasurer shall propose suitable honoraria for the secretaries of the following officers: the Secretary, the Treasurer, the Recorder, the Chairman of the Committee on Scientific Business, and upon affirmative vote of the Fellows shall send such honoraria before Christmas.

SECTION VII

RECORDER

The Recorder shall be a member of Council and serve as a Member of the Committee on Scientific Business. He shall receive copies of the Annual Oration. He shall maintain the Archives of the Academy, including copies of the Minutes, and he shall consult with Fellows who present Annual Orations and Memoirs before the Academy in regard to publication. He shall maintain the material required for publication of the *Transactions of the Philadelphia Academy of Surgery*, and shall act as Editor for the *Transactions*, arranging for their publication at intervals of approximately five (5) years as required by the Academy.

SECTION VIII

COUNCIL

The Council of the Academy shall hold meetings for the transaction of routine business upon notice from the Secretary and special meetings shall be held on call of the President or on the call of any two (2) of its own number. A quorum shall consist of not less than four (4) of its members, and notice of any unusual business or any routine business having unusual significance for the Academy shall be sent to members at least five (5) days prior to a meeting.

SECTION IX

THE COMMITTEE ON SCIENTIFIC BUSINESS

The Committee on Scientific Business shall consist of three (3) Fellows, a Chairman elected by the Academy, the Recorder, and one (1) additional Fellow appointed by the President. It shall have charge of the scientific business of the meetings, it shall be its duty to provide for the presentation of papers and discussions of subjects for each meeting, it shall arrange, at such times as may

deem proper, for the discussion of scientific subjects by the Fellows of the Academy, and it shall, when authorized by the Academy, invite members of the profession, resident or nonresident, to read papers before the Academy, or to present topics for discussion. It shall act as a committee on publication, and shall present at the annual meeting a report of the work done during the year, which shall be entered upon the minutes of the Academy.

SECTION X

ANNUAL ORATION

There shall be appointed by the President at the stated meeting in February of each year, a Fellow whose duty it shall be to deliver at a stated meeting, usually December, of that year, an address in Surgery. This address shall be delivered to the Recorder in writing at the time of its presentation, and it shall be published in the *Transactions* of the Academy. After consultation with the Recorder, it may be published in any other reputable scientific journal so long as it is identified as the Annual Oration of the Philadelphia Academy of Surgery, and so long as permission is obtained for its subsequent publication in the *Transactions* of the Academy.

The Jonathan E. Rhoads Annual Oration. There shall be appointed by the President at the stated meeting in February each year, a Fellow whose duty it shall be to deliver at a stated meeting, usually December of that year, *The Jonathan E. Rhoads Annual Oration*. No discussion of this paper is permitted at the time of presentation. This address shall be delivered to the Recorder in writing at the time of its presentation, and it shall be published in the *Transactions* of the Academy. After consultation with the Recorder, it may be published in any other reputable scientific journal so long as it is identifiable as the Annual Oration of the Philadelphia Academy of Surgery, and so long as permission is obtained for its subsequent publication in the *Transactions* of the Academy.

SECTION XI

ELECTION OF OFFICERS

At the November meeting of the Academy, the President shall nominate three (3) Fellows to act as a Nominating Committee. Insofar as possible, these shall be previous Presidents of the Academy. This Committee shall report at the December meeting each year. Additional Fellows may be nominated for any office from the floor. The Officers of the Academy shall be elected at the January meeting. The election shall be by ballot whenever more than one (1) candidate has been nominated for any office, and a majority of all those present shall be necessary to a choice. Where there is no contest, election may be by acclamation.

SECTION XII

PROPOSALS FOR FELLOWSHIP

Proposals for Fellowship shall be in writing signed by three (3) Fellows, none of whom are officers or Council members, with a letter from each vouching for the character of the candidate. The members of Council will not act as proposers or seconders of candidates for Fellowship. Completed nominations shall be considered by the Council at its next meeting. In the event action is deferred for more than three (3) consecutive meetings of Council, the President shall communicate with one or more of the candidate's sponsors.

No candidate may be proposed for Fellowship who has not made at least one (1) presentation before the Academy. The names of candidates who are to be recommended by the Council shall be (published with the notices of the meeting immediately) read at the business meeting of three consecutive meetings preceding consideration by the Fellows. Certification by the candidate's specialty board and Fellowship in the American College of Surgeons are requirements. it is expected that a candidate proposed for Fellowship will have attained some reputation in surgical practice, research, and/or teaching, and that he has demonstrated potential for making contributions to the various programs of the Academy with a minimum of three scientific publications.

SECTION XIII

ELECTION OF FELLOWS

The names of candidates proposed for Fellowship, who are approved by Council, shall be read with supporting letters from each of the three (3) proposers at a stated meeting of the Academy. Their names shall be read at a second meeting, and sent out with a call to the following meeting at which the election shall be held. Election of candidates for Fellowship who have been reported upon by the Council may take place at any stated meeting and shall be by ballot. A two-thirds vote of those present shall be necessary to elect the candidate to Fellowship.

A candidate for Fellowship failing to obtain the requisite number of votes in his favor may not again be nominated before the expiration of two (2) years.

SECTION XIV

SIGNING THE CONSTITUTION

Every person elected to be a Fellow shall pay the initiation fee and shall sign the Constitution and By-Laws. No person shall acquire the rights of Fellowship unless he makes payment of the initiation fee and signs the Constitution and By-Laws by the third meeting following his election.

SECTION XV

INITIATION FEE

Every Fellow shall, on admission, pay an initiation fee of twenty-five dollars ($25.00).

SECTION XVI

ANNUAL DUES

An annual assessment *shall be determined by Council for Active and Senior members payable within three (3) months after January 1st.* Fellows elected in November or December shall not be subject to the annual assessment for that year. Those who go on active duty with the government may have their dues remitted temporarily by action of Council. Under appropriate circumstances and with a two-thirds vote of approval by Council, additional and unscheduled assessments may be made. The total of these assessments, excluding the annual assessment, may not exceed one hundred dollars ($100.00) in any twelve-month period. Assessments shall only apply to Active and Senior members. Voluntary contributions may be solicited for specific programs on occasion.

Non-Resident Fellows: The annual assessment shall be ten dollars ($10.00).

Government Fellows: No annual assessment.

Inactive Fellows: Dues will be reduced and they are not subject to assessments.

Any fellow who requests relief from payment of dues and assessments may, at the discretion of the Council, be relieved of such dues and assessments, without loss of his fellowship or other rights.

SECTION XVII

Any Fellow in arrears for one (1) year, being notified of the fact by the Treasurer, in writing, and not paying his dues within two (2) months thereafter, shall forfeit his Fellowship; and it shall be the duty of the Treasurer to notify the Academy of such forfeiture, which shall be entered on the minutes, and the name stricken from the list of Fellows. The notice aforesaid shall contain a copy of this section.

Any active Fellow not attending at least two (2) of the Stated Meetings in any one (1) year (October through May) shall state in writing to the Secretary the reasons for this failure. The names of such Active Fellows shall then be read to the members of Council by the Secretary. The members of Council may then take whatever action they deem necessary as follows: excuse, reprimand, or expel the offending Fellow.

A majority of the Council shall have the power to expel Fellows for willful infractions of the By-Laws of the Academy, or for acts or conduct that they may deem disorderly, injurious, or hostile to the interests or objects of the Academy. (1.7.74)

SECTION XVIII

GUESTS

The Scientific Programs of the Society shall be open to any members of the medical profession and individuals in ancillary fields, including medical students and graduate students in the medical sciences, unless attendance is specifically restricted by vote of the Academy. Any Fellow may invite any medical man in good standing to a meeting of the Academy as an official guest. Such an official guest shall be introduced to the President, and to the Academy by the President, and his name entered upon the minutes. The President may invite any such person to participate in the discussion.

Business meetings shall be limited to Fellows of the Academy, except when a non-Fellow shall be invited to attend some portion of a business meeting for a particular purpose at the request of the President, who shall make known the presence of such an individual at the beginning of the meeting.

SECTION XIX

SEAL AND CERTIFICATE OF FELLOWSHIP

The Academy shall have a distinct seal, as well as a Certificate of Fellowship, to a copy of which, signed by the President and Secretary, every Fellow shall be entitled.

SECTION XX

ORDER OF BUSINESS

The order of business shall be as follows unless modified by the President:

I. Scientific Proceedings:
   1. Call to order.
   2. Introduction of guests.
   3. Introduction of new Fellows.
   4. Reading of scientific papers, including the discussion of each.

II. Business Session:
   1. Reading of minutes of the last meeting.
   2. Reports of committees.
   3. Unfinished business.
   4. New business.
   5. Election of officers.
   6. Election of Fellows.
   7. Adjournment.

SECTION XXI

RULES OF ORDER

The proceedings of the Academy shall be conducted according to *Robert's Rules of Order*.

SECTION XXII

ALTERATIONS OF THE BY-LAWS

Amendments to the By-Laws may be made at any stated meeting at which a quorum is present, providing that notice of the proposed amendment shall have been sent to the members with the call to the meeting at least five (5) days in advance. A majority vote shall suffice for amendment to the By-Laws.

Founders

Founded April 21, 1879

Incorporated December 27, 1879

*SAMUEL D. GROSS, M.D., LL.D., D.C.L., Oxon

*D. HAYES AGNEW, M.D., LL.D.

*ADDINELL HEWSON, M.D.

*RICHARD J. LEVIS, M.D.

*THOMAS G. MORTON, M.D.

*JOHN H. PACKARD, M.D.

*JOHN H. BRINTON, M.D.

*WILLIAM H. PANCOAST, M.D.

*J. EWING MEARS, M.D.

*SAMUEL W. GROSS, M.D., LL.D.

*Deceased

List of Officers, 1986

President

FRANCIS ROSATO, M.D.

First Vice-President

WILLIS P. MAIER, M.D.

Second Vice-President

DOMINIC A. DELAURENTIS, M.D.

Secretary

DAVID K. WAGNER, M.D.

Treasurer

RUDOLPH C. CAMISHION, M.D.

Recorder

WALLACE P. RITCHIE, JR., M.D., Ph.D.

Chairman, Program Committee

CLYDE F. BARKER, M.D.

Members-at-Large

FREDERICK B. WAGNER, JR., M.D.

HUNTER NEAL, M.D.

MOREYE NUSBAUM, M.D.

Samuel D. Gross Prize Fund

ROBERT D. HARWICK, M.D., Chairman

Philadelphia Academy of Surgery

Founded April 21, 1879
Incorporated December 27, 1879

Officers

1879

Temporary Chairman ..... ADDINELL HEWSON

Temporary Secretary ..... J. EWING MEARS

Temporary Treasurer ..... WILLIAM HUNT

Temporary Recorder ..... JOHN B. ROBERTS

PRESIDENT

ELECTED	
1880	SAMUEL D. GROSS
1884	D. HAYES AGNEW
1891	WILLIAM HUNT
1895	THOMAS G. MORTON
1898	DEFOREST WILLARD
1902	RICHARD H. HARTE
1904	HENRY R. WHARTON
1906	JOHN B. ROBERTS
1908	WILLIAM J. TAYLOR
1910	ROBERT G. LECONTE
1912	GWILYM G. DAVIS
1914	JOHN H. GIBBON
1916	CHARLES H. FRAZIER
1918	EDWARD MARTIN
1920	GEORGE G. ROSS
1922	JOHN H. JOPSON
1924	EDWARD B. HODGE
1926	CHARLES F. MITCHELL
1928	ASTLEY P. C. ASHHURST
1930	GEORGE P. MULLER
1932	JOHN SPEESE
1934	WALTER ESTELL LEE
1936	DAMON B. PFEIFFER
1938	J. STEWART RODMAN
1940	ELDRIDGE L. ELIASON
1942	ROBERT H. IVY
1944	HUBLEY R. OWEN
1946	JOHN B. FLICK
1948	THOMAS A. SHALLOW
1950	CALVIN M. SMYTH
1952	I. S. RAVDIN
1954	L. K. FERGUSON
1956	JOHN GIBBON, JR.
1958	ADOLPH WALKLING
1960	W. EMORY BURNETT
1962	J. MONTGOMERY DEAVER
1964	JONATHAN E. RHOADS
1965	GEORGE J. WILLAUER
1967	GEORGE P. ROSEMOND
1970	JULIAN JOHNSON
1972	WILLIAM H. ERB
1974	JOHN Y. TEMPLETON, III
1976	H. TAYLOR CASWELL
1978	DONALD R. COOPER
1980	BROOKE ROBERTS
1981	BROOKE ROBERTS
1982	PAUL NEMIR, JR.
1983	R. ROBERT TYSON
1984	CHARLES C. WOLFERTH, JR.
1985	FREDERICK B. WAGNER, JR.
1986	FRANCIS E. ROSATO

VICE-PRESIDENT

ELECTED	
1880	D. HAYES AGNEW
1880	R. J. LEVIS
1884	SAMUEL W. GROSS
1889	JOHN H. PACKARD
1891	WILLIAM W. KEEN
1891	J. EWING MEARS
1898	JOHN ASHHURST, JR.
1900	RICHARD H. HARTE
1900	HENRY R. WHARTON
1902	JOHN B. DEAVER
1904	JOHN B. ROBERTS
1905	WILLIAM J. TAYLOR
1906	ROBERT G. LECONTE
1908	G. G. DAVIS
1910	JOHN H. GIBBON
1912	CHARLES H. FRAZIER
1914	EDWARD MARTIN
1916	GEORGE G. ROSS
1918	JOHN H. JOPSON
1919	H. C. DEAVER
1920	JOHN H. JOPSON
1920	EDWARD B. HODGE
1922	CHARLES F. MITCHELL
1924	ASTLEY P. C. ASHHURST
1926	ASTLEY P. C. ASHHURST
1926	GEORGE P. MULLER
1928	JOHN SPEESE
1930	WALTER ESTELL LEE
1932	DAMON B. PFEIFFER
1934	J. STEWART RODMAN
1936	E. J. KLOPP
1938	ELDRIDGE L. ELIASON
1938	ROBERT H. IVY
1940	HUBLEY R. OWEN
1942	JOHN B. FLICK
1943	THOMAS A. SHALLOW
1945	CALVIN M. SMYTH
1948	L. KRAEER FERGUSON
1950	I. S. RAVDIN
1952	L. K. FERGUSON
1954	JOHN H. GIBBON, JR.
1956	ADOLPH WALKLING
1958	W. EMORY BURNETT
1960	J. MONTGOMERY DEAVER
1962	JONATHAN E. RHOADS
1964	GEORGE J. WILLAUER
1965	GEORGE P. ROSEMOND
1967	JULIAN JOHNSON
1976	DONALD R. COOPER
1978	BROOKE ROBERTS
1980	WILLIAM T. FITTS, JR.
1981	PAUL NEMIR, JR.
1982	R. ROBERT TYSON
1983	CHARLES C. WOLFERTH, JR.
1984	FREDERICK B. WAGNER, JR.
1985	FRANCIS ROSATO
1986	WILLIS P. MAIER

SECRETARY

ELECTED	
1880	J. EWING MEARS
1885	J. HENRY C. SIMES
1893	THOMAS R. NEILSON
1896	WILLIAM J. TAYLOR
1905	JOHN H. GIBBON
1909	CHARLES F. MITCHELL
1915	GEORGE P. MULLER
1920	J. STEWART RODMAN
1922	HUBLEY R. OWEN
1930	DEFOREST P. WILLARD
1935	HENRY P. BROWN, JR.
1940	JOHN B. FLICK
1942	L. KRAEER FERGUSON
1943	CALVIN M. SMYTH
1945	L. KRAEER FERGUSON
1948	J. MONTGOMERY DEAVER
1958	WILLIAM B. FITTS
1960	HENRY P. ROYSTER
1964	THOMAS F. NEALON
1967	DONALD R. COOPER
1974	PAUL NEMIR, JR., M.D.
1980	FREDERICK B. WAGNER, SR.

ELECTED

1981 FREDERICK B. WAGNER, JR.
1982 FREDERICK B. WAGNER, JR.
1983 JAMES G. BASSETT
1984 JAMES G. BASSETT
1985 DAVID K. WAGNER
1986 DAVID K. WAGNER

TREASURER

ELECTED

1880 WILLIAM HUNT
1891 WILLIAM G. PORTER
1904 JAMES P. HUTCHINSON
1911 EDWARD B. HODGE
1920 DUNCAN L. DESPARD
1922 WILLIAM B. SWARTLEY
1935 L. KRAEER FERGUSON
1938 HARRY E. KNOX
1947 S. DANA WEEDER
1960 ORVILLE C. KING

ELECTED

1965 EDWIN W. SHEARBURN
1974 WILLIAM T. FITTS, JR.
1980 CHARLES C. WOLFERTH, JR.
1981 CHARLES WOLFERTH, JR.
1982 WILLIS P. MAIER
1983 WILLIS P. MAIER
1984 WILLIS P. MAIER
1985 RUDOLPH C. CAMISHION
1986 RUDOLPH C. CAMISHION

RECORDER

ELECTED

1880 JOHN B. ROBERTS
1881 DEFOREST WILLARD
1884 C. B. G. DENANCREDE
1884 J. EWING MEARS
1891 LEWIS W. STEINBACH
1902 JOHN H. GIBBON
1905 JOHN H. JOPSON
1915 JOHN SPEESE
1920 HENRY P. BROWN, JR.
1922 J. WILLIAM BRANSFIELD
1926 CALVIN M. SMYTH, JR.
1937 ADOLPH A. WALKLING
1950 JONATHAN E. RHOADS

ELECTED

1952 W. EMORY BURNETT
1956 FREDERICK A. BOTHE
1960 H. TAYLOR CASWELL
1966 WILLIAM S. BLAKEMORE
1974 EDWIN W. SHEARBURN
1976 JOSEPH W. STAYMAN
1980 ELMER L. GRIMES
1981 ELMER L. GRIMES
1982 ELMER L. GRIMES
1983 ELMER L. GRIMES
1984 DOMINICK A. DELAURENTIS
1985 DOMINICK A. DELAURENTIS
1986 WALLACE P. RITCHIE, JR.

COUNCIL

ELECTED

1880 JOHN ASHHURST, JR.
1880 JOHN H. BRINTON
1894 WILLIAM B. HOPKINS
1895 HENRY R. WHARTON
1898 THOMAS R. NEILSON

ELECTED

1900 W. JOSEPH HEARN
1902 ROBERT G. LECONTE
1906 THOMAS R. NEILSON
1910 J. CHALMERS DE COSTA
1920 CHARLES F. MITCHELL

ELECTED

1922 GEORGE G. ROSS
1922 JAMES H. BALDWIN
1923 WILLIAM J. TAYLOR
1924 JOHN H. JOPSON
1924 JOHN SPEESE
1925 EDWARD B. HODGE
1926 DAMON B. PFEIFFER
1927 CHARLES F. MITCHELL
1930 ASTLEY C. ASHHURST
1930 HUBLEY R. OWEN
1932 GEORGE P. MULLER
1935 DEFOREST P. WILLARD
1936 WALTER ESTELL LEE
1936 ROBERT H. IVY
1940 J. STEWART RODMAN
1940 DAMON B. PFEIFFER
1941 EDWARD B. HODGE
1942 THOMAS A. SHALLOW
1942 ELDRIDGE L. ELIASON
1943 ROBERT H. IVY
1946 HUBLEY R. OWEN
1947 CHARLES F. MITCHELL
1948 FRANCIS C. GRANT
1950 THOMAS A. SHALLOW
1952 ADOLPH WALKLING
1952 CALVIN M. SMYTH
1954 I. S. RAVDIN
1954 FREDERICK A. BOTHE
1956 FREDERICK ROBBINS
1956 L. KRAEER FERGUSON
1957 FREDERICK ROBBINS
1958 JOHN H. GIBBON, JR.
1959 ORVILLE C. KING
1960 ADOLPH WALKLING
1960 JONATHAN E. RHOADS

ELECTED

1962 DONALD K. COOPER
1962 W. EMORY BURNETT
1964 J. MONTGOMERY DEAVER
1965 JONATHAN E. RHOADS
1967 JOHN Y. TEMPLETON
1967 GEORGE WILLAUER
1974 WILLIAM H. ERB
1974 CHARLES C. WOLFERTH, JR.
1974 JOSEPH W. STAYMAN, JR.
1976 JOHN Y. TEMPLETON, III
1976 CHARLES C. WOLFERTH, JR.
1976 R. ROBERT TYSON
1978 H. TAYLOR CASWELL
1978 ELMER L. GRIMES
1978 FREDERICK B. WAGNER, JR.
1980 DONALD R. COOPER
1980 WILLIS B. MAIER
1980 FRANCIS E. ROSATO
1981 DONALD COOPER
1981 WILLIS P. MAIER
1981 JAMES BASSETT
1982 BROOKE ROBERTS
1982 JAMES G. BASSETT
1982 HARRY V. ARMITAGE
1983 PAUL NEMIR, JR.
1983 HARRY V. ARMITAGE
1983 WILLIAM STAINBACK
1984 R. ROBERT TYSON
1984 WILLIAM STAINBACK
1984 CLIFTON F. WEST, JR.
1985 CHARLES C. WOLFERTH, JR.
1985 CLIFTON F. WEST, JR.
1985 MOREYE NUSBAUM
1986 FREDERICK B. WAGNER, JR.
1986 MOREYE NUSBAUM
1986 HUNTER NEAL

With President, Vice-President, Secretary and Treasurer

BUSINESS COMMITTEE

ELECTED

1895 WILLIAM J. TAYLOR
1895 DEFOREST WILLARD
1896 RICHARD H. HARTE
1897 ROBERT G. LECONTE

ELECTED

1900 G. G. DAVIS
1902 JOHN H. JOPSON
1905 GEORGE G. ROSS
1908 FRANCIS T. STEWART

ELECTED

1914 JOHN SPEESE
1916 WALTER ESTELL LEE
1916 MORRIS BOOTH MILLER
1917 DAMON B. PFEIFFER
1917 ASTLEY P. C. ASHHURST
1919 A. BRUCE GILL
1919 J. STEWART RODMAN
1920 ARTHUR BILLINGS
1922 DAMON B. PFEIFFER
1924 DEFOREST P. WILLARD
1928 WALTER ESTELL LEE
1930 EDWARD T. CROSSAN
1930 JOHN B. FLICK
1931 HENRY P. BROWN, JR.
1932 EDWARD T. CROSSAN
1935 B. FRANKLIN BUZBY
1936 JOHN B. FLICK

ELECTED

1938 L. KRAEER FERGUSON
1940 J. MONTGOMERY DEAVER
1942 CALVIN M. SMYTH
1943 FREDERICK A. BOTHE
1943 W. EMORY BURNETT
1944 ADOLPH A. WALKLING
1946 J. MONTGOMERY DEAVER
1949 FREDERICK A. BOTHE
1950 JOHN H. GIBBON, JR.
1950 JONATHAN E. RHOADS
1951 FRANK ALLBRITTEN, JR.
1954 EDWIN W. SHEARBURN
1960 JOHN Y. TEMPLETON, III
1964 BROOKE ROBERTS
1974 BROOKE ROBERTS
1978 R. ROBERT TYSON

With the Recorder

Officers

TRUSTEES OF THE SAMUEL D. GROSS PRIZE FUND AND LIBRARY

1894

J. EWING MEARS JOHN ASHHURST, JR. WILLIAM W. KEEN

With Samuel Ashhurst and William Hunt to serve with them on distribution of prize.

1895-1899
J. EWING MEARS
JOHN ASHHURST, JR.
WILLIAM W. KEEN

1900-1901
WILLIAM W. KEEN
J. EWING MEARS
J. CHALMERS DACOSTA

1902-1904
WILLIAM J. TAYLOR
WILLIAM L. RODMAN
JOHN B. ROBERTS

1905
WILLIAM J. TAYLOR
RICHARD H. HARTE
DEFOREST WILLARD

1910
WILLIAM J. TAYLOR
RICHARD H. HARTE
JOHN H. GIBBON

1915
WILLIAM J. TAYLOR
JOHN H. JOPSON
EDWARD B. HODGE

1920
WILLIAM J. TAYLOR
JOHN H. JOPSON
EDWARD B. HODGE

1925
WILLIAM J. TAYLOR
JOHN H. JOPSON
EDWARD B. HODGE

1930
WILLIAM J. TAYLOR
JOHN H. JOPSON
EDWARD B. HODGE

1935
EDWARD B. HODGE
CHARLES F. MITCHELL
CALVIN M. SMYTH, JR.

1940
EDWARD B. HODGE
CHARLES F. MITCHELL
CALVIN M. SMYTH, JR.

1945
DAMON B. PFEIFFER
CHARLES F. MITCHELL
CALVIN M. SMYTH, JR.

1950
JOHN H. GIBBON, JR.
FRANCIS C. GRANT
CALVIN M. SMYTH, JR.

1955
CALVIN M. SMYTH
JOHN M. GIBBON, JR.
GEORGE P. ROSEMOND

1957
CALVIN M. SMYTH
JOHN H. GIBBON, JR.
GEORGE P. ROSEMOND

1961
GEORGE P. ROSEMOND
S. DANA WEEDER
GEORGE WILLAUER

1964
PAUL NEMIR, JR. (Chairman)
S. DANA WEEDER
GEORGE WILLAUER

1974
PAUL NEMIR, JR.

1980
MOREYE NUSBAUM

1983
ROBERT D. HARWICK

Honorary Fellows

ELECTED		DIED
1881	SIR JAMES PAGET, London, England	December 30, 1899
1881	THEODORE BILLROTH, Vienna, Austria	January 5, 1894
1881	BERNHARD VON LANGENBECK, Berlin, Germany	September 30, 1887
1881	WILLARD PARKER, New York, N.Y.	April 25, 1884
1881	LEWIS A. SAYRE, New York, N.Y.	September 21, 1900
1881	MOSES GUNN, Chicago, Ill.	November 4, 1887
1881	JOHN T. HODGEN, St. Louis, Mo.	April 28, 1882
1881	W. W. DAWSON, Cincinnati, Ohio	February 16, 1893
1881	T. G. RICHARDSON, New Orleans, La.	May 26, 1892
1881	J. COLLINS WARREN, Boston, Mass.	1927
1881	W. T. BRIGGS, Nashville, Tenn.	June 13, 1894
1881	CHRISTOPHER JOHNSTON, Baltimore, Md.	October 11, 1891
1881	D. W. YANDELL, Louisville, Ky.	May 2, 1898
1898	MAURICE H. RICHARDSON, Boston, Mass.	July 31, 1912
1898	GEORGE M. STERNBERG, Washington, D.C.	November 3, 1915
1898	CHARLES W. McBURNEY, New York, N.Y.	November 7, 1913
1898	NICHOLAS SENN, Chicago, Ill.	January 2, 1908
1898	THEODORE F. PREWITT, St. Louis, Mo.	October 17, 1904
1898	L. McLANE TIFFANY, Baltimore, Md.	October 23, 1916
1898	NATHANIEL P. DANDRIDGE, Cincinnati, Ohio	1910
1898	ROSWELL PARK, Buffalo, N.Y.	February 15, 1914
1898	ROBERT F. WEIR, New York, N.Y.	1927
1898	FREDERICK S. DENNIS, New York, N.Y.	March 8, 1934
1900	W. H. A. JACOBSON, London, England	July 27, 1917
1900	THEODORE KOCHER, Berne, Switzerland	October 3, 1916
1900	VINCENZ CZERNY, Heidelberg, Germany	October 3, 1916
1906	DUDLEY P. ALLEN, Cleveland, Ohio	January 6, 1915
1906	WILLIAM J. MAYO, Rochester, Minn.	July 28, 1939
1906	ROBERT ABBE, New York, N.Y.	March 7, 1928
1906	C. B. G. DENANCREDE, Ann Arbor, Mich.	May 6, 1921
1907	JOHN C. MUNRO, Boston, Mass.	December 6, 1910
1908	J. EWING MEARS, Philadelphia, Pa.	May 28, 1919
1909	LEWIS STEPHEN PILCHER, Brooklyn, N.Y.	December 24, 1934
1916	W. W. KEEN, Philadelphia, Pa.	June 7, 1932
1920	HENRY R. WHARTON, Philadelphia, Pa.	December 3, 1925
1927	JOHN CHALMERS DACOSTA, Philadelphia, Pa.	May 16, 1933
1929	D'ARCY POWER, London, England	May 18, 1941
1929	ALBIN LAMBOTTE, Esneux, Belgium	
1929	HENRI HARTMANN, Paris, France	
1929	TH. TUFFIER, Paris, France	October 27, 1929
1929	JOSEPH GUYOT, Bordeaux, France	
1929	GEORGES JEANNENEY, Bordeaux, France	
1929	F. DEQUERVAIN, Berne, Switzerland	January 23, 1940

ELECTED		DIED
1929	BERKELEY MOYNIHAN, Leeds, England	September 7, 1936
1929	HARVEY CUSHING, Boston, Mass.	October 7, 1939
1929	EDWARD W. ARCHIBALD, Montreal, Canada	1945
1929	JOHN M. T. FINNEY, Baltimore, Md.	May 30, 1942
1929	EVARTS GRAHAM, St. Louis, Mo.	March 4, 1957
1929	ELLISWORTH ELIOT, JR., New York, N.Y.	November 2, 1945
1929	RUDOLPH MATAS, New Orleans, La.	September 23, 1957
1929	DEAN D. LEWIS, Baltimore, Md.	1941
1929	EUGENE H. POOL, New York, N.Y.	1949
1929	GEORGE W. CRILE, Cleveland, Ohio	January 7, 1943
1929	EDWARD STARR JUDD, Rochester, Minn.	November 30, 1935
1929	DALLAS B. PHEMISTER, Chicago, Ill.	1951
1933	JOHN H. JOPSON, Mills, N.C.	December 4, 1954
1954	HAROLD FOSS, Danville, Pa.	August 11, 1967
1954	DIGBY CHAMBERLAIN, Leeds, England	
1954	FREDERICK COLLER, Ann Arbor, Mich.	November 5, 1964
1954	HOWARD NAFZIGER, San Francisco, Calif.	1961
1954	ARTHUR ALLEN, Boston, Mass.	March 18, 1958
1954	ERIK HUSFELDT, Copenhagen, Denmark	
1954	ALLEN WHIPPLE, New York, N.Y.	April 16, 1963
1954	SIR JAMES PATTERSON ROSS, London, England	July 5, 1980
1979	J. ENGLEBERT DUNPHY, San Francisco, Calif.	December 27, 1981
1979	FRANCIS D. MOORE, Boston, Mass.	
1979	OWEN WANGENSTEEN, Minneapolis, Minn.	January 13, 1981
1979	CLARENCE CRAFOORD, Sweden	
1979	JOHN GOLIGHER, Leeds, England	
1979	RODNEY SMITH, The Right Honorable Lord of Marlow, London, England	
1979	WILLIAM LONGMIRE, Los Angeles, Calif.	
1979	DAVID SABISTON, Durham, N.C.	
1979	ROBERT ZOLLINGER, Columbus, Ohio	

Winners of the Samuel D. Gross Prize

1895 "Inquiry into the Difficulties Encountered in the Reduction of Dislocations of the Hip."—Dr. Oscar H. Allis, Philadelphia, Pa.

1902 "Treatment of Certain Malignant Growths by Excision of the External Carotids."—Dr. Robert H. W. Dawbarn, New York, N.Y.

1905 "The Biology of the Micro-organisms of Actinomycosis."—Dr. James Homer Wright, Boston, Mass.

1910 "An Anatomical and Surgical Study of Fractures of the Lower End of the Humerus."—Dr. Astley P. C. Ashhurst, Philadelphia, Pa.

1915 "Surgery in the Treatment of Hodgkin's Disease."—Dr. John Lawrence Yates, Milwaukee, Wis.°

1920 "Some Fundamental Considerations in the Treatment of Empyema Thoracis."—Dr. Evarts A. Graham, St. Louis, Mo.

1925 "The Surgery of Pulmonary Tuberculosis."—Dr. John Alexander, Saranac Lake, N.Y.

1930 "Abnormal Arteriovenous Communications."—Dr. Emile Holman, Stanford University, San Francisco, Calif.

1935 "The Therapeutic Problems in Bowel Obstruction."—Dr. Owen H. Wangensteen, Minneapolis, Minn.

1940 "The Role of the Liver in Surgery."—Dr. Frederick Fitzherbert Boyce, New Orleans, La.

1945 "Parenteral Alimentation in Surgery with Special Reference to Protein and Amino Acids."—Dr. Robert Elman, St. Louis, Mo.

1950 "Localization of Brain Tumors with Radio-Active Agents."—Dr. George E. Moore, Minneapolis, Minn.

1955 "Liquid Plasma—Its Safety and Usefulness in Shock and Hypoproteinemia."—Dr. J. Garrott Allen, Chicago, Ill.

1962 "The Pathogenesis of Gastric and Duodenal Ulcers."—Dr. Lester Dragstedt, Gainesville, Fla.

1967 "Cholesterol Metabolism and Atherosclerosis as Influenced by Partial Small Bowel Intestinal Exclusion."—Dr. Henry Buchwald, University of Minnesota, Minneapolis, Minn.

1972 "Hepatic Metabolism in Human Cirrhosis: The Effect of Portacaval Shunt on Liver and Brain Metabolism."—Dr. Frederick A. Reichle, Temple University, Philadelphia, Pa.

1979 "Simulation of Congenital Heart Disease in Fetal Lambs."—Dr. Noel H. Fishman.

1983 "The Application of Decision Sciences to Surgical Judgment."—Dr. John R. Clarke, Medical College of Pennsylvania, Philadelphia, Pa.

Fellows Who Have Delivered the Annual Oration

1881	S. D. Gross	1916	Edward B. Hodge	1951	J. Montgomery Deaver
1882	D. Hayes Agnew	1917	J. Edwin Sweet	1952	Herbert R. Hawthorne
1883	William Hunt	1918	None	1953	Julian Johnson
1884	John H. Brinton	1919	None	1954	George Rosemond
1885	John H. Packard	1920	John G. Clark	1955	William H. Erb
1886	R. J. Levis	1921	J. Torrance Rugh	1956	George Willauer
1887	J. Ewing Mears	1922	George P. Muller	1957	Irvin E. Deibert
1888	C. B. G. deNancrede	1923	Walter Estell Lee	1958	Orville C. King
1889	John B. Roberts	1924	Robert H. Ivy	1959	James R. Jaeger
1890	DeForest P. Willard	1925	John Speese	1960	H. Taylor Caswell
1891	William G. Porter	1926	Damon B. Pfeiffer	1961	Donald R. Cooper
1892	T. G. Morton	1927	Emory G. Alexander	1962	John Y. Templeton, III
1893	C. W. Dulles	1928	Edward J. Klopp	1963	Edwin W. Shearburn
1894	W. B. Hopkins	1929	Edward T. Crossan	1964	Henry P. Royster
1895	John B. Deaver	1930	J. Stewart Rodman	1965	C. Everett Koop
1896	James M. Barton	1931	Hubley R. Owen	1966	Kenneth E. Fry
1897	Thomas R. Neilson	1932	Eldridge L. Eliason	1967	Thomas F. Nealon, Jr.
1898	O. H. Allis	1933	George M. Dorrance	1968	R. Robert Tyson
1899	William J. Taylor	1934	DeForest P. Willard	1969	H. L. Stahlgren
1900	None	1935	A. Bruce Gill	1970	Brooke Roberts
1901	H. R. Wharton	1936	Alexander Randall	1971	William T. Fitts, Jr.
1902	J. M. Spellissy	1937	Henry P. Brown, Jr.	1972	Joseph G. Bassett
1903	R. G. LeConte	1938	Isidor S. Ravdin	1973	Lloyd W. Stevens
1904	G. G. Davis	1939	John B. Flick	1974	Joseph W. Stayman
1905	J. Chalmers DaCosta	1940	Francis C. Grant	1975	Charles Fineberg
1906	Richard H. Harte	1941	William Bates	1976	Leonard Goldman
1907	Edward Martin	1942	S. Dana Weeder	1978	David Wagner
1908	Charles H. Frazier	1943	Frederick A. Bothe	1979	Frederick Wagner
1909	John H. Gibbon	1944	Calvin M. Smyth	1980	Clyde Barker
1910	Astley P. C. Ashhurst	1945	Adolph A. Walkling	1981	Moreye Nusbaum
1911	John H. Jopson	1946	John H. Gibbon, Jr.	1982	Michael O'Conner
1912	George C. Ross	1947	L. Kraeer Ferguson	1983	Jack Kolff
1913	William L. Rodman	1948	Jonathan E. Rhoads	1984	Simon Simonian
1914	Alfred C. Wood	1949	Francis C. Grant	1985	Francis E. Rosato
1915	Frances T. Stewart	1950	W. Emory Burnett	1986	Bruce E. Jarrell

Fellows of the Philadelphia Academy of Surgery

Active Fellows

	ELECTED	BORN	SPECIALTY
Alday, Edgard, M.D. Room 605, Jefferson Medical College 1025 Walnut Street Philadelphia, PA 19107	2-1-82	1933	G.S.
Au, Francis C., M.D. 3401 N. Broad Street Philadelphia, PA 19140	1-3-83	1943	G.S.
Bacharach, Benjamin, M.D. 1025 Walnut Street Philadelphia, PA 19107	5-3-71	1939	G.S.
Baker, Arthur G., Jr., M.D. 15 Morton Avenue Ridley Park, PA 19078	5-1-72	1935	G.S.
Balsara, Rohinton K., M.D. St. Christopher's Hospital for Children 5th and Lehigh Avenue Philadelphia, PA 19133	12-1-86	1935	Thoracic
Bar, Allen H., M.D. 301 S. Eighth Street Philadelphia, PA 19106	5-4-81	1942	G.S.
Barker, Clyde F., M.D. 3400 Spruce Street Philadelphia, PA 19104	12-7-70	1932	G.S.
Berkowitz, Henry D., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-6-75	1934	G.S., Vascular
Berman, Arnold T., M.D. 230 N. Broad Street Philadelphia, PA 19102	2-7-77	1940	Orthopedics
Brockman, Stanley K., M.D. Hahnemann University Hospital Broad and Vine Streets, Mail Stop 110 Philadelphia, PA 19102	10-7-74	1928	G.S., Thoracic
Brooks, Clint, M.D. Crozer-Chester Medical Center, Lewis House 15th and Upland Streets Upland, PA 19013	11-1-82	1932	G.S., Vascular
Brown, Arthur S., M.D. 300 Broadway Camden, N.J. 08103	11-5-84	1944	Plastics
Buchheit, William A., M.D. 3401 N. Broad Street Philadelphia, PA 19140	1-6-75	1933	G.S., Neurosurgery

	ELECTED	BORN	SPECIALTY
Chen, Chijen, M.D. Suite 233, Lankenau Medical Building Philadelphia, PA 19151	1-4-71	1933	G.S.
Clarke, John R., M.D. 3300 Henry Avenue Philadelphia, PA 19129	1-8-79	1943	G.S.
Clement, Gordon S., M.D. 15 West Wood Street Norristown, PA 19401	10-1-79	1934	G.S., Vascular
Colberg, James E., M.D. 1025 Walnut Street Philadelphia, PA 19107	1-10-84	1933	G.S.
Comerota, Anthony J., M.D. Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	11-4-85	1948	G.S.
Cossa, John P., M.D. St. Agnes Medical Center 1900 S. Broad Street Philadelphia, PA 19145	5-6-74	1933	G.S.
Daly, John M., M.D. 4 Silverstein Pavilion Philadelphia, PA 19104	12-1-86	1947	G.S.
DeClement, Frederick, A., Jr., M.D. 1900 S. Broad Street Philadelphia, PA 19145	2-7-77	1933	G.S.
Dent, Thomas L., M.D. Department of Surgery Abington Memorial Hospital Abington, PA 19001	5-6-85	1938	G.S.
DeSantis, Donald, M.D. 204 E. Chester Pike Ridley Park, PA 19078	12-2-74	1937	G.S.
DiGiovanni, Alphonse, M.D. Four Martin's Run Media, PA 19063	2-4-80	1931	G.S.
Donnelly, Joseph C., Jr., M.D. Suite 308, 606 Court Street Reading, PA 19601	1-8-68	1929	G.S., Thoracic, Cardiovascular
Duckett, John W., Jr., M.D. Children's Hospital One Children's Center Philadelphia, PA 19104	4-2-73	1936	G.S.
Dunn, Jeffrey Marc, M.D. 320 Melrose Avenue Merion Station, PA 19066	5-6-85	1946	Thoracic
Dzwonczyk, John, Jr., M.D. 45 Ashby Road Upper Darby, PA 19082	10-3-83	1946	Colon/Rectal

	ELECTED	BORN	SPECIALTY
Edie, Richard N., M.D. Jefferson Medical College 1025 Walnut Street, Suite 607 Philadelphia, PA 19107	11-5-84	1937	Thoracic
Edmunds, L. Henry, Jr., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-7-74	1931	Cardiovascular
Fallahnejad, Manoucher, M.D. Room 1004, The Graduate Hospital 19th and Lombard Streets Philadelphia, PA 19146	10-6-75	1936	G.S., Thoracic, Cardiovascular
Finnegan, James O., M.D. Chairman, Department of Surgery St. Agnes Medical Center 1900 South Broad Street Philadelphia, PA 19129	11-4-74	1938	G.S., Thoracic
Frazier, Thomas G., M.D. 600 Haverford Road Haverford, PA 19041	2-5-79	1943	G.S.
Gain, Thomas B., M.D. 230 N. Broad Street Philadelphia, PA 19102	12-5-77	1930	G.S.
Gonick, Paul, M.D. 227 North Broad Street Philadelphia, PA 19107	5-6-74	1930	Urology, G.S.
Gostigian, John J., M.D. 1016 Warrior Road Drexel Hill, PA 19026	5-4-70	1929	G.S.
Griffen, Ward O., Jr., M.D. American Board of Surgery, Inc. 1617 John F. Kennedy Boulevard Philadelphia, PA 19103	1-7-85	1928	Thoracic
Grosh, Julieta D., M.D. Department of Surgery Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	10-6-80	1939	G.S.
Gross, Richard H., M.D. Paoli Memorial Medical Building Paoli, PA 19301	5-6-74	1935	G.S.
Hamilton, Ralph W., M.D. 3400 Spruce Street Philadelphia, PA 19104	11-6-78	1933	Plastics
Hardesty, William H., M.D. 433 Bellevue Avenue Trenton, NJ 08618	10-7-68	1932	G.S.
Hartford, Charles E., M.D. Crozer-Chester Medical Center Chester, PA 19013	12-4-78	1932	G.S.

	ELECTED	BORN	SPECIALTY
Holst, Hazel, M.D. 3400 Spruce Street Philadelphia, PA 19104	11-5-73	1931	G.S., Plastics
Hulnick, Stuart J., M.D. 2600 N. Lawrence Street Philadelphia, PA 19133	12-6-76	1938	Plastics
Jarrell, Bruce E., M.D. 1025 Walnut Street Philadelphia, PA 19107	10-6-86	1947	G.S., Vascular
Kholoussy, A. Mohsen, M.D. 7th and West Market Street Pottsville, PA 17901	2-4-85	1947	G.S.
Kolff, Jacob, M.D. Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	1-10-84	1938	Thoracic
Krueger, Charles S., M.D. 131 Madison Avenue Mt. Holly, NJ 08060	5-4-70	1930	G.S.
Kukora, John S., M.D. 1245 Highland Avenue, Suite 600 Abington, PA 19001	10-6-86	1948	G.S.
LaRossa, Donato, M.D. 3400 Spruce Street Philadelphia, PA 19104	12-3-79	1941	Plastics
Lemole, Gerald, M.D. Suite 260, MOB-PUPMC 39th and Market Streets Philadelphia, PA 19104	1-7-74	1936	G.S., Thoracic
Lerner, Harvey J., M.D. Germantown Hospital Medical Center One Penn Boulevard 4th Floor, Wister Tower Philadelphia, PA 19144	2-3-69	1932	G.S.
Lyness, Samuel S., M.D. 958 County Line Road Bryn Mawr, PA 19010	12-4-72	1933	Neurosurgery
MacVaugh, Horace, III, M.D. 222 Lankenau Medical Building Philadelphia, PA 19151	10-5-70	1930	Thoracic
Maier, Willis P., M.D. 3401 North Broad Street Philadelphia, PA 19140	5-4-70	1933	G.S.
Marchildon, Michael B., M.D. 300 Broadway Camden, NJ 08103	4-2-84	1940	Pediatric Surgery
Matsumoto, Teruo, M.D. Suite 7150, 230 N. Broad Street Philadelphia, PA 19102	2-7-72	1929	G.S.

	ELECTED	BORN	SPECIALTY
Mattson, Ronald J., M.D. 207 Bryn Mawr Medical Building Bryn Mawr, PA 19010	11-5-84	1945	G.S.
McCombs, Peter, M.D. 1245 Highland, Suite 600 Abington, PA 19001	4-5-82	1944	G.S.
McLaughlin, Edward D., M.D. Department of Surgery Misericordia Hospital 54th Street and Cedar Avenue Philadelphia, PA 19143	5-5-69	1931	Thoracic
Miller, Leonard D., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-5-70	1930	G.S.
Mulholland, S. Grant, M.D. 1025 Walnut Street, Room 1112 Philadelphia, PA 19107	2-2-76	1936	Urology
Mullen, James L., M.D. 1000 Ravdin Institute 3400 Spruce Street Philadelphia, PA 19104	1-2-78	1942	G.S.
Mundth, Eldred D., M.D. 830 Old Lancaster Road Bryn Mawr, PA 19010	10-3-77	1933	Cardiothoracic
Murphy, J. Brien, M.D. 888 Glenbrook Avenue Bryn Mawr, PA 19010	5-5-86	1946	Plastic Surgery
Nakhgevany, Karim B., M.D. 302 Fairview Road Narberth, PA 19072	4-2-84	1937	G.S.
Nelson, Harry M., Jr., M.D. 15 Wood Street Norristown, PA 19401	5-1-72	1931	G.S.
Noone, R. Barrett, M.D. 888 Glenbrook Avenue Bryn Mawr, 19010	4-5-76	1939	Plastics
Nusbaum, Moreye, M.D. Graduate Hospital 19th and Lombard Streets Philadelphia, PA 19146	1-7-74	1929	G.S., Thoracic
O'Connor, Michael J., M.D. 117 Maple Avenue Bala Cynwyd, PA 19004	11-3-80	1941	Neurosurgery
O'Neill, James A., Jr., M.D. Children's Hospital One Children's Center Philadelphia, PA 19104	11-7-83	1933	Pediatric Surgery
Osterholm, Jewell, M.D. Suite 501, 1025 Walnut Street Philadelphia, PA 19107	11-4-74	1929	Neurosurgery G.S.

	ELECTED	BORN	SPECIALTY
Padula, Anthony, M.D. 8815 Germantown Avenue Philadelphia, PA 19118	11-1-82	1941	G.S.
Paskin, David L., M.D. Pennsylvania Hospital Eighth and Spruce Streets Philadelphia, PA 19107	12-1-75	1938	G.S.
Pavlides, Constantinos A., M.D. The William Penn Medical Building Suite 400 245 North Broad Street Philadelphia, PA 19107	10-6-86	1940	G.S.
Pello, Mark Joel, M.D. 300 Broadway, 8th floor Camden, NJ 08103	12-1-86	1949	Colon and Rectal
Perlman, Morton H., M.D. 230 N. Broad Street Philadelphia, PA 19102	2-7-72	1930	G.S.
Perloff, Leonard Jay, M.D. 3400 Spruce Street Philadelphia, PA 19104	10-1-84	1940	Vascular Surgery
Plzak, Louis, Jr., M.D. 830 Old Lancaster Road, Suite 107 Bryn Mawr, PA 19010	5-7-79	1934	Thoracic
Quill, Joseph R., M.D. 21 W. Fornance Street Norristown, PA 19401	1976	1928	G.S.
Reichle, Frederick A., M.D. 51 N. 39th Street Philadelphia, PA 19104	12-6-71	1935	G.S.
Rhoads, Jonathan E., Jr., M.D. York Hospital 1001 S. George Street York, PA 17405	1-8-79	1938	G.S., Thoracic
Ritchie, Wallace P., Jr., M.D., Ph.D. Department of Surgery Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	5-7-84	1935	G.S.
Rombeau, John L., M.D. Department of Surgery 4th floor, Silverstein Hospital of the University of Pa. 3400 Spruce Street Philadelphia, PA 19104	11-7-83	1939	Colorectal Surgery
Rosato, Ernest F., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-4-74	1936	G.S.
Rosato, Francis, M.D. Suite 605, 1025 Walnut Street Philadelphia, PA 19107	12-6-71	1934	G.S.

	ELECTED	BORN	SPECIALTY
Rosen, Harvey Marc, M.D. 700 Spruce Street, Suite 309 Philadelphia, PA 19106	5-6-85	1947	Plastics
Sachdeva, Ajit K., M.D. 3300 Henry Avenue Philadelphia, PA 19129	12-1-86	1951	General Surgery
Savarese, Ronald, M.D. 700 Spruce Street, Suite 101 Philadelphia, PA 19106	12-7-81	1943	G.S.
Scheuermann, Henry, M.D. 115 E. Township Line Road Upper Darby, PA 19082	11-1-82	1930	Plastics
Schwartz, Gordon, M.D. Suite 510, 1015 Chestnut Street Philadelphia, PA 19107	2-1-78	1935	G.S.
Sherk, Henry H., M.D. 1210 Brace Road Cherry Hill, NJ 08034	2-3-69	1930	Orthopedics
Sigel, Bernard, M.D. 3300 Henry Avenue Philadelphia, PA 19129		1930	Vascular Surgery
Silver, Stephen C., M.D. 1010 West Chester Pike Havertown, PA 19083	10-3-83	1946	G.S.
Simonian, Simon J., M.D. 230 N. Broad Street Philadelphia, PA 19102	12-6-82	1932	Vascular
Smink, Robert D., Jr., M.D. Suite 233, Lankenau Medical Building Philadelphia, PA 19151	1-7-80	1940	G.S.
Smullens, Stanton N., M.D. Suite 6255, 111 S. 11th Street Philadelphia, PA 19107	5-1-72	1936	G.S., Thoracic
Snyder, McC., Howard, III, M.D. Children's Hospital of Philadelphia 34th Street & Civic Center Boulevard Philadelphia, PA 19104	12-1-86	1943	Urology Pediatric Surgery Surgery
Solit, Robert W., M.D. Suite 8229, 111 S. 11th Street Philadelphia, PA 19107	11-4-74	1935	G.S.
Somers, Laurence A., M.D. 1335-49 W. Tabor Road, Suite 106 Philadelphia, PA 19141	11-6-78	1931	G.S., Pediatric
Spagna, Paschal M., M.D. MOB, Suite 260 51 North 39th Street Philadelphia, PA 19104	11-6-72	1935	Thoracic
Spence, Richard K., M.D. 300 Broadway, 8th floor Camden, NJ 08103	12-2-85	1945	Vascular Surgery

	ELECTED	BORN	SPECIALTY
Stephenson, Larry W., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-5-81	1944	G.S.
Vernick, Jerome J., M.D. Suite 6015, 111 S. 11th Street Philadelphia, PA 19107	1-3-77	1937	G.S.
Wagner, David K., M.D. 3300 Henry Avenue Philadelphia, PA 19129	2-3-69	1931	Pediatric Surgery
Wallace, Herbert W., M.D. The Graduate Hospital 19th and Lombard Streets Philadelphia, PA 19146	11-4-74	1930	G.S., Thoracic
Wein, Alan J., M.D. 3400 Spruce Street Philadelphia, PA 19104	5-3-76	1941	Urology
Weintraub, William, M.D. 1260 Lakemont Road Villanova, PA 19085			
Weiss, Stephen M., M.D. Fitzgerald Mercy Office Building Suite 207 1501 Lansdowne Avenue Darby, PA 19023	10-1-84	1947	Surgical Oncology
Whitaker, Linton A., M.D. 3400 Spruce Street Philadelphia, PA 19104	1-6-75	1936	Plastics
White, Jack C., M.D. Paoli Memorial Medical Building Paoli, PA 19301	12-4-72	1928	G.S.
Williams, Kirkley R., M.D. 207 Bryn Mawr Medical Building Bryn Mawr, PA 19010	5-1-71	1931	Thoracic
Yum, Keuk Y., M.D. Lankenau Medical Building, Suite 334 Philadelphia, PA 19151	10-7-74	1936	G.S.
Zaren, Howard A., M.D. 3300 Henry Avenue Philadelphia, PA 19129	10-6-86	1941	G.S.
Ziegler, Moritz M., M.D. Children's Hospital of Philadelphia 34th Street and Civic Center Boulevard Philadelphia, PA 19104	11-7-83	1942	Pediatric Surgery

Senior Fellows

Armitage, Harry V., M.D. Suite 406, Professional Building Crozer-Chester Medical Center Chester, PA 19013	10-6-58	1916	G.S.

	ELECTED	BORN	SPECIALTY
Bassett, James G., M.D. 3300 Henry Avenue Philadelphia, PA 19129	12-4-61	1919	G.S.
Bishop, Harry C., M.D. Children's Hospital One Children's Center Philadelphia, PA 19104	5-3-71	1921	G.S.
Bower, Robert M., M.D. 1185 Seaton Ross Road Radnor, PA 19087	11-6-67	1925	G.S.
Boyd, Robert T., III, M.D. 301 Keithwood Road Wynnewood, PA 19096	11-6-67	1925	G.S.
Buyers, Robert A., M.D. 1308 DeKalb Street Norristown, PA 19401	10-1-56	1917	G.S.
Camishion, Rudolph C., M.D. 3 Cooper Plaza, Suite 411 Camden, NJ 08103	1-4-65	1927	G.S., Thoracic
Closson, Edward W., M.D. Homestead Farm 260 North Main Street Lambertville, NJ 08530	12-5-66	1914	G.S.
Cohen, Erwin A., M.D. Medical Arts Building 60 E. Township Line Road Elkins Park, PA 19117	2-4-74	1925	G.S.
Cohn, Herbert E., M.D. Suite 8229, 111 S. 11th Street Philadelphia, PA 19107	12-6-65	1930	G.S.
Cooper, Donald R., M.D. 3300 Henry Avenue Philadelphia, PA 19129	10-6-52	1917	G.S.
D'Alonzo, Walter A., M.D. 1647 S. 15th Street Philadelphia, PA 19145		1914	G.S.
Davis, Richard A., M.D. 3400 Spruce Street Philadelphia, PA 19104	1-6-69	1925	Neurosurgery
DeLaurentis, Dominic A., M.D. Director, Department of Surgery Pennsylvania Hospital Eighth and Spruce Streets Philadelphia, PA 19107	5-4-64	1925	Cardiovascular
DeTuerk, John J., M.D. 2301 South Broad Street Philadelphia, PA 19148	5-7-71	1912	G.S.
Dorian, Alan L., M.D. 9 Maple Drive Conshohocken, PA 19428		1920	G.S.

	ELECTED	BORN	SPECIALTY
Engel, Gibson C., M.D. Suite 344, Lankenau Medical Building Philadelphia, PA 19151	1934	1898	G.S.
Fineberg, Charles, M.D. 902 Locust Street Philadelphia, PA 19107	12-7-59	1921	G.S.
Frobese, Alfred S., M.D. 1245 Highland Avenue Abington, PA 19001	1952	1919	G.S.
Garrison, Sherman, M.D. 108 West Commerce Street Bridgeton, NJ 08302	11-7-77	1915	G.S.
Gartland, John J., M.D. 620D Curtis Building 1015 Walnut Street Philadelphia, PA 19107	1-7-66	1918	Orthopedics
Geist, Donald C., M.D. Penn Wynn House, Apartment 1205 2201 Bryn Mawr Avenue Philadelphia, PA 19131	1941	1901	G.S.
Gorham, William K., III, M.D. 330 South Ninth Street Philadelphia, PA 19107	1-6-69	1927	G.S.
Gowen, George F., M.D. 1133 East High Street Pottstown, PA 19464	1-4-65	1923	G.S.
Grimes, Elmer L, M.D. 108 Kings Highway South Cherry Hill, NJ 08034	4-11-60	1914	G.S.
Grotzinger, Paul J., M.D. 2121 Valley Road Huntingdon Valley, PA 19006	10-1-56	1918	G.S.
Hall, John M., M.D. 604 General Scott Road Wayne, PA 19087		1915	G.S.
Harris, James S.C., M.D. Suite 108, 666 E. Penn Street Philadelphia, PA 19144	11-2-53	1914	G.S.
Harwick, Robert D., M.D. 3401 N. Broad Street Philadelphia, PA 19140	12-6-76	1923	Surgical Oncology
Hoeffel, Joseph M., Jr., M.D. 1643 Sherwood Road Rydal, PA 19046	12-5-55	1917	G.S.
Hughes, Eugene P., Sr., M.D. Northwest Surgical Associates 8815 Germantown Avenue Philadelphia, PA 19118	5-4-64	1924	G.S.

	ELECTED	BORN	SPECIALTY
Inouye, William Y., M.D. 3400 Spruce Street Philadelphia, PA 19104	1-4-65	1920	G.S.
Johnson, Julian, M.D. Villa 49, 1400 Waverly Road Gladwyne, PA 19035	3-2-42	1906	G.S.
Jones, Robert K., M.D. Lankenau Medical Building, Suite 115 Philadelphia, PA 19151	1965	1924	Neurosurgery
Lamp, J. Curtis, M.D. 888 Glenbrook Avenue Bryn Mawr, PA 19010	11-7-66	1918	Plastics
Langfitt, Thomas W., M.D. President and CEO The Glenmede Trust Company 229 South 18th Street Philadelphia, PA 19103	2-7-66	1927	Neurosurgery
Lauby, Vincent, M.D. 3401 N. Broad Street Philadelphia, PA 19140	10-1-62	1921	G.S., Thoracic Surgery
Lemmon, William M., M.D. 1653 Mt. Pleasant Road Villanova, PA 19085	5-2-66	1920	G.S., Thoracic Surgery
Mackie, Julius A., M.D. 3400 Spruce Street Philadelphia, PA 19104	11-7-66	1927	G.S.
Marks, Gerald, M.D. 1100 Walnut Street, Suite 702 Philadelphia, PA 19107	10-5-70	1925	G.S.
McKeown, John J., Jr., M.D. Department of Surgery Mercy Catholic Medical Center Darby, PA 19023	4-3-61	1919	G.S.
Medinger, Frederick G., M.D. 1245 Highland Avenue Abington, PA 19001	1950	1911	G.S.
Morani, Alma D., M.D. 3665 Midvale Avenue Philadelphia, PA 19129	2-4-74	1907	Plastics
Morse, Dryden P., M.D. Deborah Heart & Lung Center Browns Mills, NJ 08105	5-6-63	1924	Cardiothoracic
Moss, N. Henry, M.D. Suite 104, 1335 West Tabor Road Philadelphia, PA 19141	11-2-64	1925	G.S.
Murphy, John J., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-6-58	1920	Urology

	ELECTED	BORN	SPECIALTY
Murtaugh, Frederick, Jr., M.D. Hospital of the University of Pa. 210 White Building 3400 Spruce Street Philadelphia, PA 19104	11-6-67	1917	Neurosurgery
Neal, Hunter S., M.D. Suite 334, Lankenau Medical Building Philadelphia, PA 19151	11-6-66	1923	G.S.
Nemir, Paul, Jr., M.D. Room 1200, 19th and Lombard Streets Philadelphia, PA 19146	1-3-55	1920	G.S., Thoracic Surgery
Pechin, Sergius P., M.D. 430 Sycamore Mills Road Media, PA 19063	2-4-74	1914	G.S.
Pecora, David V., M.D. Medical Arts Pavilion, Suite 102 Christiana Hospital 4745 Stanton-Ogletown Road Newark, DE 19713-2070	2-3-75	1916	G.S., Thoracic Surgery
Pierucci, Louis, M.D. 455 Route, #70 West Cherry Hill, NJ 08002	12-6-65	1928	G.S., Thoracic Surgery
Pitt, Leldon P., M.D. 301 South Eighth Street, Suite 3E Philadelphia, PA 19106	12-5-60	1920	G.S.
Randall, Peter, M.D. 3400 Spruce Street Philadelphia, PA 19104	4-1-62	1923	Plastics
Ranieri, Tito A., M.D. 2320 N. Broad Street Philadelphia, PA 19145	1951	1912	G.S.
Reagan, Lindley B., M.D. The Cadbury 2150, Route #38 Cherry Hill, NJ 08002	2-2-53	1918	G.S.
Rhoads, Jonathan E., Sr., M.D. 3400 Spruce Street Philadelphia, PA 19104	1943	1907	G.S.
Roberts, Brooke, M.D. 3400 Spruce Street Philadelphia, PA 19104	1-4-54	1917	G.S.
Roberts, John M., M.D. Northwest Surgical Associates 8815 Germantown Avenue Philadelphia, PA 19118	5-4-64	1926	G.S.
Saris, Demetrius S., M.D. 230 N. Broad Street Philadelphia, PA 19102	2-1-65	1921	G.S.
Schumann, Francis, M.D. Five Pleasant Street Machias, ME 04654	4-2-62	1914	G.S.

	ELECTED	BORN	SPECIALTY
Schwegman, Cletus W., M.D. 3400 Spruce Street Philadelphia, PA 19104	1951	1914	G.S.
Snedden, Hal, M.D. 350 Hidden River Road Narberth, PA 19072	12-1-75	1922	G.S.
Steel, Howard H., M.D. Shriners Hospital Philadelphia Unit 8400 Roosevelt Boulevard Philadelphia, PA 19152	5-6-68	1921	Orthopedics
Strong, George H., M.D. Suite 6128, 111 S. 11th Street Philadelphia, PA 19107	4-2-56	1914	Urology
Templeton, John Y., M.D. Suite 6255, 111 S. 11th Street Philadelphia, PA 19107	1954	1915	G.S.
Tropea, Frank, Jr., M.D. 2512 Cleveland Street Philadelphia, PA 19145	2-3-58	1912	G.S.
Trout, Robert, M.D. 2100 Keystone Avenue, Suite 400 Drexel Hill, PA 19026	11-3-69	1922	Thoracic Surgery
Tyson, R. Robert, M.D. 3401 N. Broad Street Philadelphia, PA 19104	12-6-54	1920	G.S.
Wagner, Frederick B., Jr., M.D. Room 306, Scott Building 1020 Walnut Street Philadelphia, PA 19107	1-7-52	1916	G.S.
West, Clifton, Jr., M.D. Rt. 3, Box 127 Chestertown, MD 21260	2-1-60	1923	G.S.
Wolferth, Charles C., Jr., M.D. 227 N. Broad Street, Suite 100 Philadelphia, PA 19107	11-1-63	1928	G.S.
Zaslow, Jerry, M.D. Medical Arts Building 60 East Township Line Road Elkins Park, PA 19117	10-6-58	1918	G.S.

Inactive Fellows

Bucher, Robert M., M.D. 2451 Fillingim Street Mobile, AB 36617	12-6-54	1920	G.S.
Caswell, H. Taylor, M.D. 715 Bryn Mawr Avenue Narberth, PA 19072	5-7-51	1913	G.S.

	ELECTED	BORN	SPECIALTY
Cayten, C. Gene, M.D. Director, Department of Surgery Misericordia Hospital Medical Center 600 East 23rd Street Bronx, NY 10466	1-7-80	1941	G.S.
Cooper, Robert A., M.D. 804 Mark 70, Route 70 Cherry Hill, NJ 08034		1917	G.S.
Culf, Norris K., M.D. 644 Norristown Road Horsham, PA 19044	11-3-75	1931	G.S., Plastics
Farrell, Harry L., M.D. 135 S. 20th Street Philadelphia, PA 19103		1905	
Gilmour, William R., M.D. 6616 Woodland Avenue Philadelphia, PA 19142	1928	1891	G.S.
Haupt, George J., M.D. Suite 306, Lankenau Medical Building Philadelphia, PA 19151	10-5-59	1924	G.S.
Hopkins, John E., M.D., 85 Crestline Road Strafford, Wayne, PA 19087	12-2-56	1921	G.S.
Kaplan, Louis, M.D. 1204 Greentree Lane Narberth, PA 19072	4-4-47	1904	G.S.
King, Orville C., M.D. 8022 Roanoke Street Philadelphia, PA 19118			
Lightfoot, William P., M.D. 13 Oyster Reef Drive Hilton Head Island, SC 29928	10-5-70	1920	G.S.
Manges, W. Bosley, M.D. Rt. 2, Box R6B Santee, SC 29142	11-6-61	1918	G.S.
Martin, William L., M.D. 402 Holly Lane Wynnewood, PA 19096	11-6-61	1918	G.S.
O'Neill, James F., M.D. 8116 Bustleton Avenue Philadelphia, PA 19152	1954	1910	G.S.
Ristine, Edward R., M.D. 17 Clinton Avenue Mantua, NJ 08051	1954	1910	G.S.
Rosemond, George P., M.D. 3401 N. Broad Street Philadelphia, PA 19140	1945	1910	G.S.
Sacks, Charles Louis, M.D. 2275 Ibis Isle Road Palm Beach, FL 33480	5-1-67	1914	G.S.

	ELECTED	BORN	SPECIALTY
Stevens, Lloyd, M.D. 1204 Round Hill Road Bryn Mawr, PA 19010	10-4-48	1914	G.S.
Singmaster, Lawrence, M.D. 272 Cheswold Lane Haverford, PA 19041	1-2-57	1916	G.S.
Gordon L. Tobias, M.D. Vice President Medical Affairs Delaware Valley HMO Greater Delaware Valley Health Care, Inc., 9 LaCrue Street P.O. Box 1111 Concordville, PA 19331	12-4-78	1927	Urology
Troncelliti, Manrico, M.D. Suite 101, DeKalb Fornance Building DeKalb and Fornance Streets Norristown, PA 19401	5-4-70	1915	G.S.
Weber, Edgar H., M.D. 3008 E. Powell Avenue Evansville, TN 47714			G.S.
Wells, J. Ralston, M.D. Stone Island Estates Enterprise, FL 32763			G.S.

Non-Resident Fellows

Ainsworth, Thomas H., Jr., M.D. 40 Country Club Drive Carmel Valley, CA 93924-1201		1920	G.S.
Allbritten, Frank F., Jr., M.D. P.O. Box 177 Cunningham, KS 67035		1914	G.S., Thoracic Surgery
Austin, George, M.D. 2323 De La Vista Ste. 104 Santa Barbara, CA 93105			Neursurgery
Bailey, Charles P., M.D. 1717 "M" Street Belmar, NJ 07719			G.S., Thoracic Surgery
Beljan, John R., M.D. 6490 Saddle Drive Long Beach, CA 90815	11-5-84	1930	G.S.
Bernhard, Victor M., M.D. University of Arizona Health Science Center, Department of Surgery Tucson, AZ 85724	4-5-82	1927	G.S.
Boland, James P., M.D. 1929 Parkwood Road Charlestown, WV 25314	10-7-68	1931	G.S., Thoracic Surgery
Crichlow, Robert W., M.D. Dartmouth Medical School Hanover, NH 03755		1932	G.S.

	ELECTED	BORN	SPECIALTY
Davila, Julio C., M.D. 1012 N. Tenth Street Wausau, WI 54401	4-1-68	1928	Thoracic Surgery
Deutsch, Joel, M.D. Director of Surgery Mt. Sinai Hospital 500 Blue Hills Avenue Hartford, CT 06112	12-7-70	1926	G.S.
Fry, Kenneth E., M.D. 621 University Avenue Walla Walla, WI 99362		1902	G.S.
Goldman, Leonard, M.D. 3013 Nottingham Drive Shreveport, LA 71115		1930	G.S.
Goldsmith, Harry S., M.D. Hitchcock Clinic Hanover, NH 03755	5-1-72	1929	G.S.
Hume, H. Alan, M.D. R.F.D. #1, Pond Road Oakland, ME 04963	2-3-64	1926	G.S.
Johnson, Robert G., M.D. 600 Shadow Lane Las Vegas, NV 89106		1920	Thoracic
Koop, C. Everett, M.D. 4 West Drive Bethesda, MD 20814		1916	Pediatric Surgery
Law, F. Dana, M.D. Port William Medical Associates Carollton, KY 41008	2-3-64	1924	G.S.
Manges, Lewis C., M.D. Seven Palm Lane Tangerine, FL 32777		1906	G.S.
Masson, Newton L., M.D. 3572 Montclair Circle Shingle Springs, CA 95682			
McNamara, Marian F., M.D. University Surgeons, P.C. University Health Center 6-B, 4201 St. Antoine Detroit, MI 48201	1-4-82	1946	Vascular
Morris, Robert S., M.D. 938 Strangler Fig Lane Sanibel, FL 33957			
Nealon, Thomas F., Jr., M.D. St. Vincent's Hospital 170 W. 12th Street New York, NY 10011		1920	G.S.
Royster, Henry P., M.D. 1507 Canterbury Road Raleigh, NJ 27608	1950	1909	Plastics

	ELECTED	BORN	SPECIALTY
Sain, Fletcher D., M.D. 1200 West Haven Boulevard Rocky Mount, NC 27803	12-5-60	1909	G.S.
Sandzen, Sigurd C., Jr., M.D. St. Paul Medical Building 5959 Harry Hines Boulevard Dallas, TX 75235	10-4-76	1932	Orthopedics
Schmidek, Henry H., M.D. Neurosurgical Practice Office Medical Center Hospital of Vermont DeGoesbriand Unit Burlington, VT 05401	2-7-77	1937	Neurosurgery
Sears, Henry F., M.D. New England Deaconess Hospital 110 Francis Street Boston, Massachusetts 02215	4-6-81	1940	Surgical Oncology
Sensenig, David M., M.D. 436A State Street Bangor, ME 04401		1921	Thoracic
Sencindiver, P. Victor, M.D. 908 S. Beach Avenue Beach Haven, NJ 08008	2-1-65	1927	G.S.
Stahlgren, LeRoy H., M.D. Director of Surgery Department St. Barnabas Medical Center Livingston, NJ 07039	11-7-60	1924	G.S.
Stainbach, William C., M.D. 2221 Buttonwood Road Berwyn, PA 19312	4-1-57	1916	G.S.
Stayman, Joseph W., Jr., M.D. Hearthstone Ridge Rt. 1, Box 308E Landrum, SC 29356	1950	1915	G.S.
Thomas, Paul A., Jr., M.D. Associate Professor of Surgery Division of Cardiothoracic Surgery P.O. Box 6998 Chicago, IL 60680	1-4-71	1923	Thoracic Surgery
Thompson, James C., M.D. Department of Surgery University of Texas Galveston, TX 77550			G.S.

Annual Oration for 1981

Vasopressin in Surgery

MOREYE NUSBAUM, M.D.

Vasopressin is the antidiuretic hormone and through biologic evolution it has become the mediator and regulator for water conservation in man. ADH is released by the posterior pituitary under conditions of water deprivation when plasma osmolarity is elevated, or when extracellular volume is depleted regardless of the level of plasma osmolarity. In man the site of ADH action is the renal collecting duct.

ADH has a number of nonrenal actions which have surgical significance. It is a potent vasopressor and was given its name originally on the basis of its vasoconstrictor action. It has recently been found to have effects on the central nervous system and on blood coagulation. In 1953 du Vigneaud determined the structure of ADH and its synthesis and received the Nobel Prize for his efforts in 1955. Vasopressin is a nona peptide with two cysteine residues forming a ring. The integrity of the ring is important for biologic activity. Amino acid substitutes dictate specific physiologic actions. Commercial preparations of ADH of pituitary origin are usually derived from both bovine and porcine pituitaries, and therefore, are mixtures of eight arginine and eight lysine vasopressin.

The kidney is the major site of enzymatic degredation. A half life of vasopressin in the circulation in man is approximately ten minutes due to inactivation by peptidases in various tissues, primarily the kidney. The development of techniques for solid phase pepitude synthesis has made it possible to screen and synthesize several analogs of ADH. Two analogs which have excited considerable attention are desamino eight arginine vasopressin with enhanced antidiuretic activity used primarily in the treatment of diabetes insipidus and triglycyl lysine vasopressin which enhances vasoconstrictor activity and is of potential great value in controlling bleeding because of its longer duration of action and less coronary vasoconstriction. Of importance to surgeons is the ADH sensitive diabetes insipidus. This results from failure to secrete adequate quantities of antidiuretic hormone, resulting in polyuria with excretion of a dilute urine with specific gravity of 1.001 to 1.005.

Trauma or surgery in the region of the pituitary and hypothalmus, central nervous system malignancies and infections may cause a decrease in ADH release. Once recognized replacement therapy may be given by intravenous intramuscular or inhalation techniques to control symptomatology.

Inappropriate secretion of ADH is of surgical significance. Excessive production of ADH with resultant retention of water and dilutional hyponatremia may occur in patients with a variety of tumors, such as carcinoma of the lung, pleura, liver and retroperitoneum, as well as idiosyncratic ADH release in the

postoperative period after a variety of stressful operations and anesthetics. Inappropriate ADH may occur in patients with head injuries, meningitis, encephalitis, pulmonary infections, and may be released by drugs such as Vincristine and Cyclophosphomide, certain antidepressants and anticonvulsants. Prostoglandins, Lithium Carbonate, Methoxoflorane and the antibiotic Demeclocycline all antagonize the actions of ADH. Demeclocycline has been used successfully to promote diuresis in patients with water intoxication due to inappropriate ADH release associated with certain postoperative states and tumors.

The non-renal actions of ADH include effects on the cardiovascular system, the smooth muscle of the gut and on blood coagulation. The pressor effects of ADH is a general one on smooth muscle of all parts of the vasculature. The effect is a direct one on contractile elements. It is neither antagonized by adenergic blocking agents nor prevented by vascular denervation or local ph changes. The effects on blood pressure are mediated by barorecptors, therefore in conscious man, quite large amounts of vasopressin must be given to produce a significant rise in blood pressure.

The effects of vasopressin on the heart are indirect and are the result of coronary vasospasm with decreased coronary blood flow and of refluxly induced alterations in vagal and sympathetic tone resulting in myocardial ischemia.

Patients with coronary insufficiency while receiving vasopressin infusions demonstrate EKG changes similar to those observed after exercise tests. Patients with coronary insufficiency experience anginal pain even with small doses given to control diabetes insipidus. Vasopressin induced myocardial ischemia and death has been reported first by Slotnick and Teigland in 1951, and by numerous other investigators since. This is an important consideration in the use of vasopressin for the control of gastrointestinal hemorrhage.

Vasopressin effects the smooth muscle of the gastrointestinal tract. Motility is markedly increased, as well as tone. The effect is greater on the large than the small bowel. Its effect on the uterus is primarily from the oxytocic analogue and its value in obstetrics is well known.

Manucci in 1977 described an unexpected action of ADH and its analogs by demonstrating its effectiveness in the management of moderately severe hemophilia and von Willebrand's disease. These peptides, particularly the analogue desmopressin, were effective in increasing levels of factor VIII, and can be administered prophylactically during surgical procedures to prevent bleeding. The mechanism of this action remains unknown.

Vasopressin has had its greatest clinical application as an adjunct in the control of gastrointestinal bleeding of multiple etiologies, including portal hypertension with gastrointestinal varices; hemorrhagic gastritis, gastric erosions, Mallory-Weiss tears, stress ulcers, duodenal and gastric ulcers, arteriovenous malformations, colonic diverticula, traumatic injuries to the pelvis and spleen, post-partum hemorrhage, bronchiectasis with hemoptysis, hemorrhagic cystitis, pancreatitis, and untold others including protection of the gastrointestinal tract from radiation therapy. Many reports are anecdotal in relation to its specific use for a specific entity. The greatest experience, however, has been with the control of portal hypertension with esophageal varix hemorrhage.

Over two hundred publications and ten thousand patients have been reportedly treated with vasopressin in the past twenty years throughout the world.

Intravenous infusion of boluses of vasopressin in the dosage of forty to eighty pressor units diluted in 200cc. of fluid over a period of thirty minutes was first used in the management of varicele hemorrhage by Kehne and his colleagues in 1956. Follow-up studies on its use rapidly proliferated with experience of Sheldon and Sherlock reported in Lancet in 1960 describing their experience with the control of bleeding from esophageal varix hemorrhage using similar techniques. The dangers, however, in the systemic use of vasopressin were quickly recognized and included decreases in cardiac output resulting from vasoconstriction of the coronary arteries, and extreme water retention resulting from its antidiuretic effect. In 1967 Nusbaum, Baum, Sakiyalak and Blakemore reported use of infusions of fractional doses of vasopressin continuously into the superior mesenteric artery in dogs and later in man, thus seemingly reducing the side effects of the vasopressin infusion. This form of therapy, although successfully utilized by numerous clinical centers with reports in the literature, originally was questioned by Donaldson in 1970. A major debate developed as to the value of infusions of vasopressin in the cases of bleeding from the gastrointestinal tract and as to whether intraarterial or intravenous route is preferable. A number of studies have been performed in animals, especially with regard to the possible side effects, emphasizing mesenteric vascular complications and cardiac complications associated with this technique. Simmons and Baum in 1977, utilizing a radioimmunossay technique for circulating vasopressin demonstrated in dogs and monkeys that the autoregulatory relationship between the hepatic artery and portal vein very effectively maintains the total hepatic blood flow during vasopressin infusions. Studies by Ring and Baum, and by Kerr and Swan, have indicated that the response in the monkey is most similar to man and primates, and would thus appear to be the most suitable model for study of the effects of vasopressin infusions in man. The studies by Barr and Rosch, Sirinek and Thomford, and others in dogs, monkeys, and man have demonstrated that continuous intravenous low dose vasopressin infusions were as effective as continuous intra-arterial infusions in decreasing superior mesenteric arterial flow, as well as portal pressure. The initial use of bolus injections of vasopressin did not produce consistently significant control of variceal bleeding, possibly because the large doses given produced a short duration of action relative to the short half life of vasopressin in the circulation. In addition, these maximum doses produced a coronary vasospasm and decreased cardiac output, and in patients with coronary insufficiency produced serious myocardial damage. This has not deterred many medical centers from continuing to use bolus techniques in vasopressin administration to control urgent bleeding varices in preparation for emergency portacaval shunts. The development of tachyphylaxis from bolus injections has been described by multiple authors demonstrating that subsequent doses of vasopressin produce less of a pharmacologic effect with time. With the use of fractional infusions of vasopressin in the dosage range of two tenths to six tenths of a pressor unit per cc per minute infused either through a superior mesenteric arterial cath-

eter or through an intravenous line, gives a continous effective dose of vasopressin in the circulation and does not lead to the development of tachyphylaxis even with infusions observed for as long as fourteen days. Multiple authors, however, have demonstrated utilizing both the experimental animal and cirrotic man, that even these fractional doses of vasopressin produce a maximal antidiuretic hormone effect, as well as decreases in cardiac output. Sirinek demonstrated that infusions of vasopressin in association with isoproterinol infusions resulted in maximum reproducible decreases in superior mesenteric flow and portal venous pressure while preventing the cardiovascular side effects of decreased cardiac output and coronary flow. Recently, Grozmann et al, have demonstrated in portal hypertensive dogs that nitroglycerine can improve cardiac performance and favorably influence the splanchnic effects of vasopressin. They have studied a series of fourteen patients who have received either intravenous vasopressin alone, or vasopressin plus nitroglycerine four tenths of a milligram sublingually and measured hemodynamic parameters. They found that the addition of nitroglycerine completely abolished the toxic effects of vasopressin on cardiac hemodynamics while further enhancing the reduction of wedged hepatic venous pressure. This last effect was achieved without a further decrease in hepatic blood flow. These results have suggested that the addition of nitroglycerine to vasopressin therapy may reduce the complications and improve the effectiveness of vasopressin in the treatment of variceal bleeding. This has resulted in a more recently aggressive approach in management of variceal bleeding by using high doses of vasopressin up to nine tenths of a pressor unit per cc per minute for short periods of infusion of four to six hours to obtain rapid cessation of hemorrhage, in association with protection of the coronary circulation by sublingual administration of nitroglycerin. Following the cessation of hemorrhage, attempts at transendoscopic sclerosing injections of the varices have resulted in the emergent control of variceal hemorrhage in a growing percentage of patients. Recent reports by Librek from the Liver Institute in France, indicated that the administration of propanalol by mouth is effective in preventing recurrent gastrointestinal bleeding in patients with cirrhosis and portal hypertension who bleed from esophageal varices and gastric erosions. A controlled study of patients free of recurrent gastrointestinal bleeding one year after inclusion in the study, was 96% in the propanalol group and 50% in the placebo group. As a result of this study, a few clinics in the United States and France are advocating immediate control of variceal hemorrhage by high dose vasopressin infusions protected by nitroglycerine, with control of acute hemorrhage, by sclerosing injections through the endoscope with maintenance propanalol therapy to decrease the risk of rebleeding in the post injection state. Whether the long term effects of such treatment protocols will obviate the necessity of emergency portacaval shunts and possibly emulate survival from shunting procedures without the production of encephalopathy remains highly speculative, but worthy of further investigation. Although the current controversy as to the similar effectiveness of intravenous and intra-arterial continuous infusions of vasopressin in the management of gastroesophageal varices seems to have been settled, the question of whether intravenous vasopressin or selective intra-arterial vasopressin infusions are of equal

value in the management of arterial gastrointestinal bleeding, remains highly controversial and anecdotal. Even though numerous clinical studies would indicate that the value of vasopressin infusion and cessation of hemorrhage from esophageal varices ranges somewhere between 75% and 85% effective, the control of gastrointestinal hemorrhage from an arterial source has only been reported effective in approximately 50% of cases. Major experience has been with the selective infusion of intra-arterial vasopressin through an indwelling selective arterial catheter placed at the time of arteriographic demonstration of bleeding, and very little experience has been reported from continuous infusions of intravenous vasopressin. Variations of effectiveness of vasopressin infusions in the control of arterial hemorrhage are great. Vasopressin infusion was effective in only 50% of cases of chronic duodenal ulcer bleeding in the collected series of patients reported. This may well be because of the dual blood supply of the duodenum having contributions from both the celiac axis and the superior mesenteric artery, resulting in continued bleeding due to the failure to constrict the alternate source of anatomic blood supply to the ulcer crater. In addition, it may well be that the chronic duodenal ulcer produces changes within the wall of the vessel that make it less reactive to vasoconstrictors. However, mucosal bleeding lesions, such as those seen in hemorrhagic gastritis and Mallory-Weiss tears and superfical erosions are very amenable to vasopressin therapy, and have proven 90% effective in stopping hemorrhage from these sources, probably because of the shunting of blood flow from the mucosa by the opening up of arterial venous shunts in the submucosa, as well as major vasoconstriction of the branches of the left gastric artery. The effectiveness of vasopressin in controlling bleeding from colonic diverticula has been reported by Baum, Ring and Roche and others in the literature. Review of the reported cases demonstrate that approximately 75% of the bleeding diverticula occur in the colon to the right of the splenic flexure. Infusions of vasopressin through the superior mesenteric artery or the inferior mesenteric artery have been successful in initially controlling 88% of these bleeding diverticula, and have resulted in avoidance of surgery in 75% of these patients reported. We have had two anecdotal experiences of the control of left sided bleeding diverticula with intravenous infusions of vasopressin. Whether this is an alternate method of managing patients with suspected diverticular bleeding, irrespective of demonstration of the source of bleeding remains to be studied in a very carefully organized clinical setting. There have been numerous extensions of the utilization of vasopressin in a variety of clinical settings of importance to the surgeon. Its use during the performance of porto systemic shunt has been reported to faciliate exposure and reduce operative time and blood loss. Vasopressin infusions have been shown to increase survival rates and reduce tissue damage in dogs subjected to acute pancreatitis by the closed duodenal loop techniques. Vasopressin infusions modified and partially reversed the disease process as evaluated by studies of pancreatic enzymes and histologic evaluation of tissues. Vasopressin may have produced these effects by either directly reducing pancreatic exorcrine flow and stabilizing pancreatic blood flow or indirectly by increasing reabsorption of excess peritoneal fluid.

Various centers studying radiotherapy protection by vasopressin have dem-

onstrated that the damaging effect of ionizing radiation is reduced in the presence of lower tissue oxygen tension by producing regional vasoconstriction by infusions of vasopressin. A major application of this type of therapy would be in the treatment of certain gynecologic malignancies if one could protect the small intestine from radiation damage. The recent introduction of nitroglycerine as a protection to the cardiac complications of vasopressin may make this technique more clinically manageable. The use of vasopressin in the control of gastrointestinal hemorrhage remains controversial not only because of conflicting reports of its effectiveness and the anecdotal nature in which such case reports are presented but because increasing varieties of complications of vasopressin infusions have been reported. These are infrequent and generally minor but on occasion they may be catastrophic. Complications may be associated with either the percutaneous catheterization or the pharmacologic effects of the hormone itself. It must be recognized that a maximum antidiuretic effect is obtained with even fractional doses of vasopressin given clinically. This results in a dilutional hyponatremia and if unrecognized and uncorrected may produce convulsions and even death. Frequent monitoring of serum electrolytes and careful attention to fluid overload and urinary output must be given to patients undergoing infusions of vasopressin. One also must recognize that vasopressin is a potent coronary vasocontrictor even in fractional doses given through an intra arterial or intravenous catheter. Sufficient amounts of active vasopressin remains in the circulation producing coronary vasocontriction. In patients with underlying myocardial ischemia, extension of coronary insufficiency and even frank infarction and death has been reported. It is important to carefully monitor all patients receiving vasopressin with continuous electrocardiographic displays and pulse rate determinations. Early evidence of myocardial ischemia by ST changes as well as inappropriate decrease in pulse rates portend the development of myocardial damage and should lead to a reduction in the dose or temporary elimination of the vasopressin infusion. The utilization of nitroglycerin in association with vasopressin infusion may prove to decrease the dangers of producing myocardial damage by decreasing the coronary vasoconstrictive effects of the vasopressin itself. In addition, the development of several analogues of vasopressin have been reported. Several of these in addition to producing a longer effect of infused vasopressin have demonstrated less coronary vasoconstrictive activity. Clinical trials instituted in Sweden and in the United States indicate that these vasopressin analogues may be the drugs of choice in the future managing of gastrointestinal bleeding. Local complications can occur in association with both intra arterial and intravenous administration. The longer the catheter is left indwelling in the femoral artery, the greater the changes of local thrombosis or pseudoaneurysm developing. Although hematomas in the groin are common and occur in 20% of patients, occlusive complications requiring arterial surgery have been reported in only approximately 2%. Recent reports indicate increasing complications with continuous intravenous vasopressin infusions, namely infiltration of vasopressin into the subcutaneous tissue with subsequent necrosis. Ischemic gangrene at these sites have resulted in occasional amputations and have also necessitated skin grafts to local areas of necrosis. Clostridial sepsis has been

reported in areas of subcutaneous infiltration and emphasizes the dangers of such administration so that one should only infuse vasopressin by cutdown or a central venous line. There has been at least one case reported of gastric ischemia produced by vasoconstriction associated with vasopressin which led to anaerobic gas gangrene within the gastric wall and a fatality. There have been several case reports in the literature of mesenteric arterial and portal venous thrombosis developing during the course of vasopressin infusion. It is difficult to evaluate the role of vasopressin in this mortal complication resulting in extensive gangrene of the gastrointestinal tract. Review of the literature demonstrates similar case reports of mesenteric arterial and portal thrombosis in patients with portal hypertension and stagnant flow resulting in death in the absence of vasopressin use. It is certainly reasonable that mesenteric thrombotic complications can result from this mode of therapy and one should be very careful to monitor local abdominal signs during infusions of vasopressin. Fortunately, this complication has been very low. There have also been reports of several cases of hepatic necrosis in patients who have had vasopressin infusion. One case report indicates that a patient with a celiac axis chronic occlusion developed extensive hepatic necrosis following the infusion of superior mesenteric artery with vasopressin for several days. Under normal anatomic conditions vasopressin infusions appear to be protective of the hepatic artery blood flow and even direct infusions of vasopressin into the hepatic artery have resulted in only initial small decreases in hepatic arterial flow which have then returned to normal and super normal levels following constriction of the superior mesenteric artery by systemic leakage of vasopressin. Another unusual complication associated with vasopressin infusion has been the development of spontaneous bacterial peritonitis. The peritonitis differed from the characteristic picture of spontaneous peritonitis in the cirrhotic patient in which a single strain of bacteria is found, in that multiple enteric organisms are found on aspiration of the peritoneal cavity. It is suspected that reduced perfusion resulting from the action of vasopressin renders the intestinal mucosal barrier less effective and thus allows migration of bacteria from the intestine into the peritoneal cavity. Although the role of vasopressin in the management of gastrointestinal bleeding remains controversial, it has stimulated a tremendous amount of research and clinical activity in an effort to decrease the morbidity and mortality in this group of seriously ill patients. Several outstanding clinical surgeons have commented in discussions concerning vasopressin therapy in surgery that gastrointestinal bleeding is a surgical emergency and should be treated with dispatch by surgical therapy. We do not disagree with those surgeons who have been critical of extreme delays in patient management resulting from arteriographic studies and attempts at vasopressin infusion particularly by the medical gastroenterologic community to the point where the surgeon is the consultation of last resort resulting in poor surgical results that might otherwise have been better in salvaging these very sick patients. Gastrointestinal bleeding is a surgical emergency and deserves prompt and early surgical consultation. Management of patients by non-operative techniques including vasopressin should be by the surgeon himself or at least in conjunction with the surgeon. If early surgery promises success inordinate delays for arteriographic and infu-

sional approaches should not be taken in any given institution. Surgical judgement remains the sole decision maker in when and when not to operate on a given patient with gastrointestinal hemorrhage. It is, however, the duty of the surgeon to investigate all means by which he may decrease morbidity and mortality from his surgical procedures and to the extent that vasopressin infusions may do this. He is certainly duty bound to consider its use in judgement decisions concerning surgery in such patients.

Annual Oration for 1982

Defects in Cerebral Function and Metabolism and the Role of Revascularization

MICHAEL O'CONNER, M.D.

(Manuscript Not Available)

Annual Oration for 1983

The Artificial Heart in Human Subjects

JACK KOLFF, M.D.

Patients with end-stage cardiac disease who are not accepted for cardiac transplantation may be candidates for implantation of a total artificial heart (TAH). The design of a TAH was first undertaken by Drs. T. Akutsu and W. J. Kolff[1] in 1958. They described a pneumatically driven, polyvinyl chloride heart consisting of two collapsible sacs in one plastic air chamber. The simplicity of design and the ability to control the heart from an externally located energy and control the heart from an externally located energy and control module have made pneumatic ventricles the most successful in terms of animal longevity.[2–4]

Several groups have reported calf survival for over 6 months. These long-term experiments have resulted in complications that may well be unique to the calf. Continuous growth of the young calves causes their needs to exceed the capabilities of the implanted TAH. Calcification of the polyurethane flexing diaphragm also has been observed after prolonged survival and may be similar to the early calcification seen in valve heterografts implanted in children.[5] Because of the successful bench and animal trials with the Utah (J-7) TAH,† the Food and Drug Administration has given permission to proceed with clinical trials in selected human subjects at the University of Utah. Dr. Barney Clark was the first patient selected in that study. In preparation for these and future studies in living human subjects at Temple University, our cardiac surgical team considered it necessary to test the anatomic fit and the functional capabilities of the ventricles in brain-dead human subjects.

Methods and Materials

The Utah TAH is constructed of polyurethane and has a continuous smooth inner surface. It is pneumatically powered and consists of two separate ventricles. As in the original design by Kwan-Gett, the blood chamber of the J-7 designed by Jarvik is separated from the air chamber by a flexible diaphragm.[6] The maximum end-diastolic volume of each ventricle is 150 ml and its maximum stroke volume is 100 ml, for an ejection fraction of 67%. Two Björk-Shiley disc valves achieve unidirectional flow in each ventricle. Connections to the natural atria and great vessels are made with a "quick-connect" system. This system consists of a cloth cuff or graft with a flexible polyurethane ring which, after it is sewn onto the atrium or artery, snaps over a rigid collar onto either the inflow or outflow port of the ventricle.

The source of compressed air is controlled by electrically driven solenoid valves within the Utah Heart Driver (UHD). The UHD controls heart rate, per-

cent systole, and maximum ventricular driving pressure for each ventricle. The UHD is connected to the intrathoracic ventricles via two 6 foot long air hoses.

The artificial cardiac cycle consists of systole and diastole. During systole a pulse of air enters the air chamber and empties the blood from the juxtaposed blood chamber. During diastole the air chamber is vented to the atmosphere so that venous pressure fills the ventricle with blood. The stroke volume of each ventricle is therefore controlled by the venous pressure in its respective atrium, an arrangement which results in a balanced circulation. This principle is considered to be Starling's law of the TAH[7]. Either ventricle can be stopped by reducing the air driving pressure to zero.

Standard radionuclide ventriculographic techniques gated to the UHD were used to obtain filling rates, emptying rates, and ejection fractions of either the right or the left ventricle.

Experience in Human Subjects

From May, 1981, to December, 1982, five TAHs were implanted in five human cadavers, three of whom were kidney donors. Informed consent for organ donation and the implant studies was obtained from the relatives. The fourth and fifth implants are presented in detail.

CASE 4. A young man 5 feet, 10 inches tall and weighing 160 pounds, with an external anteroposterior chest diameter of 19.5 cm, was pronounced dead according to accepted criteria for brain death.[8] In the operating room after one kidney was taken for renal transplantation, the patient was placed on total cardiopulmonary bypass and the natural ventricles were removed along the atrioventricular groove.

Atrial quick-connect rings were sewn onto the open tricuspid and mitral anuli and long arterial grafts were sutured to the transected aorta and pulmonary artery. The left pleura was opened wide and the artificial left ventricle was placed inside the pleural cavity so that there would be sufficient room in the pericardial sac for the right ventricle.

In an atmosphere of carbon dioxide, the left and right ventricles were attached to their inflow and outflow conduits. The subject was slowly weaned from cardiopulmonary bypass and the ventricles were allowed to take over full circulatory support. After decannulation the sternum and skin were closed.

In the intensive care unit he required fluid and potassium replacement, both depleted by diabetes insipidus. He also exhibited hypoxia, acidosis, and pulmonary edema. These were controlled by positive end-expiratory pressure and by maintaining low left atrial pressures while phenylephrine was used to support the blood pressure. Eventually, these pulmonary difficulties were controlled and all pharmacologic agents except for vasopressin were stopped. Hemodynamically, the subject's condition became progressively more stable, and we were able to test the heart by varying systole from 20% to 80% and the heart rate from 60 to 180 beats/min, while measuring ejection fractions by radionuclide methods. The experiment was electively terminated 41 hours after implantation to accommodate funeral arrangements.

CASE 5. A young man 6 feet, two inches tall and weighing 145 pounds, with an external arteroposterior chest diameter of 19.5 cm, was pronounced dead but was rejected for kidney donation because of a prolonged hypotensive episode. Two J-7 ventricles were inserted to replace the natural heart, but their positioning was different from that used in Case 4. The artificial right ventricle was placed in the right pleural cavity and the left

ventricle was placed within the pericardial sac. A new right atrial outflow port was created on the lateral side of the right atrium. The tricuspid anulus was closed with tricuspid valve tissue and an anterior flap of right ventricular myocardium.

Cardiopulmonary bypass was discontinued and the TAH supported the entire circulation. Postoperative bleeding tendencies were corrected with fresh-frozen plasma and platelets. After transfer of the subject to the intensive care unit, various pneumatic, angiographic, computed axial tomographic and ventricular radionuclide studies were done to determine mechanical fit and ventricular function. The experiment was electively terminated after 72 hours to accommodate funeral arrangements.

Results

All five human subjects had a normal heart and normal-sized pericardium for height and weight. A summary of the pertinent clinical data of the five subjects is shown in Table I.

Anatomic considerations were critical for proper hemodynamic function. In Case 2, late pulmonary edema developed because of pulmonary venous compression by the artificial ventricles. For subjects with a small natural heart, as in Case 3, we separated the two artificial ventricles and placed the right in the right pleural cavity and the left in the pericardial sac. In Case 4, we placed the left artificial ventricle in the left pleural cavity and allowed the right artificial ventricle an orthotopic position in the pericardial sac. Details of the surgical implantation techniques are described in another report.[9]

The adequacy of cardiac output in Case 4 is reflected in part by the urine output. Excessive urine production caused by diabetes insipidus was controlled by vasopressin. Actual cardiac outputs of each ventricle were obtained by measuring the air flow exhausted from each ventricle during diastole. A computer calculates the volume of blood that enters the ventricle during diastole and multiplies it by the heart rate for cardiac output of each ventricle.[10] The cardiac output of the left ventricle was consistently greater than that of the right ventricle.

During studies of ventricular function in Case 5, we found that the right ventricle could be stopped from actively pumping while the subject still maintained an adequate blood pressure and a marginal cardiac output from the left ventricle. A vacuum of 10 cm H_2O on the UHD during left ventricular diastole and a central venous pressure of 20 cm H_2O were sufficient to allow blood to flow through the right ventricle and lungs. The effect of atrial contractility on pulmonary flow could not be assessed.

Standard radionuclide ventriculography techniques show ventricular filling and emptying in Fig. 8. Under a fixed set of conditions, i.e., heart rate 120 beats/min and 33% systole, the ejection fraction was 53%. The curve indicates that the heart became slightly more than two thirds filled at the end of diastole and emptied completely to its minimal residual volume of 50 ml. The ventricle thus maintained an inherent degree of response to changes in venous pressure.

Discussion

During the fifteenth century Leonardo da Vinci attempted to study the anatomic and functional interactions of man's internal organs.[11] However, da Vinci had a very meager amount of human material to study, as it was not popular to study human beings. In his drawings of the internal organs of man, he needed to

TABLE I. Summary of five brain-dead, hemodynamically stable human subjects in whom the Utah (J-7) TAH was implanted

Case No.	Age (yr)	Sex	Height	Weight (lb)	AP chest diameter (cm)	Kidney donated	Ventricular position		TAH pump time (hr)	Reason terminated
							Left	Right		
1	54	F	5′7″	132	—	Neither	Ortho.	Ortho.	2	Bleeding
2	26	M	5′10″	150	—	Left	Ortho.	Ortho.	10	Pulmonary edema
3	20	F	4′11″	93	—	Left	Ortho.	Hetero.	1½	Bleeding
4	23	M	5′10″	160	19.5	Left	Hetero.	Ortho.	41	Elective
5	19	M	6′2″	145	19.5	Neither	Ortho.	Hetero.	72	Elective

Legend: AP, Anterposterior. TAH, Total artificial heart. Ventricular position refers to whether or not the particular ventricle lies within its usual intrapericardial position. Orthotopic (Ortho.) means within and heterotopic (Hetero.) means outside the pericardial sac.

borrow from the anatomic knowledge of animal organs. He took some artistic liberties to make the heart of an ox fit inside a man's chest. He had no idea of its function, since the course of the circulation of blood had not yet been discovered. Today, five centuries later, we were confronted with the question of whether or not an artificial heart successfully tested in calves would fit and function in man. But how to proceed in man with some assurance of success? An artist's drawing or a human cadaveric fit says little about the ability of the device to function when so placed.

We propose that there is a new opportunity for human experimental studies which far exceeds the purely anatomic and comparative studies of da Vinci. Today it is possible to test the functional capabilities of intrathoracic blood pumps in brain-dead but hemodynamically stable human subjects at no risk, so that it is not necessary to learn the fundamentals of fit and function in patients. Permission to use the body or organs of brain-dead human subjects has benefited thousands of renal transplant recipients. It is in such kidney donors that the functional studies of the artificial heart were carried out. The relatives of these donors have been extremely supportive of our experiments. Their hope is that through these studies others may live longer and more comfortably.

In regard to our fit studies, the fact that the artificial heart consists of two independent pneumatic ventricles allows them to be separated completely so that either ventricle may be placed outside the pericardial sac in a heterotopic position. After encountering left atrial and pulmonary venous compression with unilateral pulmonary edema in Case 2, we did not think it advisable to place both ventricles in their usual intrapericardial positions in a small subject of 93 pounds (Case 3). Therefore, we utilized heterotopic positioning of either the left or the right ventricle in Subjects 3, 4, and 5 and proved that this is a viable surgical option.

In regard to functional studies, one of our concerns was to be able to maintain low filling pressures and adequate cardiac outputs to provide peripheral organ perfusion. It was also desirable to maintain a degree of inherent regulation so that a balanced but not necessarily equal output between the right and the left ventricles could be sustained for long periods without constant adjustments of the heart driving parameters. Although the left ventricle was expected to pump more than the right because of the bronchial circulation, a rather larger than expected difference occurred in the middle of the time course of Case 5. Additional factors that could account for this difference include a $\pm 10\%$ inaccuracy of each measurement when determined on a mock circulation and the increased regurgitation of the left-sided valves. Studies with electromagnetic flowmeters around the pulmonary artery and aorta are planned for the future. The important point, however, is that the right and left ventricles never pump equal amounts, and the TAH has to be designed to allow differences in output while maintaining physiological atrial pressures. The TAH functioned successfully for up to 72 hours.

In conclusion, these ongoing studies in brain-dead human subjects show that the Utah J-7 TAH can take over the role of the natural heart in a human being. This experimental model is unique and necessary to evaluate the fit and function of the device in preparation for future implantation into a wide selection of persons who would rather be tethered to this device then die of end-stage heart disease.

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Annual Oration for 1984

The Influence of Renal Transplantation On Biology and Medicine

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Renal transplantation has been conceived, born and nurtured by the creativity and experiments, of many biologists and clinicians.

Transplant physicians have trained themselves to be adept in biology and in research and as a result have strengthened established programs of medicine and surgery and have established new programs of transplantation. The collaboration between the laboratory and the clinic has been close in transplantation as has the relationship between science and service in taking care of patients. New and strong basic and clinical interdisciplinary working relationships have been formed to advance transplantation research, education, training, patient care and clinical practice. A number of transplant surgeons have become Chairpersons of Departments of Surgery in Medical Schools.

Before pharmacological immunosuppression was discovered in 1959, apart from occasional identical twin kidney transplants, which worked and did not reject because of tissue identity, there was not any group of patients with functioning renal allografts. Renal transplantation currently, in living related kidney donor transplants has a patient survival rate of 95% and an allograft survival rate of 90 per cent, at one year. The respective rates for cadaver renal donors for patient survival are 90 per cent and for graft survival are 80 per cent.[1] During the last quarter of a century renal transplantation and research has influenced to the better many areas of biology and medicine.

Classical and cellular immunology.[2] Some of the transplant related advances in immunology have been: The demonstration that rejection of foreign tissue graft alloantigens is an immune response, mediated by lymphocytes. The "clonal selection theory". The heterogenicity of lymphocytes. Thymus dependent T cells. Bursa of Fabricius originated B cells. Subsets of T cells: helper, suppressor and effector cells. The "network" theories of the immune response. T cell and B cell reporters. The induction of immunologic tolerance. Immunologic control and feedback mechanisms.

Autoimmunity.[3] During allograft rejection, antigens shared by donor and recipient may lead to an autoimmune type of response.

Immunogenetics.[4] The discovery of the human major histocompatibility complex (MHC) on the short arm of the sixth chromosome. The human leucocyte antigen (HLA) system: HLA-A, B, C, D and Dr MHC antigens are essential for the recognition of foreignness and for the regulation of the immune response.

The correlation of a good prognosis for transplantation of the kidney in living related donors who are closely matched by HLA histocompatibility typing.

Evolution.[5] The study of the MHC has provided important information on the evolution of the vertebrates: some of the bony fishes, some amphibians, all birds and mammals.

Mammalian reproduction.[6] Pregnancy is a natural transplantation without immunosuppression and demonstrates maternal acquired tolerance to fetal antigens, half of which are foreign paternal antigens. New research information on the relationship is being obtained in the hope of finding and applying its secrets to organ transplantation.

Immunosuppression.[7] Before 1960 whole body irradiation alone was used for immunosuppression without success. After 1961 Imuran and Prednisone were used for prophylactic immunosuppression which eventually resulted in a graft survival rate of 50 percent at one year. Antilymphocyte globulin (ALG) was added by 1965 improving graft survival rate in certain centers to 75 per cent. Antithymocyte globulin (ATG) was introduced in 1978 with effects similar to ALG. Cyclosporin was released by the Food and Drug Administration in the United States in 1983 and proved to be superior to Imuran for kidney transplants and transformed transplantation of the liver, heart and heart-lung into reliable clinical practises resulting in less rejection episodes and average graft survival rates of about 80 per cent. Rejection is currently being treated with one or more of these agents: prednisone, methylprednisolone, ALG, ATG and monoclonal antibodies. All the above means of preventing and treating rejection are immunologically non-specific methods of immunosuppression. In man, specific immunological suppression or tolerance or enhancement or chimerism are goals to be achieved by further research.

Microbiology and infectious disease.[8] Immunologically compromized patients develop opportunistic infections with cytomegalovirus, Herpes simplex virus, Herpes zoster virus, Aspergillus, Nocardia, Pneumocystis carnii and Listeria monocytogenes. Common organisms that also cause infections are E. Coli, S. aureus, D. pneumonia, hepatitis B and M. tuberculosis. The development of infectious disease specialists and the new specialty of immunopharmacology have been encouraged. Precautions are taken to prevent infections in the donor from being transplanted to the recipient.

Nephrology.[9] Nephrology and the end stage renal disease patient on hemodialysis provided the stimulus to clinical renal transplantation which improved the quality of life of these patients. Transplantation research is providing a better understanding of the pathology of end stage renal disease.

Artificial organs.[10] The most successful artificial organ is the artificial kidney developed by Kolff and others. Other artificial organs in various stages of development are the artificial heart (Akutsu and Kolff and others), the artificial pancreas (Soeldner and others), the artificial lung (Dennis and others), the artificial liver and the artificial gut (Sribner and others).

Bone marrow transplantation.[11] Bone marrow transplantation has been possible using immunosuppression for patients with aplastic anemia and acute leukemia, provided these patients have HLA-idential sibling donors.

Plastic surgery.[12] Burned patients have been treated with agents of immunosuppression to allow longer survival of skin allografts. The technology of reducing antigenic activity by freeze drying has helped develop the porcine xenograft, bovine collagen and bone. Autotransplantation of free flaps has given the stimulus to microsurgery.

Liver pathophysiology.[13] Liver transplantation has given new insights into better techniques of liver surgery, a deeper understanding of liver physiology, of the hepatotrophic concept and of several inborn errors of metabolism and their treatment: glycogen storage disease, type II homozygous hyperlipidemia, alpha$_1$-antitypsin deficiency.

Type I diabetes and the pancreas.[14,15] The impact of transplantation on diabetes has been on (a) its treatment: by (1) kidney transplantation which is successful and lifesaving in diabetic induced end stage renal disease and (2) by pancreatic transplantation which with Cyclosporine is still only partially successful, resulting in a graft survival rate of 40 per cent. (b) understanding the secondary nature of microvascular and other lesions of diabetes.

Metabolic disease.[16] Transplantation and transplantation research has provided better understanding and occasionally better treatment of certain metabolic diseases. Transplantation of the liver in alpha$_1$-antitrypsin deficiency. Transplantation of the thymus in DiGeorge's Syndrome. Bone marrow transplantation in Wiskott-Aldrich's Syndrome.

Cardiac transplantation.[17] Heart transplantation has been an acceptable treatment for end stage heart disease. Cardiac transplantation has also improved knowledge of cardiac anatomy, cardiac physiology, coronary artery disease, cardiac biopsy and brain death.

Tumors.[18] Tumor immunology and tumor immunotherapy have benefited from the establishment of the biologic laws of tissue transplantation.

Cancer.[19] Transplantation has increased our understanding of cancer in two areas: (a) the transplantation of cancer in immunosuppressed patients, (b) the de novo development of epithelial cancers and lymphomas in immunocompromized organ transplant recipient patients.

Society.[20] Transplantation has stimulated passage of brain death laws, has affected the ethics of removing a kidney from living related normal kidney donors, has born the cost of kidney transplantation, cadaver organ and tissue donation of heart, lung, liver, pancreas and other tissues such as corneas, skin and bones. Organ transplantation has restored and rehabilitated to healthy and useful living many young adults who would have otherwise died of a terminal single vital organ disease.

Organ procurement agencies. Organ procurement centers have been established to coordinate the removal, sharing and distribution of healthy organs and tissues from cadaver donors for transplantation.

Organ banks. Organ banks are being developed for storage of tissues such as skin and bones.

Organ preservation.[21,22] The preservation of organs removed from cadaver donors has been developed. Kidneys may be stored up to 48 hours in an asanguinous cold (4°C) state in Collins solution which contains intracellular sodium ions and hyperosmolar agents. Pulsatile cold perfusion is another equally good but more expensive method of kidney preservation. For heart, liver and pancreas cold storage preservation is good for only four to six hours during which period these organs have to be transplanted to resume function. Research is in progress to improve the length of storage of these latter organs.

Radio biology. Transplantation has expanded the field of radiobiology with radionuclide scanning techniques for the diagnosis of rejection and angiography for evaluation of the arterial system. Transluminal angioplasty has been successfully performed for stenosis of renal arteries following transplantation. Local kidney transplant irradiation has been practised to prevent and treat rejection, although without much success. Total lymphatic irradiation has been shown experimentally to prolong allograft survival with a reduced dosage of immunosuppression.

Pathology. The impact of transplantation on pathology in understanding the process of transplant rejection and stimulating research into the mechanisms of end stage organ disease has been valuable.

Molecular biology.[23,24,25] Transplantation research has stimulated molecular biologists to look into the MHC, lymphokines and HLA antigens to improve our understanding of these and related areas of mutual interest.

HLA and disease.[26] Transplantation research has increased our knowledge of the association between HLA and disease. Ankylosing spondylitis and HLA-B27, multiple sclerosis and DW2 and others. There is an HLA and disease registry in Copenhagen.

Transplantation of the liver, heart, heart-lung and pancreas. With the use of Cyclosporine, liver transplantation has a graft survival rate of about 75 percent, heart transplantation 80 per cent, heart-lung transplantation 70 per cent and pancreatic transplantation 40 per cent, all at one year.

Transplant surgeons and surgery. Surgeons refused to accept the current immunological dogma of the 1950's that the rejection of a foreign graft was an essential survival feature of evolution and that its control with pharmacological agents would result in ending the life of the host. Surgeons used immunosuppressive agents and discovered that by using the right dose hosts would accept foreign grafts. They discovered that many rejection episodes could be reversed with steroids and other agents. They showed that after a while the transplanted organ adapted to survive in the host provided immunosuppressive agent administration was maintained. Surgeons improved the quality of life of patients on dialysis after renal transplantation and gave a second chance of life to patients who needed a vital organ such as a heart or a liver transplant. Surgeons stimulated research into end stage organ disease in the hope that one day these diseases could be prevented in many young patients and thus eliminate the need for organ transplantation. Experimental and clinical research on immunosuppressive agents was performed and developed mostly by transplant surgeons.

Hahnemann. Renal transplantation in Philadelphia is believed to have been first performed by Robert Bower, M.D., surgeon and Albert Brest, M.D., internist, on R.W. a 25 year-old male who received a kidney from his 23 year-old brother, at Hahnemann Medical College and Hospital, on August 13, 1963.

Since 1964 this author has pursued experimental and clinical transplantation. After the lung alone or the heart and lung were autotransplanted in dogs, follow-up lung and heart-lung function were found to be near normal.[27,28] While doing experiments on the antigenic and nutritional control of antibody formation in rats, by an unexpected accident, the genetic control of antibody formation in the rat was discovered.[29,30] When mice were pretreated with irradiated tumor cells, methylcholanthrene fibrosarcoma could not be induced.[31] When dogs or rats were pretreated with solubilized donor specific antigens, the survival of renal allografts were found to be enhanced.[32,33] Using column chromatography the purified active IgG fraction was extracted from antilymphocyte serum.[34] The mechanism of prolonged renal allograft survival in dogs was analyzed.[35] Antimacrophage serum could not prolong the survival of renal allografts in rats.[36] A protein was linked to a cytotoxic agent as a model for immunologically and specifically killing malignant and allograft rejecting cells.[37,38] The conversion of an arteriovenous shunt to an arteriovenous fistula was described.[39] Acute renal allograft rejection was successfully reversed in patients with antithymocyte globulin.[40] Using Cyclosporine A in 32 patients, renal allograft survival was 75 per cent and patient survival was 94 per cent at about one year.

Summary

It has been shown that renal transplantation has had a significantly valuable impact on biology and medicine, as relating to the basic sciences of anatomy, physiology, biochemistry, pharmacology, microbiology and immunology, and pathology. As well as on the clinical sciences of medicine, surgery and other clinical specialties.

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Annual Oration for 1985

The Surgical Management of Tumors of the Liver (Abstract)

FRANCIS E. ROSATO, M.D.

Increasingly, the surgeon has become involved in the treatment of various pathological conditions of the liver. Doctor William Longmire was one of the earliest advocates of the surgical treatment of liver tumors. Since then, the armamentarium of the surgeon has expanded from simple wedge resections to extended lobectomies and even the possibility of liver transplantation.

At Thomas Jefferson University Hospital, in a six year period, 18 patients underwent major liver resections. Approximately one-fourth of these resections were for benign disease—hemangiomas, adenomas and focal nodular hyperplasia. The major concern with these lesions is the possibility of spontaneous hemorrhage and, therefore, resection is generally recommended if they are symptomatic and/or large. The remaining patients had malignant liver tumors, the majority of which were metastatic lesions from colorectal primary cancers. These latter patients largely were detected by postoperative elevation of CEA. Of patients who survived a curative liver resection for isolated metastatic lesions, 25% survived five years. Two patients in this series had the malignant carcinoid syndrome with hepatic involvement. With surgical extirpation of this tumor, these patients experienced marked improvement in their symptoms. Only a single patient in this series had primary liver cancer and survived three years; the prognosis for these patients remains poor.

The clinical presentations and surgical approach to these various hepatic diseases was reviewed. Surgical management, with its decreasing mortality and morbidity, has become an important aspect in the management of these hepatic tumors.

Annual Oration for 1986

The Role of the Endothelial Cell in Vascular Prosthesis Technology

BRUCE E. JARRELL, M.D.

History of Vascular Grafts

The replacement of natural blood vessels with prosthetic conduits is a recent innovation in medicine that has developed over the past four decades. Before it could occur, the concepts of vessel suture, repair and grafting had to be developed, with notable contributions by Carrel, Guthrie, Matas and Goyanes. The next major steps toward artificial replacement for native vessels occurred during the three most recent wars when ultimately arterial ligation was replaced by repair and native vessel replacement with autogenous vein graft. The technology developed in these embryonic periods was then applied to commonly encountered diseases of the aorta, particularly of the abdominal aortic bifurcation. Oudot first reported in 1951 the replacement of an aortic bifurcation with a homograft and this was followed closely by Dubost who successfully resected an abdominal aortic aneurysm and replaced it with a homograft. These early procedures were followed by wide application of homografts in aortic surgery. It was apparent that tissue grafts had major shortcomings, including short supply as well as long term mural degeneration and aneurysmal dilatation. The precise explanation for degeneration is unclear but it is most likely related to the method of preservation. In addition, a preserved homograft is not composed of living cells. The cells are destroyed during preservation and only the collagen scaffolding remains.

As a result of the failure of homografts as a vascular substitute, the need for an artificial vascular substitute became evident. As early as 1912 tubes made glass and different metals had been used with a low success rate. With the emergence of polymer chemistry in the 1940's, synthetic materials were also used as vessel replacements but no clinically useful graft resulted. In the early 1950's it was observed by Voorhees and associates that a silk strand placed within the blood stream in the right ventricle of a dog could become coated with a smooth thrombus-free glistering surface. This observation was followed by the concept that fibrin plugs could close the interstitial holes in a porous graft allowing a nonpermeable conduit to result. Cloth made of a nylon derived material, Vinyon N, was then fashioned into a tubular configuration and implanted into dogs in 1953 and used clinically in patients shortly thereafter. These tubes were functionally acceptable and useful for many large vessel applications.

In spite of this major advance, there remained many practical inadequacies with these tubes. First, they had a tendency to unravel at the ends. This was

improved by formic acid treatment resulting in stabilization of the nylon mesh. Secondly, machines had to be developed to weave the material into tubular formations rather than surgically suturing a flat cloth into a tubular shape. Lastly, the tubes had a strong tendency to kink, a problem resolved by using the novel approach of crimping. As vascular surgeons in the 1980's, we accept these graft properties as "self-evident", but their introduction into clinical surgery was a great step forward.

Current Status of Vascular Grafts

The use of synthetic vascular grafts as conduits for bypassing arterial occlusive lesions has increased significantly over the past several decades. Progress has in part been based on our increased ability to diagnose vascular disease at an earlier stage and to intervene with grafting prior to irreversible tissue ischemia. Vascular procedures are not always safe for a patient. Surgeons have learned over the years to compare the potential benefits of a vascular graft with the possible complications. This is a critical one where the potential benefit of salvaging a limb or revascularizing an ischemic heart must be compared to the risk posed to a patient's life by the procedure itself. In most cases, the decision is based on the overall vital organ status of the patient as well as the proven efficacy of the bypass graft intended for use.

Tubular dacron grafts have become firmly established as the standard prosthesis for large vessel replacement. Although there have been minor modifications in conformation, cloth construction, porosity and other variables, the dacron graft has repeatedly demonstrated its durability and long term patency. When used in the abdominal aorta, the early patency rate exceeds 95% and late patency rate at 10 years is over 70%. This is remarkable in that a patient with an abdominal aortic aneurysm or severe aortic occlusive disease can expect a virtual "cure" of that disease with a dacron graft. In fact, the usual cause of death following aortic grafting is acute myocardial infarction, suggesting the major obstacle to long term survival remains occlusive disease of the smaller arteries.

While dacron grafts have been extremely successful for the replacement of large diameter arteries, the results with smaller artery replacement are less promising. When dacron grafts smaller than 6 mm in diameter have been used in positions such as the femoral popliteal regions, poor results have been obtained. At one year, patencies as high as 70% have been reported, but the majority of series demonstrate 20 to 30% one year patency. Most surgeons have abandoned the use of dacron in that position as a result of this poor experience.

More recently, the expanded, reinforced polytetrafluoroethylene (PTFE) grafts have been introduced into clinical surgery. These grafts are composed of PTFE that has been extruded into a tubular shape. PTFE in this form is highly porous but because of the hydrophobic surface qualities, relatively impermeable to liquids. PTFE grafts have been used in many locations with variable results. In general, they perform well for large vessel replacement when used above a diameter of 6 mm. These applications have included aortic and femoral popliteal grafting.

When used above the knee with good vascular runoff, the patency has been

excellent. This ranges from 75 to 90% 1 year patency, which is equivalent to the saphenous vein graft results. When used below the knee, the patency is determined primarily by the vascular runoff. At the infrapopliteal region, patency rates at one year range from 40 to 80%. When extended to the tibial vessels, results drop significantly with patency rates at one year in the 20 to 40% range.

One PTFE application that has achieved widespread acceptance has been with hemodialysis vascular access. These PTFE grafts are typically 4–5 mm in diameter at the arterial anastomosis and rapidly enlarge to 6 to 8 mm in diameter at the venous end. They have a high flow rate, often exceeding 1000 milliliters per minute, because they are placed as an arteriovenous fistula. The patency has been excellent over the short term, but they frequently need declotting, revision or replacement on a yearly basis.

A third class of vascular graft is the biologically derived conduits. The most significant is the glutaraldehyde-stabilized human umbilical cord vein graft developed in the 1970's. This graft is prepared by isolating the umbilical vein from the umbilical cord followed by tanning with glutaraldehyde to crosslink the collagen molecules. Tanning increases the mural tensile strength as well as renders the vessel non-antigenic. These grafts function primarily as a cross-linked collagen tube and do not re-endothelialize.

Results obtained by Dardik and associates have demonstrated patency rates equivalent to that obtained using saphenous vein as the bypass graft, with 1 year patencies of 80%. Other investigators have observed a considerably lower patency rate when used in the infrapopliteal and tibial vessels. The graft has not gained widespread use for these reasons as well as the fact that it is difficult to surgically manipulate.

There are numerous other grafts available on the market, but no grafts have currently out-performed the previously discussed grafts. In order to become accepted, they will need to perform better than current prosthetic grafts and in an equivalent fashion to human saphenous vein grafts.

Biological Response to Graft Implantation

The biological response to a prosthetic material is a function of the material implanted, the method of evaluation and, most importantly, the animal species in which it is evaluated. Porous prosthetic grafts implanted into *research* animals clearly demonstrate the spontaneous development of an endothelial cell lining supported by a substrate of smooth muscle cells, fibroblasts, and other cells as well as collagen and other connective tissue components. There appears to be a strong cellular response to the graft in the form of a foreign body reaction as well as a neovascularization which is observed as capillary ingrowth into the graft.

Porous grafts implanted into *humans* result in a significantly different response. An endothelial lining of the entire graft does not occur and a greatly suppressed cellular response occurs when compared to the experimental animal model. Capillary ingrowth does not occur to a significant degree. The human response to a graft also varies as a function of the graft base polymer. Dacron grafts, for example, demonstrate foreign body grant cells within the graft wall whereas PTFE grafts demonstrate a very sparse cellularity with a marked in-

crease in adhesive protein content including fibronectin and fibrinogen. Understanding the species- and polymer-dependence of these biological responses is complex and poorly understood. Considerable effort has been concentrated upon understanding the initial events that occur upon a prosthetic surface. Areas of significant interest include protein deposition upon the prosthetic surface, blood responses to prosthetic surfaces and spontaneous mechanisms of endothelialization.

The dynamics of protein deposition have been examined extensively by many investigators. It is a very complex process characterized by sequential deposition and release of proteins including albumin, fibrinogen, immunoglobulins and other major proteins. These responses are concentration and time dependent and probably represent the earliest response to the graft. Protein deposition is a very significant area because it is these proteins that the blood stream ultimately "sees" initially. Two disparate responses to the protein layer may occur. One response is activation of platelets and coagulation proteins. This develops into a surface coating of platelets, red blood cells and a fibrin meshwork and can result in a very thrombogenic surface with thrombosis of the graft. The other response is the deposition of a protein layer that "passivates" surfaces, resulting in a very low activation of thrombotic mechanisms. This process is poorly understood but is capable of generating a very non-thrombogenic surface in some models. Most clinical grafts develop a variant of the first type in that a thrombogenic surface is present but not reactive enough to thrombose when used in large diameter locations.

Additional blood responses to a surface are also important variables in graft surface events. Dacron, for example, is well known to activate the complement system when compared to PTFE. This may result in activation of the coagulation cascade as well as act as a migratory stimulant to leukocytes. Leukocytes attach to the surface and stimulate an inflammatory response, which also contributes to thrombus formation. These mechanisms are extremely complex but capable of resulting in graft thrombosis and failure.

The third important aspect of the biological response to grafts is the interaction between endothelial cells and the prosthetic surface. Multiple *in-vivo* mechanisms exist which would allow a spontaneous endothelial mono-layer to form on an implanted vascular surface. All involve migration of nearby endothelial cells onto or into the graft. The first way, termed pannus ingrowth, has been repeatedly observed in both non-human and human species. With this mechanism, endothelial cells on the surface of the native vessel migrate slowly across the suture line and attach and grow to the prosthetic surface. One important aspect of pannus formation is pannus arrest. Once the pannus advances to a certain distance, the endothelial cells cease to continue migration.

The rate of pannus ingrowth shows some variability between animal species. When porous vascular grafts are implanted into mammals, pannus formation begins at each anastomotic site. In laboratory animals, this process proceeds at a variable rate. Dogs are able to cover approximately 1 cm of each end of the graft by 1 year while baboons can cover a larger surface (up to 5 cm) in a shorter period of time. In human studies, only about 1 cm is covered and this requires

one to two years to become complete. Thus, although this mechanism of graft endothelialization has appealing aspects, its major drawbacks are its incompleteness and the amount of time required.

A second method of graft endothelialization is transinterstitial ingrowth of microvessel endothelial cells through the graft and onto the luminal surface. Certain non-human species are capable of endothelializing porous dacron grafts in the central portion of the graft well before pannus ingrowth could account for coverage in that area. Sauvage proposed that central graft endothelialization occurred because of transinterstitial ingrowth of micro-vessel endothelial cells. Central graft endothelialization could be prevented in his studies by wrapping the porous graft with an impervious material. Additional studies by Clowes and co-workers revealed that a PTFE graft of large pore size (60 microns diameter) allowed microvessel ingrowth in baboons while standard grafts composed of 30 micron pores prevented ingrowth. Dye studies demonstrated continuity between external, intramural and luminal endothelial cells. These studies are compatible with the concept that microvessel endothelial cells are capable of not only spontaneously colonizing a surface but also performing some functions of large vessel endothelial cells. This includes the formation of a cobblestone monolayer that appears anti-thrombogenic with the ability to withstand arterial wall shear stresses.

This process has not been observed to occur in grafts implanted into humans. Anderson and associates have examined grafts removed from humans and compared them histologically to grafts implanted into animals. They examined the midportion of the graft, therefore excluding the areas of pannus ingrowth. An absence of endothelial cells was observed in the human grafts. Absence of endothelial cells in the midportion of grafts implanted into humans appears to be the case in most series that have been reported. This is unique to the human species in that multiple studies have demonstrated spontaneous islands of endothelial cells within the mid-portion of porous grafts implanted into dogs, pigs, rats and baboons. This disparity in healing response suggests that a species specific difference in microvessel endothelial cell response to prosthetic grafts may be present.

A third potential method of graft endothelialization is migration of a pluripotential blood-borne cell from a distal site to the graft surface where it attaches and subsequently covers the surface. Several investigators have suggested that fibroblasts, monocytes or other cells are capable of transforming into cells with endothelial-like characteristics. Since both vascular endothelial cells and blood cells are thought to originate from hemangioblasts of mesodermal origin, this mechanism has a scientific basis. It has been recently observed that trauma to upper extremity veins may dislodge endothelial cells from the luminal surface. Once dislodged, they are potentially free to migrate throughout the blood stream and subsequently attach to a receptive surface. Thus this represents another mechanism whereby a blood-borne endothelial cell could attach to a graft and initiate surface endothelialization. Unfortunately all blood-borne mechanisms of graft endothelialization seem improbable, requiring more convincing evidence before widespread acceptance will occur.

Mechanisms of Graft Failure

The success of a given vascular reconstruction is largely dependent upon the anatomic location of the graft, type of graft implanted and the flowrate through the graft. In general, high flowrate, large diameter prosthetic grafts perform well in most patients regardless of the type of material used. When a prosthetic graft is placed in a less favorable location such as where flow rates are lower, the type of material used for the bypass becomes critical. Autogenous grafts in this setting continue to provide satisfactory long term patency. Synthetic grafts in low flow locations tend to occlude at a high rate due to their inherent thrombogenicity.

General observations regarding graft failure may be categorized based on their probable origin. The most preventable cause of failure relates to technical aspects of graft insertion. There are many nuances to vascular surgery that have been developed to prevent stenosis at the anastomosis, graft kinking and other mechanical factors. These are avoided by the surgeon trained in vascular surgery.

A second cause of failure is patient-related problems. Blood flow rate through the graft is an important factor. If a patient has a low flow state secondary to congestive heart failure or extensive multiple vascular occlusions from atherosclerosis, the patency rate will be reduced. The parameters that correlate with decreased patency are: small diameter (<6 mm), long graft length, reduced blood flow into the graft and reduced blood outflow due to poor runoff. This patency does not correlate simply with blood flow rate. The blood rheology at the blood-surface interface has been demonstrated to be of importance. This is certainly highlighted by the difference in patency between aortic and inferior vena caval (IVC) grafts. The bulk flow rate is roughly equivalent but aortic grafts have an extremely high patency whereas IVC grafts uniformly thrombose. The blood surface velocity and shear rate are much lower in the IVC due to a lack of pulsatility and other flow effects, suggesting their importance. Areas of flow turbulence have also been associated with graft failure.

The graft surface itself is a third important variable. As previously mentioned, all prosthetic surfaces activate some aspects of the thrombotic process. These include platelet deposition and activation, activation of coagulation proteins and complement activation. There are many *in-vitro* and *in-vivo* tests to allow comparisons between surfaces, but no one test reliably predicts surface behavior when implanted into an experimental animal. Additionally, no laboratory animal results predict results in human implants. This is a major shortcoming for research in this field and unfortunately translates into human trials to properly evaluate surfaces.

Cellular hyperplasia at the site of graft-native vessel anastomosis is the last major cause of graft occlusion. This is perhaps most significant in small diameter occlusions but certainly occurs in all applications. This has been repeatedly observed in both animals and humans. Luminal stenosis is usually observed for 1 to 2 cm of length on the native vessel immediately adjacent to the graft. This occurs at both ends of the graft but is usually most prominent at the distal or outflow aspect. The surface is often a white glistening material and is frequently covered

by endothelium. The subendothelium is composed of heavy cellular material with dense connective tissue infiltration. Many of these cells are smooth muscle cells (SMC) and myofibroblasts as determined by immunocytochemical staining, but other cells including fibroblasts and macrophages are present. This hyperplasia results in progressive stenosis and occlusion of the lumen over months to years.

Many investigators have examined this problem but few answers have been forthcoming. Clowes and coworkers have noted that many cellular events occur as soon as the vessel is sutured and thereby injured. Smooth muscle cells undergo a burst of mitosis as well as migration within 6 hours following injury. The cells undergo several multiplications over the next several days but then demonstrate no further mitosis. The area undergoes further rearrangement and collagen deposition. These later events continue over months to years and are undoubted implicated in anastomotic stenosis. There have been multiple experimental attempts to prevent the occurrence of stenosis. The most successful intervention has been using the drug heparin. If heparin or fragments of the heparin molecule are given within 18 hours following injury, the burst of mitosis does not occur. Longer term studies suggest that this may result in less cellular hyperplasia when compared to control experiments.

Other explanations for anastomotic hyperplasia have also been suggested. It was noted many years ago that insertion of a rigid tube into the pig aorta resulted in marked anastomotic hyperplasia. This suggested that compliance mismatch between the graft and the native vessel played a significant role in cellular hyperplasia. This has been extensively evaluated by Abbott and coworkers with no definite conclusion as to cause and effect. A problem in interpretation exists in any event because of the known decrease in compliance with increasing atherosclerosis in arteries. Thus further information is necessary.

Rationale for Endothelialization of Prosthetic Surfaces

While many differences exist between thrombogenic polymer surfaces and a native blood vessel surface, one obvious and important difference is the presence of a living endothelial cell lining on the native vessel luminal surface. As a direct result of our ability to culture human adult endothelial cells, a new understanding of the integral role of endothelium in blood vessel homeostasis has evolved. Although these cells form only a single monolayer separating the blood from body tissues, they are not passive cells. Rather, they are metabolically active and interact with blood and tissues to control many different processes. Certain endothelial cell functions are integral to the maintenance of a smooth anti-thrombogenic surface. They demonstrate anticoagulant properties in their native state to prevent blood coagulation, yet when injured or in certain pathologic states they change to a pro-coagulant state, resulting in blood coagulation. The endothelial cell has been shown to be an active participant in the transport of materials between blood and tissue. Numerous investigators have demonstrated endothelial cell participation in the immune response. They play a role in lipid metabolism and may be a major participant in the development of atherosclero-

sis. They have become the focus of many investigators because of their diverse differentiated functions.

It is sobering to realize the multitude of endothelial cell functions identified during just the last 10 years and the failure of an intensive polymer surface research effort over the past 30 years to even closely approximate one cellular function, namely the anti-coagulant activity. The shear number of different endothelial cell functions raises a question as to whether a passive polymer surface will ever be developed that will successfully reproduce endothelial anticoagulant function. It is not enough to prevent platelet and fibrin deposition on a surface as the only important characteristic of a prosthetic surface. The surface must also prevent leukocyte and macrophage attachment, bacterial attachment, complement activation and should be able to maintain those qualities indefinitely. As we learn more about polymer characteristics necessary for long-term patency, a major difficulty persists with respect to the ability to effectively evaluate a surface even if identified. Specifically, there is no good animal model to predict blood-surface interactions. Thus potential human applications must be extrapolated from data derived in less than ideal animal models.

An alternative approach to creating an artificial, non-thrombogenic surface is to develop a graft amenable to the creation of an endothelial cell lining. While basic research has defined mechanisms which help explain the anti-thrombogenic nature of the endothelium, clinical studies have established the importance of an intact endothelial lining for the maintenance of normal blood vessel function. The high success rate with saphenous vein or internal mammary artery grafts indicates the superiority of a native "living" vessel. Additional studies have suggested that long term patency may be related to the preservation of the endothelium in the saphenous vein after removal. Several investigators have shown that poor surgical technique, pressure distention of the vein, and vasospasm may adversely affect endothelial integrity. In addition, a cold preservation medium or anticoagulated blood within the graft while it is awaiting implantation probably affords better endothelial preservation than room temperature saline solution. These pieces of evidence suggest that cellular integrity within the vessel wall is important for subsequent normal vascular wall function. "Cellular integrity" is not necessarily limited to the endothelial cell monolayer. Normal function of the smooth muscle cells within the vessel wall may play an important regulatory role in endothelial cell function. Thus, preservation of all the cellular and extracellular elements of the vessel wall may be the most important end product of proper vessel handling and preservation.

A second observation about the response of the native vein when placed in an arterial position is vessel wall hypertrophy in response to arterial pressure. This may allow the thin venous wall to better accommodate to the higher mural stresses encountered in an equivalent size artery. Regardless of the mechanism of hypertrophy, this response implies a complex vessel wall structure/function relationship. The net effect of hypertrophy upon endothelial cell function has not been examined, but is undoubtedly fertile ground for study.

Another important aspect of native vessels is their innate ability to repair injuries. Endothelial cells are frequently injured during surgery due to local

trauma and preservation injury. When injury occurs, the cell may lose its anticoagulant function, resulting in either thrombin activation on the injured cell or cell slough with subsequent exposure of the subendothelial surface. When this occurs, nearby cells are able to migrate into the region and reendothelialize the surface. This ability to repair cellular defects in saphenous veins used for bypass grafts has been documented in humans and laboratory animals. Regeneration of an endothelial monolayer may be more rapid when the "in-situ" method of grafting is used when compared to the standard excised and reversed saphenous vein graft. In the "in-situ" procedure, the vein is handled less and this may contribute to the improved long term patency. When large areas of vessel surface have been denuded, the migratory ability of nearby endothelium may be inadequate to reendothelialization the surface. This occurs in long endarterectomy sites and may explain the discontinuance of endarterectomy procedures in locations of lower flow rate such as occurs in the superficial femoral artery.

Lastly, the composition and structural integrity of the collagen lattice which underlies and surrounds the cellular components of the vessel wall has been shown to influence endothelial cell function and morphology. Since collagen synthesis and structure may be regulated by such factors as shear rate and synthetic polymer structure, further work must be done to determine the role of collagen in native graft function. Interactions between collagen and vessel wall cells could be important in regulating numerous vessel wall functions.

Methods to Induce Graft Endothelialization

A. Endothelial Cell Seeding. One mechanism that can expedite graft endothelialization is the process of "seeding" the graft. This process involves the procurement of endothelial cells isolated from either an autologous large vessel or microvessel source and placement of these cells upon the graft surface allowing adherence to occur. The early seeding studies by Herring and co-workers utilized the isolation of endothelial cells from a vein by mechanical scraping. These endothelial cell were then mixed with blood and passed through the interstices of the graft. Eight to twelve weeks following implantation into dogs, the grafts demonstrated an increased degree of endothelial cell coverage compared to control grafts. This finding has been repeatedly demonstrated in animal models and even recently reported in one human graft.

There are a number of uncertainties which persist in canine seeding studies, including the observation that spontaneous endothelialization occurs in the canine model even in the absence of seeding procedures. In addition, the origin of endothelial cells ultimately forming the monolayer was originally assumed to arise from the seeded endothelial cells. This assumption has recently been challenged by Hollier and associates who observed that porcine endothelial cells seeded onto grafts implanted into dogs yielded results similar to autologous seeding. This study suggests that an endothelial cell-derived factor rather than the cells themselves may be responsible for the more rapid endothelialization. This line of investigation should be continued in that it may yield information useful for stimulating transinterstitial EC ingrowth.

B. Endothelial Cell Sodding. A second method of graft seeding involves placing a large number of endothelial cells directly upon the graft surface rather than mixing the cells within a clot and allowing the mixture to jell upon the graft surface. The principle advantage of this method is that it allows a monolayer to form very rapidly. A mature monolayer may be stimulated to form in less than one hour when a sufficient quantity of endothelial cells is seeded upon a surface that is receptive to endothelial cell attachment and spreading. An immediate monolayer opens the possibility that a surface could be actively antithrombogenic at the time of blood flow restoration through the graft. Many variables affecting monolayer formation must be examined including endothelial cell source, incubation parameters, monolayer stability in the presence of shear forces and factors that influence endothelial cell anti-coagulant activity. But, once characterized, immediate monolayers offer prosthetic surface applications in areas where current surgical techniques fail. These include not only small diameter low flow arterial grafts, but also venous grafts, other intravascular prostheses and artificial organs.

Endothelial Cell Biology and Surgery

Endothelial cells from animals sources have been studied in culture since the 1920's. In 1973 Jaffe et al. successfully cultured endothelial cells from human umbilical veins and these cells have been extensively characterized functionally. These cell lines demonstrated growth potential, but the total number of cells produced from a single umbilical vein was usually quite limited to the range of a 10- to 100-fold increase in harvested endothelial cells. Maciag et al. subsequently demonstrated that the addition of endothelial cell growth factor (ECGF), a crude preparation of bovine hypothalamus, to the culture medium resulted in an up to 10^{10}-fold increase in the number of cells produced using human umbilical vein endothelial cells. When applied to human adult endothelial cells, the endothelial cell proliferation unfortunately remained severely limited. The phase of rapid cell proliferation was brief and early senescence of the cells occurred. In our own earlier studies, attempts to serially passage adult endothelial cells were uniformly unsuccessful. Johnson was successful in establishing HAEC from pulmonary arteries and veins, but again only short-term studies were possible. Glassberg and associates were successful in growing human iliac artery endothelial cells, but no cultures survived past 12 passages. In that study, only 50 to 500 viable cells were obtained per 5-inch vessel segment, indicating a very low yield. Fry and associates were also successful in culturing adult endothelial cells from abdominal arteries removed at the time of cadaver donor nephrectomy, but these cells also demonstrated early senescence with a maximum of five population doublings.

In 1983, Levine and associates at the Wistar Institute observed that the combination of ECGF with heparin greatly increased the *in-vitro* lifespan of cultured human umbilical vein endothelial cells. This effect was quite remarkable in that the cells had a doubling time of 24 hours and were capable of growing over 60 cumulative population doublings. In collaboration with Levine, we applied this technique to human adult large vessel endothelial cells. Vessels that

were ordinarily discarded during the operative procedure of cadaver donor nephrectomy were obtained. The vessels were preserved in culture medium at 4°C for up to 3 days prior to cell isolation. The vessels were removed and the luminal surface incubated with a crude bacterial collagenase for 30 minutes. The detached cells were collected by flushing the vessel luminal surface with medium, centrifugation and seeding into a 25 cm^2 tissue culture flask precoated with 1% gelatin. These cultures were then rewashed after 30 minutes and resuspended in fresh culture medium (M199) containing 20% fetal calf serum, 90 ug/ml porcine heparin and ECGF. Cultures were incubated at 37°C in a humidified 5% CO_2 atmosphere. Subcultivation was performed by brief trypsinization and cell counting performed at each split with a Coulter Counter.

Endothelial cell identity must always be confirmed in any isolation before conclusions regarding cell behavior can be reached. The two most convincing pieces of information are seen in the next two figures. These cells routinely assume a cobblestone morphology that is contact-inhibited. In addition, indirect immunofluorescence with rabbit anti-human factor VIII-related antigen and goat anti-rabbit IgG-FITC demonstrate intense punctate fluorescence. Further studies have documented production of angiotensin-I-converting enzyme, prostacyclin and plasminogen activator.

Endothelial Cell - Prosthetic Surface Interactions

The ability to culture endothelial cells long term allowed us to examine the growth and adherence of human endothelial cells upon prosthetic graft material. Experiments were designed to examine endothelial cell adherence to different surfaces using various techniques including morphological examination and radiotracer assays. We have developed several quantitative assays for the morphological evaluation of cellular adherence. In the first, cells are placed on the graft surface at a specific density and permitted to interact for an appropriate period of time. Non-adherent cells are removed by washing the surface and both adherent and non-adherent cells quantified. The non-adherent cells are counted using a Coulter counter. The adherent cells are washed with buffer, stained with Gill's hematoxylin and quantified by computer assisted grain counting or examined by electron microscopy. A second procedure utilizes EC labelled with Indium^{111} oxine. Radiolabelled EC are permitted to associate with graft surfaces for appropriate times, the surfaces are washed and the number of cells in both the adherent fraction and the non-adherent fraction are evaluated by gamma counting. The radiotracer method is more easily performed and allows more rapid assessment of cell adherence. In addition, multiple time points and thus the kinetics of attachment can be more readily examined.

Using the second method, radiolabelled EC at a density of 10^5 cells per cm^2 were allowed to incubate with human plasma coated woven dacron for 1 to 120 minutes. At appropriate times, the incubation was halted, the surfaces washed vigorously with phosphate buffered saline expelled from a pipette, and adherent EC counted using a gamma counter. Firmly adherent cells are observed within 10 minutes and adherent cell numbers reached 2.5×10^4 EC/cm^2 within 1 hour, and 3.3×10^4 EC/cm^2 by 2 hours. Since the original seeding density was 10^5 EC/

cm^2 (ie. a large vessel confluent density), approximately one-third of the cells originally added had firmly adhered by 2 hours.

While the best surface for EC adherence is not known, we have used a plasma coating of grafts since this protein solution is easily prepared. Certainly plasma coated dacron may not be the ideal surface for EC adherence. To examine different surfaces, we performed a similar experiment using cultured large vessel EC (10^5 EC/cm^2) and various matrix surfaces. The adherence kinetics for EC on plain dacron, plasma coated dacron and human amnion coated dacron were examined. Human amnion contains a thin layer of Type IV collagen on one surface and Type I/III collagen on the other. It may be attached to collagen I/III impregnated woven dacron using glutaraldehyde, allowing the Type IV collagen surface to be exposed for the subsequent seeding experiments. This surface was chosen because it closely resembles native vessel basement membrane. Adherence was most rapid to plasma coated dacron, but by 1 hour equivalent adherence had taken place on the amnion coated dacron. Much slower adherence was seen on untreated dacron. When these surfaces were examined by scanning electron microscopy, excellent cell interaction with EC confluence was observed on amnion coated dacron whereas poorer cell attachment and little cell to cell interaction was observed on plasma coated dacron. Untreated dacron showed essentially no cell spreading after 2 hours. Thus for cultured large vessel EC, amnion coated dacron allowed somewhat slower surface attachment than with plasma coated dacron, but the nature of the attachment was superior, allowing a confluent monolayer to form rapidly.

Scanning (SEM) and transmission (TEM) electron microscopy have been used in many laboratories for examining endothelial cell attachment to on prosthetic surfaces. It allows one to examine the surface topography of cells to determine extent of cell spreading, cell-cell interaction and the degree of surface coverage. An endothelial cell monolayer on a prosthetic surface that looks morphologically similar to native vessel endothelium is desirable and more likely to function physiologically when exposed to the blood elements. Thus we have used electron microscopy as an important method of evaluation of the interaction of these cells with the surface. There are several limitations to this technique including the time, expense and effort that goes into sampling a large number of specimens. In addition, the process of fixation obviously kills the cells, allowing only static image studies to be performed. In spite of these limitations, careful use of this technique has allowed us to observe cell association with the surface. In a timed sequence we studied cell association with the amnion-coated dacron surface. The cells initially contacted the surface following a one minute incubation. They retained their spherical morphology typical of cells in suspension. The TEM at this time point revealed a close association but not actual contact with the surface. This association probably represents electrostatic attractive forces in an equilibrium position in that the cells cannot be removed by washing. Five to 20 minutes later, the cells demonstrate a spreading morphology on SEM. TEM reveals that the earliest step is the presence of a cell foot process that extends down to the surface. This process becomes more extensive within minutes and is associated with cellular flattening and cytoplasmic attenuation. The micrographs at 30 and 60 minutes demonstrate marked cell spreading and significant association with neighboring cells. TEM confirms this observation. Perinuclear adherence to the surface is most developed and cytoplasmic adherence is beginning. Adjacent cells demonstrate interdigitation between cellular membrane projections that effectively isolate the underlying surface from the liquid phase. Later time periods are accompanied by maturation of cell to cell junctions and cellular spreading.

One difficulty with electron microscopy is the necessity of permanent cell fixation before visualization is possible. This prevents real-time, or intravital, observation of the actual process of cell spreading and other cellular processes. Traditionally, this has been evaluated by inverted phase microscopy. Unfortunately, endothelial cell adherent to dacron or PTFE surfaces are not visible with light microscopy because of the translucency of the polymers. In addition, these surfaces autofluoresce at excitation wave-lengths of 300 to 450 nm, thus interfering with cell visualization using many of the standard fluorescent labels. To circumvent this difficulty in our laboratory, we have examined different fluorescent dyes and found that Rhodamine 123 possesses qualities that are suited for endothelial cell visualization on graft material. This compound is a cytoplasmic dye that concentrates in mitochrondria, and has excitation and emission maxima of 510 nm and 550 nm respectively. Endothelial cells labelled with Rhodamine 123 demonstrated a growth curve and adherence kinetics that are indistinguishable from unlabelled cells. This dye does not stain PTFE or dacron and thus allows cytoplasmic visualization of the cell in the living state. Endothelial cells treated with Rhodamine 123 incubated with plasma coated dacron for 90 minutes appear round and do not seem to be in close contact with neighboring cells. In contrast, fluorescent cells associated with a different protein matrix surface appear markedly spread and are in close proximity with nearby cells. These staining properties might allow one to observe the actual process of monolayer formation during static conditions and the subsequent behavior of the cells after exposure to flow.

Difficulties with Cell Culture as a Method of Graft Endothelialization

The kinetics of EC association with a receptive surface such as amnion suggest that an endothelial monolayer could be generated very rapidly. If this were possible within 30 to 90 minutes, it might even be possible to generate a monolayer at the same time as a graft operation was actually being performed. This could allow a monolayer to be present when blood flow through the graft was restored. In order to accomplish this, large numbers of autologous endothelial cells would be required. It is doubtful that these cells could be obtained from a native donor vessel such as the patient's saphenous vein. Assume that 10^5 cells/cm^2 would be required for a confluent monolayer to be re-established on a prosthetic graft. The usual femoral-popliteal bypass graft may contain a luminal surface area between 150 to 200 cm^2. Thus 1.5 to 2×10^7 cells might be necessary to endothelialize the graft. Since the usual yield of cells from a saphenous vein is only 5 to 20% of the actual cells present, the required total surface area of vein would greatly exceed the surface area of the graft. This is obviously an unobtain-

able goal, particularly in peripheral vascular patients where veins are always at a premium.

One solution of this dilemma is the generation of large numbers of cultured EC. If the cell doubling time is 24 hours, more than enough cells could be grown in 2 weeks to endothelialize a graft. The question then becomes whether these cultured cells maintain the necessary requirements for adhesion and anti-thrombogenicity when placed upon a prosthetic surface. The science of cell culture has examined one aspect of this question in some detail. Most cell culture work is performed on tissue culture polysytrene, often without any protein coating such as a gelatin. It has been repeatedly demonstrated that as cells are maintained in culture, they undergo *in-vitro* senescence. This means that with increasing *in-vitro* age, they lose the ability to grow, produce products and remain viable. For endothelial cells, it has been noted that angiotensin-I-converting enzyme production decreases as *in-vitro* age increases. This data would suggest that we must examine the effects of *in-vitro* age on cells if we propose to use these cells for subsequent graft endothelialization.

We chose to initially examine the effect of *in-vitro* age on human adult endothelial cell adherence and growth to dacron. Both arterial and venous endothelial cells were isolated and grown in culture. They were then divided into a low *in-vitro* age group (low passage) and a high *in-vitro* age group (high passage). We observed that the high passage cells adhered poorly to dacron and failed to grow. The cell number actually decreased significantly with time. The low passage cells adhered much more efficiently and maintained cell number. If the dacron was precoated with a protein substrate such as collagen or human plasma, the adherence and growth improvement was significant. This suggested to us that the use of cell culture to increase cell number prior to graft endothelialization was possible if the number of passages was kept low. It also demonstrated a principle that we have repeatedly noted. Dacron is not a receptive surface for cell adherence. Even when precoated with proteins, the cells do not behave normally. Although not presented here, the same has been true for PTFE.

A second effect of cell culture that we felt should be investigated was the effect on cellular genetic material. This was an issue because it would definitely be undesirable to place cultured cells into a human if the cells in any way exhibited genetic alteration. Dr. Warren Nichols, a member of our program project group, began to examine the chromosome composition of our cultured human endothelial cells. This was performed with the classical chromosome isolation method of Giemsa. Normally, one would expect a diploid cell with 46 chromosomes. This was found for cells that were freshly isolated. *In-vitro* aging produced a progressive marked chromosomal abnormality. Umbilical vein endothelial cells had developed greater than 90% polyploidy by the time that 25% of their *in-vitro* lifespan had elapsed. Polyploidy means that multiple copies of a particular chromosome are present and suggests a defect in cellular replication and metabolism. Human adult large vessel endothelial cells developed polyploidy at a much slower rate, with an incidence of approximately 10% by the first quarter of their *in-vitro* lifespan. Equally distressing was the observation that multiple types of chromosomal abnormalities occurred. These ranged from

translocations and gene omissions to transformation into chromosomal patterns associated with tumors. This data is very important in its relationship to our studies. If we are going to use cultured cells for vascular grafts, we must be very critical of the quality of cell that we are implanting.

It has also been proposed by other investigators that a "bank" of non-immunologically active endothelial cells might be established. This is based on the hope that serial passage in culture would cause the cell to de-differentiate and lose immune markers and function. It has been demonstrated repeatedly that human endothelial cells possess blood group antigens, human leukocyte histocompatibility antigens (HLA A, B, & C) and can be stimulated to express HLA-D locus antigens when exposed to gamma interferon and other immune stimulants. We have examined whether these immune markers change with *in-vitro* cell age. Low and high passage cells have been stimulated with gamma interferon and the appearance of HLA-D locus antigens has been measured using fluorescence activated cell sorting. We have found that the dose-response curve and the antigen concentration versus time curve is identical and independent of *in-vitro* age. This confirms to us that it is unlikely that cell culture will render these cells non-antigenic.

Microvessel Endothelial Cells

Large vessel endothelial cells seem to be unsuitable for vascular graft endothelialization either because of their unavailability or because of artifacts introduced by cell culture techniques. To obtain large numbers of endothelial cells, therefore, alternate sources need to be investigated. One obvious alternate source is the microcirculation. The microcirculation consists of arterioles, venules and capillary endothelial cells. These cells interface with the blood borne elements and possess the antithrombogenic characteristics necessary for a vascular conduit. Microvessels are well known to have wide variations in blood flow through a particular region. At one period of time, the microvessel may have no flow because of low metabolic needs of the regional tissues whereas other times there may be very high flow such as during exercise. To be able to retain their anticoagulant function during periods of no flow suggests that they possess a high degree of antithrombogenic potential. Microvessel endothelial cells possess many of the other characteristics of large vessel endothelial cells including expression of factor VIII related antigen, angiotension-I-converting enzyme and prostacyclin production. Transport of solutes across capillary endothelial cells is a well described and important function of the microcirculation. This function is dependent upon the organ within which the vascular bed is present.

When microvessel endothelial cells have been isolated from animal tissues, the individual cells possess the ability to attach to a polystyrene or collagen-type surface and form a monolayer. These cells may form a cobblestone monolayer or, under certain conditions, may form tubes upon the tissue culture surface. These tubes are reminiscent of the neovascularity seen in tumors and wound healing. However, immunocytochemical studies suggest that the original luminal surface is actually on the outer surface of the tube in cultured cell lines. The demonstration of different endothelial cell morphologies as a function of substrate con-

forms with the general observation of cellular phenotypic modulation by substrate seen for most types of cells in culture. One important aspect of microvessel endothelial cells in culture is that they possess the ability to form a cobblestone monolayer. This is important when considering their usefulness in vascular graft endothelialization. It suggests that if placed in the proper environment, microvessel endothelial cells could in fact endothelialize a vascular graft.

In order to study these cells, considerable effort has been put forth to develop techniques to isolate and culture these cells. Some of the original studies were done by Dr. Stuart K. Williams, a member of this laboratory. The following section describes the current method of isolation and culture of microvessel endothelial cells as used in our laboratory.

Isolation of Human Adult Microvascular Endothelial Cells

Human peri-nephric, omental or subcutaneous fat may be obtained from brain-dead, heart-beating cadaver organ donors or patients undergoing unrelated surgical procedures in accordance with the institutional review board protocol. The fat is mechanically minced and placed with collagenase 4 mg/ml and bovine serum albumin 4 mg/ml. The flasks are incubated for 25 minutes at 37° C with constant agitation. The contents of the flask are then centrifuged at 200 × g for 7 minutes. The cell preparation at this point demonstrates both single cells and microvascular tufts.

The resultant EC are resuspended in 45% Percoll and centrifuged at 20,000 × g for 20 minutes at 4°C. The tufts of capillary EC migrate to a milky-white layer at the top of the density gradient while vessel fragments, red cells and cellular debris migrate to positions lower within the gradient. The capillary EC are washed and resuspended in medium 199 with 20% fetal calf serum. After this step in the isolation, a single cell suspension results. These cells can be cultured resulting in confluent monolayers of EC (Figure 10).

Using this technique, adipose tissue was obtained from 13 individual donors and included perinephric and omental fat sources. EC were successfully isolated from all 13 donors. Elapsed time for the three stages of EC isolation were 29.9 ± 1.3 minutes for collagenase, 20 minutes for Percoll and 30 minutes for washes and handling for cell counts. We have subsequently performed this isolation for over 150 different samples of human fat and found it to produce highly consistent cells.

Identification as Endothelial Cells

Morphological examination of a primary fat isolate reveals predominantly single cells with occasional clusters of cells which maintain their tube-like structure. The identification of these cells as endothelium is difficult at the time of isolation since the most commonly used marker, the expression of factor VIII related antigen, is routinely performed on cells grown on glass coverslips. Therefore, to identify the cells as endothelial cells, we routinely establish primary cultures of each isolate and subsequently analyze these cultures for the expression of metabolic activities and surface markers expressed by endothelium. The most definitive test remains expression of factor VIII related antigen which is observed

on the vast majority of cells in the primary isolate. Metabolic markers including angiotensin-converting enzyme activity, prostacyclin production and type IV collagen synthesis are also expressed by these primary isolates. By itself, each metabolic activity is not necessarily endothelial cell specific. However, expression of a combination of these functions provides strong evidence for the endothelial origin of these cells. Evaluation of primary cultures of microvessel endothelium for all of these markers has established that endothelial cells are the predominant cell in the primary culture and therefore it is likely that endothelial cells predominate in the primary isolate.

Use of Microvascular Endothelial Cells on Vascular Grafts

We have begun to study both freshly isolated and cultured human microvessel endothelial cells and their interactions with vascular grafts. Our initial studies examined endothelial cell interaction with dacron. Microvessel endothelial cells demonstrated an interaction that was similar for large vessel endothelial cells. In particular, they showed poor interaction with the surface itself and a limited ability to multiply. This could be somewhat enhanced by the addition of substrate to the surface of the dacron. We examined both human plasma as well as collagen type I/III and found that these substrates could improve adherence and, under certain circumstances, even allow these cells to grow to confluence. We are initially excited about this in that growth to confluence suggested a surface that was receptive to these cells. One difficulty with these and similar types of studies is that the critical variable then becomes adherence of substrate to dacron. Dacron is a very non-wettable surface. Thus, when a substrate is placed upon this surface, there is a poor physical interaction between the two materials. Few ionic or covalent bonds form. Thus even though the substrate coating remains temporarily adherent, its long term adherence may be impaired. Large areas of substrate detaching from the graft surface in spite of the presence of endothelial cells would certainly limit the effectiveness of substrate-coated vascular grafts. Experiments similar to this on PTFE demonstrated a similar observation. Untreated PTFE demonstrated a poor interaction with endothelial cells. However when substrate was added, good endothelial cell adherence was seen. If these cultures were held in tissue culture for several weeks, one noted that the entire endothelial cell-substrate layer floated off the graft surface into the tissue culture medium.

As our studies proceeded, it became increasingly apparent that the critical variable for a desirable interaction between endothelial cells and grafts was adherence between the cells and the graft polymer surface. We suspected this to be important because we had noted that if the cell could adhere, then growth was likely. If, however, the cell could not adhere, then obviously growth could not take place. We also began to focus on freshly isolated microvessel endothelial cells rather than cultured cells. Immediately following digestion of fat and isolation of endothelial cells, the cells are placed upon the graft material and the interaction examined. This avoids the introduction of growth stimulants and selection for adherent cells by tissue culture techniques. Instead, it examines direct adherence of the isolated cells to the surface.

If one plans to run a clinical trial without the need for tissue culture, then freshly isolated microvessel endothelial cells would be the most pertinent cell to examine. In our *in-vitro* studies we noted that plasma and gelatin coated dacron surfaces did support the adherence of microvessel endothelial cells. This adherence was slightly slower than that observed from large vessel endothelial cells. For large vessel endothelial cells, we have previously noted that rapid adherence takes place to plasma coated dacron within ten minutes and to amnion coated dacron within thirty minutes. When microvessel endothelial cells were used, 60 to 120 minutes were necessary for adherence to plasma coated dacron to occur. This difference may represent either incomplete collagen removal from the endothelial cell by the collagenase or surface tension problems encountered in the freshly isolated cell. Further studies are under way to determine the critical variables.

Under laboratory conditions with freshly isolated cells on plasma coated dacron, rapid cell spreading and areas of cell-to-cell interaction occurred. If several hours were allowed to pass, areas of definite monolayer could be seen. In order to quantitate the degree of adherence, we then exposed these adherent cells to a defined shear stress. Defined shear stress was created in a parallel plate slit flow chamber that was developed in our laboratory. These conditions expose the cells to shear stresses between 0 and 120 dynes/cm^2. In all experiments, greater than 50% of the cells remained adhered to the surface in the presence of the shear stress. In later experiments we have observed that only a 15 to 20 minute incubation on polystyrene is necessary for strong adherence to occur. We are currently extending this approach to other plastic materials with vascular graft potential. A number of biocompatible, compliant surfaces have allowed equally rapid cell attachment to occur. If identification of the optimal surface is possible, it suggests that a graft could be rapidly endothelialized with freshly isolated microvessel endothelial cells. The time factors involved in the isolation technique (30–60 minutes) as well as the attachment process (30–60 minutes) are quite compatible with an operating room technique. All of the procedures used in the isolation are quite amenable to an operating room facility. With this is mind, we have begun to examine what other important variables would need to be understood prior to undertaking such a procedure. There are many variables including donor variables, surface variables and cellular interaction variables. We have attempted to examine some of these to determine their net effect.

Other Important Variables

A. Donor Variables. Since endothelial cells are divided from individual donors, there exists the possibility that the cells may demonstrate donor to donor variation. Although there are many possible important donor factors, it is probable that two very significant variables may be the donor age and the presence of systemic disease. The effect of *in-vivo* donor age on endothelial cell morphology was suggested by Repin and associates. They observed increasing endothelial cell size with increasing age on human adult arterial vessels. Increasing bovine and human endothelial cell size *in-vitro* has been observed by Levine and associates to be related to increased endothelial cell age in culture, particularly as cellular

senescence begins. Diabetic patients may also exhibit altered endothelial cell function. We have noted that the isolation yield and the adherence to dacron of microvessel endothelial cells from diabetic patients is significantly less than from nondiabetic patients. These and many other undefined variables suggest that the success rate for endothelializing a surface may be donor dependent.

B. Surface variables. The second major determinant for a successful endothelial cell monolayer is the polymeric surface. A major problem in this area is the well documented observation that dacron or PTFE as supplied by the manufacturers does not support optimal human endothelial cell adherence or growth. As a result, many investigators have resorted to a "coating" placed upon the surface prior to the introduction of the endothelial cells. These coatings have included collagens, fibronectin, laminin, blood-derived plasma, blood and glycosaminoglycans. One difficulty with this approach is the potential non-uniformity of the coating. We have observed on multiple occasions that the polymer surface as received from the manufacturer frequently needs extensive degreasing before a uniform surface as determined by endothelial cell adherence occurs. This undoubtedly affects protein adsorption as well as cell adherence and may also affect protein conformation on the surface. A second difficulty with coatings is the possible metabolism of surface coatings by the cell itself. Grinnell has demonstrated fibroblast degredation of fibronectin adsorbed onto polystyrene. This might affect adsorbed as well as covalently linked proteins and could result in cellular detachment. One potential solution to this problem is the identification of polymeric surfaces that do not require exogenously added proteins for rapid cellular attachment and spreading to occur.

C. Interaction Variables. The third group of variables involved in understanding the process of graft endothelialization is interaction variables. Clearly environmental conditions will be important in endothelial monolayer formation. These variables include pH, temperature, cell sodding concentration, as well as many others. Understanding these interaction variables may be critical in any individual experiment in determining the success or failure of monolayer formation. We have begun to study these variables but at this point, no conclusive data on this is available.

Once we understand the cell, surface, and interaction variables that are important in monolayer formation, we will then begin to implant endothelialized surfaces into experimental animals as well as humans. Before we begin human studies, a number of important factors will have to be examined. These relate specifically to the stability of the monolayer in the presence of flow as well as other methods to evaluate the surface.

Endothelialized Surface Evaluation

In many respects, the evaluation of an endothelialized surface is similar to the evaluations performed on a pure polymer graft. These evaluations include measurements of thrombogenicity, surface stability, compliance and downstream effects such as effects on anastomotic cellular hyperplasia. While nonthrombogenicity has been the goal in polymer prosthesis research, endothelialized grafts should be anti-thrombogenic, implying active surface properties.

These properties include clotting protein degredation and release of factors such as plasminogen activator and prostaglandins.

Although this represents a complex *in-vitro* and *in-vivo* series of tests, the major advantage for the endothelialized graft is the standard, which is native vessel endothelium, available for comparison. While the final endpoint of "non-thrombogenicity" is difficult to define for pure polymer surfaces, the anti-thrombogenic features of an endothelialized graft can be evaluated. Initial requirements for cell monolayers would include normal morphology of the monolayer and its cellular components, including a mature junctional complex and production of a full complement of membrane proteins. Further requirements for an endothelial cell monolayer on a prosthetic surface are the ability to degrade thrombin, to release plasminogen activator, to produce prostaglandins and to demonstrate normal endothelial cell immunological surface markers. Since endothelial cells are known to possess the ABH blood group system and the HLA A, B and D histocompatibility system, these antigens should be present or inducible on the graft monolayer. In addition, the graft endothelial cell should not stimulate autogenous lymphocytes in mixed endothelial cell-lymphocyte co-culture differently from native vessel endothelial cells. Any immunological attack upon the monolayer might result in monolayer destruction and loss of the anti-thrombogenic surface. Lastly, there should be evidence that there has been no genetic damage to the EC during the isolation procedure.

Another area of concern is the long term *in-vivo* stability of the endothelial cell monolayer. Grafts seeded with endothelial cells have been noted to develop a subendothelial network of cells and connective tissue over the course of months following graft implantation. These layers have ranged up to several hundred microns in thickness. The precise explanation for these layers is unknown but could relate to a cellular response to wall stress, endothelial cell incompatibility with the prosthetic surface, lack of a smooth muscle cell substrate, or a "healing" response. If subendothelial matrix remains static after a period of time, then no further concern is necessary. If the matrix is unstable or there appears to be a significantly elevated baseline mitotic index of the overlying monolayer, then smooth muscle cells and/or a collagen-based graft structure maybe necessary for a stable monolayer to occur. Another possible problem with the endothelial cell monolayer may be related to the use of microvessel endothelial cell to establish a monolayer in a large diameter vessel. Microvessel endothelial cell are known to have the capacity to form small tubular structures rather than a cobblestone monolayer. This tendency to express multiple cellular morphologies may be important if it affects the antithrombogenicity of the graft.

After completion of the steps necessary to allow a monolayer to form, it would be very desirable to know whether a monolayer truly exists. This quality control step is necessary to avoid implanting a graft that fails to endothelialize and therefore that would be doomed to thrombosis and failure. It is exceedingly difficult to visualize living endothelial cells on dacron and PTFE surfaces because of their opacity at visible light wave lengths and autofluorescence in the 400 to 500 nm wavelength range. Staining with conventional nuclear dyes yields cell number, but is destructive and gives little information regarding cell spreading or cell to cell contact. Therefore cell visualization must be performed using cytoplasmic dyes that exhibit fluorescent light emission in the 500 to 600 nm wavelength range. This is perhaps optimally utilized on the implanted graft itself rather than a "control" segment to maximize the chance of graft success. Thus the dye should be non-toxic and acceptable for intra-arterial use.

A second cause of vascular graft failure is the development of strictures at the anastomotic junction between native vessel and graft. The precise mechanism for these strictures is unknown, but cellular hyperplasia is a major component histologically. One factor important in its development may be compliance differences between the graft and vessel. These variables may be important regardless of the presence or absence of endothelium. A second factor in anastomotic stenosis may be the activation of platelets and coagulation factors by the thrombogenic prosthetic surface. Addition of an endothelium could greatly alter these factors and change cellular stimuli occurring at the anastomosis. A related matter is the effect of pulsatile contraction and expansion on endothelial cell monolayer function. Previous investigators have noted changes in EC prostaglandin production when pulsatile perfusion is added to a rigid static experimental system.

Clinical Possibilities

Once the *in-vitro* technique for rapidly and reproducibly establishing a microvessel endothelial cell monolayer upon a vascular graft has been perfected, the clinical technique will have to be developed. One current concept is that the entire procedure may be performed during surgery. The initial step requires the removal of 30 to 40 grams of subcutaneous fat from the patient. This may be performed before or after the induction of anesthesia. The surgeon would then proceed with the vascular operation as usual. The fat would be handed to a technician at the back table who would digest the fat with collagenase and separate a pure isolate of endothelial cells. A graft of approximately the correct size and length would be selected. It is likely that this graft would not be composed of dacron or PTFE but rather a material empirically chosen from the laboratory studies. The cells and medium would be placed into the graft, which would be sealed at each end. Axial rotation of the graft would be used to allow a uniform distribution of cells throughout the graft. During this rotation, the cells would attach, spread and form a monolayer. A segment of graft would then be excised for quality control purposes. If fluorescently labelled cells are used, then simple observation with a fluorescent microscope can be performed and an immediate result obtained. Otherwise, a segment may be permanently fixed for light and electron microscopy. The endothelialized graft may then be implanted into the patient. There will have to be minor alterations in surgical technique in three major areas. First, no clamps may be applied to the new surface. Second, the graft may not be forcibly pulled through tissue tunnels or otherwise manipulated. Third, it is extremely important to keep the cells in contact with culture medium until blood flow is restored. This will prevent cell dessication and death. Blood flow through the graft will be restored gradually, but no other precautions seem necessary at our present state of knowledge. If the process has been exe-

cuted properly, then the first blood to contact the graft should "see" functional endothelium. Presumably then we will test the hypothesis of whether an endothelialized graft is less thrombogenic and demonstrates longer patency than the currently available grafts.

In summary, either spontaneously generated or seeded endothelial lined prosthetic surfaces will emerge in the clinical arena over the next decade. These surfaces should be evaluated using many of the currently available techniques that have been applied to pure polymer surfaces. Endothelialized surfaces offer the first step in replicating normal vessel wall function. Careful quality control measures must be undertaken to assure that the morphology and function of the EC monolayer are very similar to the native vessel.

Transactions of the Philadelphia Academy of Surgery

Regular Meeting

January 5, 1981

The meeting was called to order by President Brooke Roberts at 8:15 p.m.

Scientific Session

"A Classification of Surgical Decisions" was presented by John Clarke, M.D. and discussed by R. Robert Tyson, M.D.

"Non-Invasive Laboratory Evaluation of Deep Venous Thrombosis in Critically Sick Patients" was presented by Teruo Matsumoto, M.D. and discussed by President Brooke Roberts.

"Total Parenteral Nutrition in the Cancer Patient" was presented by J. J. Steinberg, M.D. and discussed by Dr. Thomas Frazier, Dr. Jonathan Rhoads, and Dr. Francis Rosato.

Regular Meeting

February 2, 1981

The meeting was called to order by President Brooke Roberts at 8:15 p.m.

Scientific Session

"Leiomyosarcoma of the Abdominal Aorta: A Case Report" was presented by John Millili, M.D. and Raymond LaFare, M.D. There was no discussion because of the rarity of the lesion but a question was posed by Dr. Plzak.

"Non-resectional Treatment of Abdominal Aneurysms" was presented by Ronald P. Savarese, M.D., Joel C. Rosenfeld, M.D., and Dominic DeLaurentis, M.D. and discussed by Charles Wolferth, M.D. and William Hardesty, M.D.

"A Monoclonal Antibody Against Colon Cancer" was presented by Henry F. Sears, M.D. and Paul Grotzinger, M.D. and discussed by Dr. Clyde Barker.

Conjoint Meeting with New York Surgical Society

The annual Joint Meeting with the Philadelphia Academy of Surgery was held at the University Club, 1 West 54th Street, New York City, on March 11, 1981 at 2:00 p.m.

Papers

I. M. Modlin, M.D., A. Sank, M.D., D. Albert, M.D., B. Jaffe, M.D. "The Diagnosis of Zollinger-Ellison Syndrome."

Discusser: Dr. Charles C. Wolferth, Jr.

V. Charoenkul, M.D., P. H. Tey, M.D., A. Ahmed, M.D., E. C. Peirce, II, M.D., A. J. McElhinney, M.D. "Hemodynamic Improvement After Percutaneous Transluminal Angioplasty."

Discusser: Dr. Clyde F. Barker

F. P. Herter, M.D., A. M. Cooperman, M.D., T. N. Ahlborn, M.D., C. Antinori, M.D. "Surgery for Pancreatic and Periampullary Cancer."

Discusser: Doctor Jonathan E. Rhoads, Sr.

B. S. Gingold, M.D., W. F. Mitty, Jr., M.D., M. Tadros, M.D. "Importance of Patient Selection in Local Treatment of Rectal Carcinoma."

Discusser: Dr. Gerald Marks

Frank Glenn, M.D., Carl G. Becker, M.D. "The Induction of Acute Cholecystitis in Animals."

Discusser: Dr. James G. Bassett

S. Philipshen, M.D., J. Smith, M.D., S. Yeh, M.D., A. Fracchia, M.D., T. Hakes, M.D., D. Kinne, M.D. "Significance of Abnormal Bone and Liver Scans in Stage II and III Breast Cancer Patients."

Discusser: Dr. Hunter S. Neal

Regular Meeting

April 6, 1981

The meeting was called to order by Dr. Robert Tyson in absence of Dr. Brooke Roberts.

Scientific Session

"The Ruptured Abdominal Aortic Aneurysm—A Surgical Enigma" was presented by Dominic DeLaurentis, M.D., Joel C. Rosenfeld, M.D. and Ronald Savarese, M.D. and discussed by Drs. Reichle and Rosato.

"The Clinical Correlation of the Burn Wound Biopsy Quantitative Culture" was presented by Charles Hartford, M.D., S. Randolph May, M.D., and Cynthia Panoc, R.N. and was discussed by Dr. DeClement.

"Chronic Recurrent Breast Abscess" was presented by Willis P. Maier, M.D., H. Taylor Caswell, M.D. and Alan Berger, M.D. and discussed by Dr. Wagner.

Regular Meeting

May 4, 1981

The meeting was called to order by President Brooke Roberts at 8:15 p.m.

Scientific Session

"The Routine Use of Fine Needle Aspiration Cytology in the Evaluation and Treatment of Breast Masses" was presented by Thomas G. Frazier, M.D. and discussed by Dr. Clifton West and Dr. William Stainback.

"Closure of Diverting Colostomy—Ileostomy Personal Experience with 100 Consecutive Cases" was presented by R. Anthony Carabasi, M.D. and discussed by Dr. Gerald Marks.

"Phantom Sensations Following Mastectomy for Malignant Disease" was presented by Harry Rosenblum, M.D. and discussed by Dr. Thomas Frazier, Dr. Elmer Grimes, and Dr. Gordon Schwartz.

Regular Meeting

October 5, 1981

The meeting was called to order by President Brooke Roberts at 8:15 p.m.

Scientific Session

"The Management of Arterial and Venous Injuries" presented by Drs. Bokhari, Fallanhnejad and Nemir and discussed by Drs. Templeton and Gowen.

"Total Fecal Diversion by the Loop Transverse Colostomy" presented by Drs. Rombeau and Miller and discussed by Dr. Fischer.

"Malignant Duodenocolic Fistulas: A Case Report and Review of the Literature" presented by Alfred E. Chang and Jonathan E. Rhoads and discussed by Drs. Marks, Buyers, and Troncelliti.

Regular Meeting

November 2, 1981

The meeting was called to order by President Brooke Roberts at 8:15 p.m.

Scientific Session

"Role of Real-time Ultrasonic Imaging in the Diagnosis of Carotid Artery Stenosis" was presented by Anthony J. Comerota, M.D. and discussed by Dr. John Clark, Dr. Stanton Smullens and Dr. Gardner.

"Bowel Obstruction in Cancer Patients—Relationship between Site of Primary and Prognosis" was presented by Dr. John Skibber and discussed by Dr. Wagner, Dr. Moss, and Dr. Weiss.

"Radical Treatment of Pilonidal Cyst and Sinus Tract" was presented by Dr. Edgardo Alday and discussed by Dr. Troncelliti.

Regular Meeting

December 7, 1981

The meeting was called to order by President Brooke Roberts at 8:15 p.m.

Scientific Session

"The Aorta-Enteric Fistula: Diagnosis and Management" was presented by Victor Bernhard, M.D. and discussed by Drs. Wolferth and Roberts.

"The Use of Diagnostic Ultrasound in Evaluation of Dysplastic Breast" was presented by Thomas Frazier, M.D. and discussed by Dr. Roberts.

The annual oration was delivered by Moreye Nusbaum, M.D.

Annual Report of the Secretary
December 1981

In 1981 there were eight formal meetings of the Philadelphia Academy of Surgery, seven regular meetings in Philadelphia and a Conjoint Meeting held in New York in conjunction with the New York Surgical Society.

The Conjoint meeting began at 2:30 p.m. at the University Club, on March 11, 1981. There were appoximately 75 members from the New York group and 37 members from the Philadelphia group. Dr. Brooke Roberts from Philadelphia and Dr. Keith Reestma from New York each presided over one half of the meeting. Six papers were presented by the New York Fellows and discussed by Fellows of the Philadelphia Academy of Surgery. A sumptious dinner with appropriate beverages was hosted by the New York members amid lively conversation and deepening friendship.

In January, the election of officers was held and a new slate of officers and members of Council were elected as follows:

President—Brooke Roberts, M.D.
First Vice-President—Paul Nemir, M.D.
Second Vice-President—R. Robert Tyson, M.D.
Secretary—Frederick B. Wagner, Jr., M.D.
Treasurer—Charles Wolferth, M.D.
Recorder—Elmer L. Grimes, M.D.
Chairman, Committee on Scientific Business—Francis E. Rosato, M.D.
Members-at-Large—Donald R. Cooper, M.D.; James G. Bassett, M.D.; Willis P. Maier, M.D.
Chairman of the Samuel D. Gross Prize Fund—Moreye Nusbaum, M.D.

Two members of the Academy died during the year and their memoirs were presented as follows:

Alex Ulin, M.D. presented by Paul Grotzinger, M.D.
Herbert Hawthorne, M.D. presented by Paul Nemir, Jr., M.D.

On December 7, 1981, the Annural Oration was given by Dr. Moreye Nusbaum entitled "Vasopressin in Surgery."

Frederick B. Wagner, Jr., M.D.

Regular Meeting
January 4, 1982

The meeting was called to order by President Brooke Roberts.

Scientific Session

"Chronic Sialadenitis of the Parotid" presented by Dr. Henry Scheuermann. There was no formal discussion but many questions were asked.

"Enterocystoplasty to Augment Bladder Capacity" to be presented by Dr. Alan Wein was not delivered in view of his unexplained absence.

"Prophylactic Mastectomy" was presented by Anne Barnes, M.D. and discussed by Dr. Rosato and other Fellows participated in the questioning.

Regular Meeting
February 1, 1982

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

"Echinoccal Cyst of the Liver" was presented by Clint Brooks, M.D. and Sergius Pechin, M.D. and discussed by Drs. Rosato and West.

"The Delayed-Transfer Burn Patient: Decisions, Complications, and Outcomes" was presented by S. Randolph May, M.D. and there was no discussion.

"Modified Hill Repair in Hiatus Hernia with Reflux" was presented by Anthony Coletta, M.D. and discussed by Drs. Gowen and Fallahnejad.

Joint Meeting with New York Surgical Society

The annual Joint Meeting with the Philadelphia Academy of Surgery was held at the Philadelphia College of Physicians, on March 10, 1982 at 2:00 p.m.

Papers

W. C. Hargrove, M.D., E. J. King, M.D., G. K. McLean, M.D., D. B. Freiman, M.D., H. D. Berkowitz, M.D. and B. Roberts, M.D. "Recanalization of Totally Occluded Vein Grafts Using Low Dose Streptokinase."

Discusser: Dr. Philip N. Sawyer

Charles C. Wolferth, Jr., M.D., Martin F. Hayes, Jr., M.D. and George Amrom, M.D. "The Changing Bacteriologic Spectrum in Infected Prosthetic Vascular Grafts."

Discusser: Anthony M. Imparato, M.D.

Gerald M. Lemole, M.D., Michael D. Strong, M.D.*, Paschal M. Spagna, M.D.*, N. Peter Karmilowicz, M.D.* "Improved Results for Dissecting Aneurysms Intraluminal Sutureless Prosthesis."

Discusser: Anthony Acinapuva, M.D.

G. Marks, M.D., R. Boova, M.D., and Mohiuddin, M.D. "Full Dose Preoperative Radiation Therapy and Anal Sphincter Preservation in the Management of Rectal Cancer."

Discusser: Warren E. Enker, M.D.

Howard H. Steel, M.D. "Convex Rib Resection in Scoliosis."

Discusser: John J. Gartland, M.D.

Thomas G. Frazier, M.D., R. Barrett Noone, M.D. "Immediate Reconstruction in Carcinoma of the Breast: Is This a Rational Alternative?".

Discusser: David V. Habif, M.D.

*By Invitation

Regular Meeting
April 5, 1982

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

"Exposed Vascular Prosthesis" was presented by R. Barrett Noone, M.D. and discussed by Dr. Brooke Roberts, Dr. Tyson, Dr. Fallahnejad, and Dr. Randall.

"Intraarterial Thrombolytic Therapy in Acute Arterial Occlusions" was presented by Dr. Comerota and discussed by Dr. Sol Sherry and Dr. Templeton.

"Why are We Abandoning Radical Mastectomy in the Treatment of Breast Cancer?" was presented by Charles Fineberg, M.D. and discussed by Dr. Rosato, Dr. Grimes, Dr. Grotzinger and Dr. Frazier.

Regular Meeting
May 3, 1982

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

"The Psychological Characteristics of Women Who Develop Breast Cancer" was presented by Dr. Gordon Schwartz and no discussion followed.

"Endoscopic Diagnosis of Familial Polyposis in the Upper and Lower GI Tracts" was presented by George Gowen, M.D. and discussed by Dr. Schumann and Dr. Troncellitti.

"Cloacogenic Carcinoma" was presented by Jon Dzwonczyk and discussed by Dr. Frederick Wagner.

Regular Meeting
October 4, 1982

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

"Phosphorus-31 Nuclear Magnetic Resonance Evaluation of Peripheral Vascular Disease" was presented by Dr. Henry Berkowitz and discussion followed by Dr. Brooke Roberts.

"Treatment of Postinfarction Ventricular Septal Defects by Early Surgical Intervention" was presented by Paschal Spagna, M.D. and discussion followed by Dr. Henry Edmunds.

"The Role of Cytology in the Diagnosis of Gastric Malignancy" was presented by Francis Au, M.D. and discussed by Dr. George Gowen and Dr. Henry Moss.

Regular Meeting
November 1, 1982

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

"Breast Reconstruction after a Mastectomy" was presented by Charles Pappas, M.D. and discussed by Drs. Randall, Rosato and Gayer.

"Thoracic Aorta to Femoral Artery Bypass" was presented by Ronald Savarese, M.D. and discussed by Dr. Erwin Cohen.

"Gastric Bypass for Morbid Obesity" was presented by David Sensenig and discussed by Drs. Rosato and Troncelliti.

Regular Meeting
December 6, 1982

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

"Routine Blood Conservation During Cardiac Surgery with Use of Hemanatics Cell Saver" was presented by Dr. Nick Cavarocchi.

"Surgery of Abdominal Aortic Aneurysms in Peculiar Anatomic Situations" was presented by Dr. Dominic DeLaurentis.

The annual oration was given by Michael O'Connor, M.D.

Annual Report of the Secretary
December 1982

In 1982, there were eight formal meetings of the Philadelphia Academy of Surgery, seven regular meetings in Thompson Auditorium and a Conjoint Meeting with the New York Surgical Society held in Mitchell Hall. The average attendance at the regular meetings was 85.

The Conjoint Meeting began at 2:00 p.m. at the College of Physicians on March 10, 1982. There were approximately 35 members from the New York group and 65 members from the Philadelphia group. Dr. Paul Nemir from Philadelphia and Dr. Gerald W. Shaftan from New York each presided over one half of the meeting. Six papers were presented by the Philadelphia Fellows and discussed by Fellows from New York. A dinner at the Union League was hosted by the Philadelphia Academy of Surgery, and Mrs. Maxwell Whiteman, historian of the Union League, gave a thumbnail sketch of its founding and historical highlights.

At the December meeting, the slate of new officers and members of Council were proposed by the Nominating Committee as follows:

President—R. Robert Tyson, M.D.
1st Vice President—Charles C. Wolferth, Jr., M.D.
2nd Vice President—Frederick B. Wagner, Jr., M.D.
Secretary—James G. Bassett, M.D.

Treasurer—Willis P. Maier, M.D.
Recorder—Elmer L. Grimes, M.D.
Chairman, Committee on Scientific Business—Francis E. Rosato, M.D.
Council at Large—Paul Nemir, M.D.
Harry V. Armitage, M.D.
William Stainback, M.D.
Chairman of the S.D. Gross Prize—to be appointed by President Nemir

One Member of the Academy died during the year: Alfred Ayella, M.D.

On December 6, 1982, the Annual Oration was given by Michael J. O'Connor, M.D. entitled "Defects in Cerebral Function and Metabolism and the Role of Revascularization."

Regular Meeting
January 3, 1983

The meeting was called to order by President Paul Nemir at 8:15 p.m.

Scientific Session

The Samuel D. Gross Prize Oration was given by John R. Clarke, M.D. "The Application of Decision Sciences to Surgical Judgement." There was no discussion following the presentation.

Regular Meeting
February 7, 1983

"Streptococcus Bovis Endocarditis and it's Association with Colon Carcinoma" Stephen C. Silver, M.D. (by invitation), Sergius Pechin, M.D.

"Central Catheter Fibrin Sleeve—Heparin Effect," Robert P. Ruggiero, M.D. (by invitation) R. Robert Tyson, M.D.

"CT Scanning of the Lumbar Spine" Stephen J. Bosacco, M.D. (by invitation) Arnold Berman, M.D.

Conjoint Meeting with New York Surgical Society

The annual Joint Meeting with the Philadelphia Academy of Surgery was held at the UNIVERSITY CLUB, 1 West 54th Street, New York City, on March 9, 1983 at 2:00 P.M.

Papers

David W. Kinne, M.D. and Susan Groshen, P.D.*: "One-Stage Versus Two Stage Procedure for Breast Cancer, The Memorial Hospital Experience." Discusser: Hunter F. Neal, M.D.

G. R. Orangio, M.D.*, S. D. Pitlick, M.D., P. Della Latta, Ph.D.*, L. J. Mandel, M.D.*, C. Marino, M.D.*, J. J. Guarneri, Ph.D., J. A. Giron, M.D.* and I. B. Margolis, M.D.: "Soft-Tissue Infections in Parenteral Drug Abusers: Bacteriologic And Immunologic Aspects." Discusser: Rudolph C. Camishion, M.D.

William Doscher, M.D.*, Bala Viswanathan, M.D., Theodore Stein, M.S.*, and Irving B. Margolis, M.D.: "Hemodynamic Assessment of the Circulation in 200 Normal Hands." Discusser: R. Robert Tyson, M.D.

T. Heimann, M.D.*, R. J. Kurtz, M.D.* and A. H. Aufses, Jr., M.D. "Mucosal Protectomy Using the Ultrasound Scalpel." Discusser: Gerald Marks, M.D.

Michael R. Treat, M.D.* and Kenneth A. Forde, M.D.: "Colonoscopy, Technetium Scanning And Angiography In Acute Rectal Bleeding—an Algorithm For Their Combined Use." Discusser: Ernest S. Rosato, M.D.

Arvinder Singh, M.D.*, Norman D. Bloom, M.D.*, Susanna Cunningham-Rundles*, Michael Neuwirth*, William H. Stahl, M.D.: "Quantitative and Qualitative Alterations In the Immune System With Progressive Surgical Stress." Discusser: Clyde F. Barker, M.D.

*By Invitation

Regular Meeting
April 2, 1983

"Antenatal Diagnosis of Choledochal Cysts—Implications Regarding Etiology and Therapy"—Michael B. Marchildon, M.D. (by invitation) Rudolph C. Camishion, M.D.

"Recent Results with Esophageal Atresia"—James N. O'Neill, M.D. (by invitation) L. Henry Edmunds, Jr., M.D.

"Long-Term Follow-Up in Aortocoronary Saphenous Vein Grafting with Severe Impairment of Ventricular Function"—Scott Goldman, M.D. (by invitation) Stanley K. Brockman, M.D.

Regular Meeting
May 3, 1983

"En Bloc Exicsion of Cadaver Kidneys for Transplantation"—James E. Colberg, M.D. (by invitation) Francis E. Rosato, M.D.

"Thoracic Outlet Syndrome—The Total Picture"—Stanton Smullens, M.D., Scott Jaeger, M.D. (by invitation).

"Evaluation of the Burn Wound with Perfusion Fluorometry"—John Gatty, M.D. (by invitation) Donato LaRossa, M.D., David G. Silverman, M.D. (by invitation) Charles E. Hartford, M.D.

Regular Meeting
October 3, 1983

Scientific Session

"Implantable Pump for Infusion Chemotherapy," Stephen M. Weiss, M.D. (by invitation), Francis E. Rosato, M.D.

"101 Consecutive Colon Operations Without Wound Infection: Principles

and Techniques," M. J. Pello, M.D. (by invitation) W. Beauregard, M.D. (by invitation), K. Shaikh, M.D. (by invitation), Rudolph Camishion, M.D.

"Etiology of Diffuse Intravascular Coagulation (DIC) in Patients with Le-Veen Shunts." V. Paul Addonizio, Jr., M.D. (by invitation) William Inouye, M.D.

Regular Meeting

November 7, 1983

The November 7, 1983 meeting of the Philadelphia Academy of Surgery was called to order at 8:15 P.M. by President Robert Tyson.

Scientific Session:

"Hemodialysis on Cardiopulmonary Bypass for Acute Renal Failure," Gerald M. Lemole, M.D., Paschal M. Spagna, M.D.

"Obstruction Schwannoma of Hepatic Duct," Elmer L. Grimes, M.D.

"Transverse Pulmonary Embolectomy," Bruce E. Jarrell, M.D. (by invitation), John Moore, M.D. (by invitation), Francis Rosato, M.D.

Regular Meeting

December 5, 1983

"Hartmann Procedure for Treatment of Complicated Diverticular Disease of the Sigmoid Colon"—Karim B. Nakhgevany, M.D. (by invitation), Donald Cooper, M.D.

"Subcutaneous Capsule Placement for Long-Term Venous Access for Chemotherapy"—Constantinos Pavlives, M.D. (by invitation) Thomas Gain, M.D., Bruce MacDonald, M.D. (by invitation) James Laird, M.D. (by invitation), Isadore Brodsky, M.D. (by invitation) Teruo Matsumoto, M.D.

Annual Oration

"Evaluation of Artificial Hearts in Human Subjects," Jacob Kolff, M.D.

Annual Report of the Secretary

1983

There were 8 formal meetings of the Philadelphia Academy of Surgery in 1983. Seven of these meetings were held in Thompson Auditorium, and the Conjoint Meeting with the New York Surgical Society was held at the University Club in New York City. The average attendance at the regular meetings was 87. The dinner meetings preceding the regular meeting were well attended with an average of 65 members in attendance.

The Conjoint Meeting began at 2:00 p.m. at the University Club in New York City on March 9, 1983. There were 62 members of the Philadelphia Academy of Surgery present at that meeting, and about the same number of members from the New York group. Dr. James Humphries, Executive Director of the

American Board of Surgery presented a brief discussion of the role of the American Board of Surgery and its future.

Dr. Robert Tyson appointed a Nominating Committee consisting of Chairman, Paul Nemir, M.D., Brooke Roberts, M.D. and Donald R. Cooper, M.D. This committee submitted the following slate of officers and council members:

President—Charles C. Wolferth, Jr., M.D.
1st Vice President—Frederick B. Wagner, Jr., M.D.
2nd Vice President—Francis E. Rosato, M.D.
Secretary—James G. Bassett, M.D.
Treasurer—Willis P. Maier, M.D.
Recorder—Dominic A. DeLaurentis, M.D.
Chairman, Committee on Scientific Business—Clyde F. Barker, M.D.
Council-at-Large—R. Robert Tyson, M.D.,
William C. Stainbach, M.D.,
Clifton F. West, Jr., M.D.

Chairman of the S. D. Gross Prize Committee: to be appointed by the President

Two members of the Academy passed away during 1983. These were: Sherman Eger, M.D. and David Y. P. Lin, M.D.

Jacob Kolff, M.D., Professor of Surgery at Temple University School of Medicine, presented the Annual Oration on December 5, 1983, entitled "Evaluation of Artificial Hearts in Human Subjects."

Regular Meeting

January 10, 1984

The meeting was called to order at 8:15 p.m. by President Robert Tyson.

Scientific Session

"Alkaline Reflux Gastritis: A Diagnosis in Search of the Disease," Wallace P. Ritchie, Jr., M.D., (by invitation), R. Robert Tyson, M.D.

"Intraoperative Cholangiography: A Review of Indications and Analysis of Age-Sex Groups," Stephen B. Levine, M.D. (by invitation) Harvey J. Lerner, M.D., Elizabeth D. Leifer, M.D. (by invitation), Steven R. Lindheim, B.S. (by invitation).

"A Five-Year Study of the Results of Chemo-Nucleolysis—A Canadian Experience," Robert K. Jones, M.D.

Regular Meeting

February 6, 1984

The meeting was called to order at 8:15 p.m. by President Wolferth.

Scientific Session

"Wither Away General Surgery," James Humphries, M.D. (by invitation), R. Robert Tyson, M.D.

"Drool Control in Cerebral Palsy: A Team Control," Arthur S. Brown, M.D. (by invitation), Peter Randall, M.D.

"Studies of the Pancreas Allograft," Leonard J. Perloff, M.D. (by invitation), Clyde F. Barker, M.D.

Conjoint Meeting—March 14, 1984

The annual meeting of the Philadelphia Academy of Surgery and New York Surgical Society was held at the College of Physicians at 2:00 p.m.

Program

"Dissection and Significance of Interpectoral Lymph Nodes in Breast Cancer," Gordon F. Schwartz, M.D., Garry Ott, M.D. (by invitation), John Rhee, B.S. (by invitation).

"Critical Assessment of Gastric Bypass for Morbid Obesity," Charles E. Hartford, M.D.

"Inflammatory Abdominal Aortic Aneurysms," Ronald P. Savarese, M.D., Joel C. Rosenfeld, M.D. (by invitation), Dominic A. DeLaurentis, M.D.

"Carotid Endarterectomy Without A Shunt in Patients With Contralateral Carotid Artery Occlusion or Severe Stenosis," Rudolph C. Camishion, M.D., Richard K. Spence, M.D. (by invitation).

"Long-Term Growth of Adult Human Endothelial Cells in Tissue Culture," Bruce Jarrell, M.D. (by invitation), Elliot Levine, M.D. (by invitation), Francis E. Rosato, M.D.

"Total Revascularization Using Bilateral Sequential Internal Mammaries," Gerald Lemole, M.D., Paschal Spagna, M.D., Nadiv Shapia, M.D. (by invitation), N. Peter Karmilowicz, M.D. (by invitation).

Regular Meeting

April 2, 1984

The regular meeting of the Philadelphia Academy of Surgery was called to order at 8:15 p.m. by President Wolferth, Jr.

Scientific Session

"Comparison of the Clinical Performance of St. Jude and Procine Heterograft Aortic Valve Prostheses," Richard N. Edie, M.D. (by invitation).

"The Dioxin Dilemma," John R. Beljian, M.D. (by invitation).

"ERCP in the Diagnosis of Cystic Duct Syndrome," George F. Gowen, M.D.

Regular Meeting

May 7, 1984

The regular meeting of the Philadelphia Academy of Surgery was called to order at 8:10 p.m. by First Vice-President Frederick B. Wagner, Jr., M.D. in the absence of Charles C. Wolferth, Jr., M.D.

Scientific Session

John Y. Templeton, III, M.D., former president of the Philadelphia Academy of Surgery and the current President of the Pennsylvania Medical Society, spoke to the issue of the medical liability insurance crisis.

"Duplex Scanning of the Carotid Artery," Kathleen R. Noll, M.D. (by invitation), discussed by Dr. Henry Berkowitz.

"Clinical Use of Perfluorocarbons," Richard K. Spence, M.D. (by invitation), discussed by Drs. Henry Moss and Henry Berkowitz.

"Vascular Rings and Slings: Long-term Follow-up of Pulmonary Function," Jeffrey M. Dunn, M.D. (by invitation), discussed by Horace MacVaugh, M.D.

Regular Meeting

October 1, 1984

The regular meeting of the Philadelphia Academy of Surgery was called to order on Monday, October 1st, at 8:40 p.m. by President Wolferth, Jr.

Scientific Session

"Intestinal Valve Formation by Simple Invagination Technique," Doctors Kholoussy and Yand, discussed by Drs. Frank Rosato, Gerald Marks, and closed by Dr. Kholoussy.

"Patient Controlled-Analgesia—Update," Ward O. Griffen, Jr., M.D., discussed by Wallace Ritchie Jr. and Teruo Matsumoto, and then closed by Dr. Griffen.

"Tumor Cell Kinetic Alterations Produced by Parenteral Nutrition," Gordon Buzby, M.D., discussed by Wallace Ritchie, Frank Rosato, Charles Wolferth, and closed by Dr. Gordon Buzby.

Regular Meeting

November 5, 1984

The monthly meeting of the Philadelphia Academy of Surgery was called to order at 8:15 p.m. by President Wolferth.

Scientific Session

"Emergency Room Thoracotomy: A 26-Month Experience at Teaching Trauma Center." C. William Schwab, M.D. (by invitation) and Rudolph Camishion, M.D. Discussion was opened by Dr. James O. Finnegan and continued by Dr. Robert Trout and President Wolferth.

"The Healing of the Thin-Walled Expanded Polytetrafluoroethylene Graft" A. Mohsen Kholoussey, M.D. (by invitation), Kenji Takenaka, M.D., Ph.D. (by invitation) and Teruo Matsumoto, M.D. Discussion was opened by Dr. Dominic DeLaurentis and continued by Drs. Brooke Roberts and Rudolph Camishion.

"Pneumothorax During Mechanical Ventilation" John Mannion, M.D. (by

invitation) and Larry Stephenson. Discussion was opened by Dr. Brooke Roberts and continued by Drs. Herbert Cohn and Larry Stephenson.

Regular Meeting
December 3, 1984

The December 3rd meeting of the Philadelphia Academy of Surgery was called to order by President Charles C. Wolferth, Jr., M.D. at 8:20 p.m.

Scientific Session

"Surgical Management of Malignant Tumors of the Craniofacial Complex," Harvey M. Rosen, M.D., discussed by Linton Whitaker, M.D.

The Annual Oration was presented by Simon Simonian, M.D. The title of his talk was "The Influence of Renal Transplantation on Biology and Medicine."

Annual Report of the Secretary
1984

There were eight formal meetings of the Philadelphia Academy of Surgery in 1984. Seven of these meetings were held in Thompson Auditorium, and the Conjoint Meeting with the New York Surgical Society was held in Mitchell Hall, both at the College of Physicians of Philadelphia. The average attendance at the regular meetings was 130. The dinner meetings preceding the regular meetings were well attended with an average of 105 members in attendance.

The Conjoint Meeting began at 2:00 p.m. in Mitchell Hall of The College of Physicians of Philadelphia. There were 107 members of the Philadelphia Academy of Surgery present at that meeting, and about the same number of members from the New York group. Charles C. Wolferth, Jr., M.D. and Gerald W. Shaftan, M.D. presided over the individual sessions of the meeting. Six papers were presented by the Philadelphia Fellows and discussed by the membership of the New York Surgical Society. Dinner was held at the Union League hosted by the Philadelphia Academy of Surgery.

Dr. Charles C. Wolferth, Jr., appointed a Nominating Committee consisting of Chairman, Donald R. Cooper, M.D., Brooke Roberts, M.D., and Robert Tyson, M.D. This committee submitted the following slate of officers and council members:

President—Frederick B. Wagner, Jr., M.D.
First Vice-President—Francis E. Rosato, M.D.
Second Vice-President—Willis P. Maier, M.D.
Secretary—Dominic A. DeLaurentis, M.D.
Treasurer—Rudolph C. Camishion, M.D.
Recorder—David K. Wagner, M.D.
Chairman, Committee on Scientific Business—Clyde F. Barker, M.D.
Council-at-Large—Charles C. Wolferth, Jr., M.D., Clifton F. West, M.D., Moreye Nusbaum, M.D.

Simon Simonian, M.D. of Hahnemann University presented the Annual Oration of the Academy on December 3, 1984. His talk was entitled, "The Influence of Renal Transplantation on Biology and Medicine."

James G. Bassett, M.D., Secretary

Regular Meeting
January 7, 1985

President Charles C. Wolferth, Jr., M.D. called the Regular Combined Scientific/Business Meeting of the Philadelphia Academy of Surgery to order at 8:20 p.m.

Scientific Session

"The Three Synchronous Bilateral Lung Tumors: A Case Report" presented by Sindy M. Paul, M.D.

"114 Consecutive GI Fistulae Treated with Intravenous Hyperalimentation" presented by David Rose, M.D.

"Intersphincteric Proctectomy" presented by Thomas Logan Dent, M.D.

Regular Meeting
February 4, 1985

President Frederick B. Wagner, Jr., M.D. called the regular combined scientific/business meeting of the Academy of Surgery to order at 8:15 p.m.

"Carcinoma of the Esophagus Recent Experience" presented by Dominic A. DeLaurentis, M.D.

"Hyperparathyroidism" presented by Alfonse J. D. Giovanni, M.D.

"Control Platelet Reactivity During Open Heart Surgery" presented by Jeffrey Cappa, M.D. (by invitation sponsored by Clyde F. Barker, M.D.).

Conjoint Meeting with the New York Surgical Society—March 13, 1985

Abstracts presented at the Conjoint Meeting of the Philadelphia Academy of Surgery and New York Surgical:

"The Nissen Fundoplication: Results of Operation for Severe Reflux Esophagitis" Thomas H. Gouge, M.D., Department of Surgery, New York University, New York, NY. Discusser: Ernest F. Rosato, M.D.

"Laser Technology in the Treatment of Gastrointestinal Disease" A. Ghazi, M.D. and O. K. McSherry, M.D.—Beth Israel Medical Center and the Mount Sinai School of Medicine of the City University of New York. Discusser: Howard Zaren, M.D.

"Increasing Operability in Infrapopliteal Limb Salvage Surgery" Enrico Ascer, Frank J. Veith, Larry A. Scher, Sheila White-Flores, Sushil Gupta, Russell Samson, and Seymour Sprayregan, Montefiore Medical Center—Albert Einstein College of Medicine, New York, New York. Discusser: Frederick Reichle, M.D.

"Ileoanal Reconstruction After Urgent and Elective Colectomy for Ulcerative Colitis in the Adult" Kenneth Eng, M.D., New York University School of Medicine. Discusser: Gerald Marks, M.D.

"Adjuvant Immunotherapy Using Liposomesand Tumor Associated Antigens in an Experimental Colon Cancer Model" T. S. Ravikumar, M.D., G. D. Steele, Jr., M.D., J. N. Cunningham, Jr., M.D., Maimonides Medical Center, Brooklyn, New York. Discusser: Frank E. Rosato, M.D.

"Aortoiliac Femoral Endarterectomy: A Reappraisal" Neil Weintraub, M.D., Anthony M. Imparato, M.D., Thomas S. Piles, M.D., Patrick J. Lamparello, M.D., N.Y.U. Medical Center, New York, New York. Discusser: Dominic DeLaurentis, M.D.

Regular Meeting
April 1, 1985

President Frederick B. Wagner, Jr., M.D. called the regular combined scientific/business meeting of the Academy of Surgery to order at 8:00 p.m.

Scientific Session

"A Second Look at the Neostigmine Morphine Test," presented by George A. Gowan, M.D.

"Preoperative Use of Oral Iodine Solution in Hyperthyroid Patients," presented by Frank C. Au, M.D.

"Experience with Esophagastric Devascularization Procedure at the Hospital of the University of Pennsylvania," presented by Donna Barbot, M.D. and Ernest F. Rosato, M.D.

Regular Meeting
May 6, 1985

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Frederick B. Wagner, Jr., M.D. on Monday, May 6, 1985 at 8:00 p.m.

Scientific Session

"Long-Term Follow-Up for Double Prosthetic Aortic Valve" presented by Robert Mirabile, M.D., Anastasios Pelias, M.D., and Rudolph Camishion, M.D.

"Femoral-Tibial Bypass Grafts: Improved Patency by Early Detection of Graft Stenosis," presented by Henry D. Berkowitz, M.D.

"Preoperative Hemodynamic Monitoring in Patients Undergoing Aortic Surgery," presented by Lorenz Iannarone, M.D., and Dominic DeLaurentis, M.D.

Regular Meeting
October 7, 1985

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Frederick B. Wagner, Jr., M.D., on Monday, October 7, 1985 at 8:00 p.m.

Scientific Session

"Direct Vision of Valvotomy" presented by Teruo Matsumoto, M.D., Ph.D., and discussed by Brooke Roberts, M.D. and Dominic A. DeLaurentis, M.D.

"Broncho-Alvolar Cell Carcinoma of the Lung" presented by Richard Greco, M.D., sponsored by Herbert Cohn, M.D. and Frank John McKeown, M.D.

"Palliative Use of the Nd-YAG Laser in Advanced Upper Gastrointestinal Tract" presented by Howard Z. Zaren, M.D., sponsored by Harvey J. Lerner, M.D.

Regular Meeting
November 4, 1985

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Frederick B. Wagner, Jr., M.D. at 8:00 p.m.

Scientific Session

"A Biologic Motor—One Week Experience" presented by Michael Acker, M.D., sponsored by Larry Stephenson, M.D., and discussed by Pascal M. Spagna, M.D.

"Eight Year Experience with the Ionescu-Shiley Pericardial Valve in the Aortic Position" Presented by Lorenzo Gonzales-Lavin, M.D., sponsored by Charles C. Wolferth, Jr., M.D., and discussed by Eldred Mundth, M.D.

"Primary Gastrointestinal Lymphoma—A Review of 46 Cases" Presented by Ann Carp, M.D. and sponsored by Thomas L. Dent, M.D.

Regular Meeting
December 2, 1985

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Frederick B. Wagner, Jr., M.D. on Monday, November 4, 1985 at 8:00 p.m.

Scientific Session

"Zollinger-Ellison Syndrome, 1986" Presented by Clifford W. Deveney, M.D., sponsored by Brooke Roberts, M.D., and discussed by Francis E. Rosato, M.D.

The Annual Oration—"Surgical Management of Tumors of the Liver" Presented by Francis E. Rosato, M.D.

No Annual Report of the Secretary for 1985 Was Submitted

Regular Meeting
January 6, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Frederick B. Wagner, Jr., M.D. on Monday, January 6th, 1986 at 8:00 p.m.

Scientific Session

"Immediate Endoscopic Placement of the Long Intestinal Tube in Small Bowel Obstruction" Presented by George F. Gowen, M.D., Dominic DeLaurentis, M.D. and Michael Stefan, M.D. (by invitation), and discussed by Thomas Dent, M.D.

"Follow-up of Patients with Colorectal Cancer." Presented by Karen Deveney, M.D. (by invitation from Clyde F. Barker, M.D.), and discussed by Gerald Marks, M.D.

"Primary Hyperparathyroidism in Infancy." Presented by Arthur J. Ross, M.D. (by invitation from Harry C. Bishop, M.D.), Harry C. Bishop, M.D., and Leonard J. Perloff, M.D., and discussed by Clyde F. Barker, M.D.

Regular Meeting
February 3, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Francis Rosato, M.D. on Monday, February 3rd, 1986 at 8:00 p.m.

Scientific Session

"Management of Occlusive Lesions of the Innominate Artery" Presented by Joel Rosenfeld, M.D. (by invitation), Ronald P. Savarese, M.D., and Dominic A. DeLaurentis, M.D. This was discussed by John Y. Templeton, M.D.

"Regional Therapy for Colorectal Hepatic Metastasis." Presented by John Daly, M.D., (by invitation), and sponsored by Clyde F. Barker, M.D. This paper was discussed by Steven Weiss, and Tom Frazier, M.D.

"Acute Appendicitis in Women of Child-Bearing Age" Presented by Karim B. Nakhgevany, M.D. It was discussed by James G. Bassett, M.D., Elmer Grimes, M.D. and Jonathan E. Rhoads, M.D.

Conjoint Meeting with the New York Surgical Society—March 12th, 1986

The annual meeting of the Philadelphia Academy of Surgery and the New York Surgical Society was held at the College of Physicians at 2:00.

Presiding:
Francis E. Rosato, M.D., President, Philadelphia Academy of Surgery

"Experience with Neonatal Extracorporeal Membrane Oxygenation" Philip J. Wolfson, M.D., Discusser: Charles Stolar, M.D.

"The Use of Ultrasound Mammography in the follow-up of Primary Radiation Therapy for Carcinoma of the Breast," Diane R. Gillum, M.D., Thomas G. Frazier, M.D., David Rose, M.D., J. Thomas Murphy, M.D., Discusser: John M. Daly, M.D.

"Rebuilding a Heart with Skeletal Muscle," Larry W. Stephenson, M.D., Michael A. Acker, M.D., John D. Mannion, M.D., Discusser: Henry Spotnitz, M.D.

Presiding: Carlo E. Grossi, M.D., President, New York Surgical Society

"Colonoscopic Decompression: Treatment of Choice for Acute Pseudo-Obstruction of the Colon (OGILVIE's SYNDROME)," Thomas L. Dent, M.D., Discusser: J. P. Morrissey, M.D.

"The Usefulness of Ultrasonic Imaging During Pancreatic Surgery," Bernard Sigel, M.D., Discusser: Dana K. Anderson, M.D.

"Intra-operative Intra-Arterial Thrombolytic Therapy: An Adjunct to Revascularization in Patients with Distal Vessel Thrombosis," Anthony J. Comerota, M.D., John V. White, M.D., Julieta Grosh, M.D., R. Robert Tyson, M.D., Discusser: Thomas S. Riles, M.D.

Regular Meeting
April 7, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by Dominic A. DeLaurentis, M.D., on Monday, April 7, 1986 at 8:00 p.m.

Scientific Session

"Lessons Learned from Venous Valvular Transplantation," Peter McCombs, M.D.

"The Effect of Superoxide Dismutase on Macromolecular Leakage and White Blood Cell Sticking in the Microcirculation of the Hairless Mouse Ear After Ischemia," Richard Bartlett, M.D., (by invitation), sponsored by Paul Nemir, M.D., and discussed by Harvey Rosen, M.D.

"Continent Reconstruction of the Lower Urinary Tract Utilizing the Mitrofanoff Principle," presented by Howard McC Snyder, III, M.D., sponsored by John W. Duckett, M.D., and discussed by Alan Wein, M.D.

Regular Meeting
May 5, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by President Francis Rosato, M.D. on Monday, May 5, 1986.

Scientific Session

"Percutaneous Endoscopic Gastrostomy," presented by Adjit K. Sachdeva, M.D., sponsored by D. R. Cooper, M.D., and discussed by Thomas Dent, M.D.

"Interstital Hyperthermia for Recurrent Tumors" presented by Stephen M.

Weiss, M.D. and discussed by James G. Bassett, M.D., and George Alexander, M.D.

"Prognosis Following Cardiopulmonary Resuscitation for Children with Smoke Inhalation and Burns," presented by Stuart J. Hulnick, M.D., and Charles W. Wagner, M.D., and discussed by Ed Hartford, M.D.

Regular Meeting
October 6, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by Francis Rosato, M.D. on Monday, October 6, 1986 at 8:00 p.m.

Scientific Session

"Sphincter-Preservation Surgery for Cancers of the Distal 6 cm. of Rectum Utilizing Full-Dose Radiation Therapy: A Prospective Study" was presented by Gerald Marks, M.D. and discussed by Robert Goodman, M.D. (by invitation).

"Appendicitis: The Risk of Perforation with Observation" was presented by John R. Clarke, M.D. and discussed by Karim B. Nakhagevany, M.D.

"Use of the Cephalic Vein in Lower Extremity Bypass Surgery" was presented by Erwin A. Cohen, M.D., and discussed by Dominic A. DeLaurentis, M.D.

Regular Meeting
November 3, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by Francis Rosato, M.D., on Monday, November 6th, 1986 at 8:00 p.m.

Scientific Session

"Correlation of CCK Radionuclide with ERCP in Patients with Acalculous Cholecystitis" presened by George F. Gowen, M.D. and discussed by Franz Goldstein, M.D. (by invitation), Ward O. Griffen, M.D., and Henry Moss, M.D.

"Computer Use of a Database in a Surgical Setting," presented by John Dzwonczyk, M.D. and discussed by Herbert Cohn, M.D. and John R. Clarke, M.D.

"The Role of Skeletal Muscle in Shock," presented by Haywood Blum, M.D. (by invitation), Gordon Buzby, M.D., Britton Chance, Ph.D., D.Sc. (by invitation), and discussed by Herbert Cohn, M.D.

Regular Meeting
December 1, 1986

The stated meeting of the Philadelphia Academy of Surgery was called to order by First Vice-President Willis P. Maier, M.D. at 8:00.

Scientific Session

"Combined Hepatic and Gastric Artery Aneurysm and Other Visceral Artery Aneurysms," presented by Michael Weingarten, M.D., sponsored by Tito A. Ranieri, M.D. and discussed by Brooke Roberts, M.D.

ANNUAL ORATION: "The Future of Endothelialized Vascular Grafts" presented by Bruce E. Jarrell, M.D.

Annual Report of the Secretary
1986

There were eight formal meetings of the Philadelphia Academy of Surgery in 1986, seven of which were held in Thompson Auditorium at the College of Physicians of Philadelphia. The Conjoint Meeting was held in Mitchell Auditorium. The average attendance of the regular meetings was 100. The dinner meetings preceding the result meetings were well attended with an average of 75 members.

The Conjoint Meeting with the New York Surgical Society was held in Philadelphia on March 12th. The Scientific Session began at 2:00 p.m., and there were 98 members of the Philadelphia Academy of Surgery and 39 members from the New York group present. Francis Rosato, M.D., and Carlo E. Grossi, M.D., Presidents of the Philadelphia Academy of Surgery and New York Surgical Society respectively, presided over the individual sessions. Six papers presented by the Philadelphia Fellows were discussed by the membership of the New York group. The Philadelphia Academy of Surgery hosted dinner at the Union League of Philadelphia.

Francis Rosato, M.D. appointed a Nominating Committee consisting of Chairman, John Y. Templeton, M.D., along with H. Taylor Caswell, M.D. and Brooke Roberts, M.D. This committee submitted the following slate of officers and council members for 1986:

President—Willis P. Maier, M.D.
1st Vice-President—Dominic A. DeLaurentis, M.D.
2nd Vice-President—Clyde Barker, M.D.
Secretary—David K. Wagner, M.D.
Treasurer—Rudolph C. Camishion, M.D.
Recorder—Wallace P. Ritchie, Jr., M.D., Ph.D.
Chairman, Committee on Scientific Business—Moreye Nusbaum, M.D.
Council-at-Large—Gerald Marks, M.D., Hunter Neal, M.D., Francis Rosato, M.D.

Bruce E. Jarrell, M.D., of Thomas Jefferson University presented the Annual Oration of the Academy on December 1st, 1986. His talk was entitled, "The Future of Epithelialized Vascular Grafts."

Memoirs

ALFRED AYELLA, JR. M.D.
1921–1982

Alfred Ayella, Jr. was born in Philadelphia on March 29, 1921. He attended public school and was graduated from South Philadelphia High School. He matriculated at the University of Pennsylvania in 1938 and graduated in 1942 with an A.B. degree. He entered medical school immediately and was awarded his M.D. in 1945. Thereafter, he served his internship at Hahnemann University Hospital and from 1945 through 1950, his residency in surgery at that same institution.

He was certified by the American Board of Surgery in 1951. In addition to membership in this academy, he held membership in the American College of Surgeons, the College of Physicians, the American Medical Association, the Pennsylvania Medical Society, and the Philadelphia County Medical Society.

He married Agnes Gaffigan January 16, 1951. They had a lovely family of six children, three girls and three boys. Only recently, Dr. Ayella's first grandchild, Alfred Ayella, IV, was born. Unhappily, he didn't live to see him.

He was a member of several hospital staffs. His primary assignments were as a clinical associate professor of surgery from 1970 to the present time at the Hahnemann Medical College, St. Agnes Hospital where he served as a senior attending physician from 1963 to the present, and West Park Hospital where he served as chief of the department of surgery from 1979 to the present time. In his later years, he was extremely active at West Park Hospital working on many committee assignments, and serving as president of the medical board and as a member of the board of trustees.

Dr. Ayella's father was a physician who fostered his son's interest in sport and particularly in boxing.

Early in his career, because of his interest in boxing, he was appointed chief physician and surgeon to the Pennsylvania State Athletic Commission. He served as a member of the medical advisory board for that commission beginning in 1978 until his death.

His interest in boxing led him to study and report on the injuries and trauma associated with that sport.

Dr. Ayella was honored as the recipient of the first annual physician appreciation award presented by West Park Hospital. This award was given to the physician whose professional conduct contributed to the continuation and further development of harmonious working employee relationships at that institution.

In 1978 while swimming his 100 laps a day, he noticed some difficulty breathing. Investigations demonstrated an intensive pleural effusion and subsequently a non-hodgkins lymphoma. He endured his illness without complaint,

achieving a heroic dimension to his life. He did not permit illness to impair his ability to function as a physician, teacher and colleague. His life was the embodiment of the committed, thoughtful, kindly physician. Quietly, on May 23, 1982, Dr. Ayella passed away. It is a privilege to join in tribute to this honored and honorable man—James Bassett M.D.

JOHN V. BLADY, M.D.
1905–1985

John V. Blady died on November 24, 1985 in his sleep of probable cardiac arrhythmia after one of his usual active and busy days. He was born on 12/16/05 in Milwaukee, Wisconsin and received his B.S. degree from the University of Wisconsin. As Wisconsin only offered the first two years of medical school at that time, he was a member of the first graduating class of Duke University Medical School in 1932. As an active alumnus of both medical schools, he interviewed Philadelphia area applicants for many years. After an internship at Duke, he completed residency training in Radiology and Radiation Therapy at Temple University Hospital in 1935 under Dr. W. Edward Chamberlin.

Although for the rest of his medical career his radiologic abilities were a great asset in the management of surgical problems, it was his contact with cancer patients in radiation therapy that inspired him to take a Rockefeller Fellowship in Cancer at Memorial Center for Cancer and Allied Diseases from 1936–1939. The training received was that of the total care of a cancer patient with all the modalities available at the time. Selected cases even received Coley's toxins, perhaps the first chemotherapeutic agent, conceived by William Coley. Surgery was initially done for the complications of and in support of primary treatment with irradiation in the early years of Memorial Hospital. As its limitations became evident, surgery in all the specialities became more important and the attending staff became innovators of surgical techniques that rapidly advanced and improved results in the care of the cancer patient. John Blady was fortunate to have his surgical training at Memorial at this time, and he easily acquired the meticulous techniques of his mentors. Of equal importance was the influence of James Ewing, the tumor pathologist and chief of the hospital. The year after John Blady completed his residency, James Ewing retired. Dr. Blady conceived of the idea of a Memorial Alumni Society and in 1940 with four others, formed the James Ewing Society, named after the father of the multidisciplinary approach to the study and treatment of cancer. Yearly meetings were held until 1948 when the format was changed to a yearly cancer symposium. This was the year when John Blady was president. About ten years ago, the society's membership having expanded to include leaders in the field from other institutions, the name was changed to the Society of Surgical Oncology indicating its broadly-based membership devoted to advances in surgical research and treatment.

In July, 1939, Dr. Blady returned to Temple University Hospital and at the request of Dr. Chamberlin, organized the Department of Radiation Therapy. For the first time in Philadelphia, a long-term follow-up program of cancer patients was started. Records were typed and detailed and are still a valuable resource. Cancers of the Head and Neck were of special interest to him, and he soon confined his practice to this area. He formed the Head and Neck Tumor Clinic and gained successive promotion in the Department of Surgery until 1951

when he was made a clinical professor. Over the years he contributed many innovations in the evolving surgical treatment of tumors of the head and neck area. He was one of the first to recognize the need for reconstructive techniques and rehabilitation of these patients. When Hayes Martin and Grant Ward formed the Society of Head and Neck Surgeons in 1954, he was one of the select few to be chosen as a charter member. A leading radiotherapist who had some of his early training with Dr. Blady told me that he was one of the few oncologists he knew who understood the need to integrate all of the specialties that care for cancer patients in the treatment of a patient's problem. For many years he was a member of the American Radium Society and was its president in 1971. Dr. Blady was one of five founding members who incorporated the Philadelphia Division and the Pennsylvania Division of the American Cancer Society. He was president of the Philadelphia Division in 1959 and 1960 and put into practice the societies' programs of public and professional education by his active chairmanship of the Cancer Control Committee of the County Medical Society from 1947 to 1958.

He served on and was chairman of many other committees of the Philadelphia County Medical Society but none demonstrated his organization, leadership and effectiveness more than his chairmanship of the New Building Committee from 1963–1966. Although the need was great, the effort floundered for many years until his committee gave us our present County Medical Society Building. He was president of the Philadelphia County Medical Society in 1967 and received its prestigious Strittmatter Award in 1985. He was equally active at the State Society level and was president of the Pennsylvania Medical Society in 1977.

His awards include a Certificate of Merit for Distinguished Service in Cancer Control given by the British Government in 1972 and the Bronze Medal from the Chapel of the Four Chaplains in 1984.

He was an avid gardener, stamp and coin collector and a golfer who appreciated every facet of the game and was a delight to have in your foursome. He was president of the Philadelphia Doctors' Golf Association in 1968.

With all his profesional and medical society activities, his family was always foremost. He was a devoted husband to his wife Dee and a supportive and loving father to his children John, Kathryn and Mary Frances. No one more deserves than John Blady the accolade, "Well done, thy good and faithful servant."—Robert Harwick, M.D.

SHERMAN A. EGER, M.D.
1904–1983

Doctor Sherman Alfred Eger died suddenly on May 23, 1983, at the age of 78, from a complication of cardiac disease that had restricted his activities in recent years. He was born in Reading, Pennsylvania, the only child of George and Bertha Eger, originally from McKeesport, and of German descent. As a boy, while working with his father, who was a skilled mechanic, Sherman grabbed a steam pipe and suffered a burn that resulted in a contracture of his right ring finger, fortunately in the position of function, that in no way limited his future skill as a surgeon. It was evident early on that he was academically inclined, for he skipped two grades in school and was always among the top members of his class. His father took great pride in his gifted son, on whose behalf he donated the "Eger Gate" at the entrance to Ursinus College upon his graduation in 1925.

After graduation from Jefferson Medical College in 1929 and serving an internship at the Reading Hospital, he took a residency in surgery at the Crile Clinic, working under the influence of its founder, the elder George Crile. Dr. Eger then returned to Philadelphia and secured a teaching position in the surgical department of his alma mater, at the bottom of the ladder as assistant demonstrator. He became a Fellow of the American College of Surgeons in 1936. While building his private practice he also worked surgically for several insurance companies. From 1941 to 1946 he was the private assistant to Dr. Thomas A. Shallow, the Samuel D. Gross Professor at Jefferson. The teamwork of these two surgeons attracted visitors from other hospitals, some of whom came weekly to observe the latest operative techniques. In those years one could enter the operating room simply by putting a clean gown over street clothes and donning a cap and mask. Operations, participated in by Dr. Eger, were also performed before the combined junior and senior classes in an open amphitheater. He administered many spinal anesthesias, and it was a rare occasion when he did not find the subarachnoid space on the first try. In 1944 Dr. Eger became certified by the American Board of Surgery and in the same year a Fellow of the Philadelphia Academy of Surgery. He regularly attended the meetings of the Academy and especially enjoyed the conjoint meetings with the New York Surgical Society. He retained an interest in Ursinus College and gave talks for some years before one of their science societies to stimulate interest in the medical profession. He was a member of the Jefferson Society for Clinical Investigation, the College of Physicians of Philadelphia, the County and State Medical Societies, and the American Medical Association. In the ensuing years he was author and co-author of many articles on a broad spectrum of surgical disorders, and prepared exhibits for local and national meetings. His particular interest, which had been stimulated initially by Dr. Crile, was in surgery for hypertension by denervation of the adrenal glands. He rose in successive promotions to the rank of Clinical Professor of Sur-

gery and became Honorary in 1976, but retired from active surgical practice at the age of 65.

In his earlier years Dr. Eger enjoyed fishing and was a good golfer. He won a "hole in one" trophy of the old Philadelphia Country Club when it was located in the Fairmount Park area. In later middle life his time was devoted almost solely to surgery and his family. He worked compulsively, was meticulous in dress, always an intellectual, never verbose, and strictly practical. Bluntness in his opinions and dealings with people contrasted with the warm welcome one received in his Bala-Cynwyd home where nothing was too lavish for his guests. He experienced 49 years of happy marriage with his wife, Evelyn, who was active in all affairs of the faculty wives at Jefferson. She died in 1978, after which he noticeably lost his zest for life. They had three daughters, Sarah, Lynn, and Jane, and a son, Sherman, Jr., all of whom are married and have added eight grandchildren.

The Academy of Surgery laments the loss of a polished educator, accomplished surgeon, and loyal Fellow.—Frederick B. Wagner, Jr., M.D.

WILLIAM HENRY MOYER ERB, SR., M.D.
1907–1987

Dr. William Henry Moyer Erb was born in New Berlinville, Pa. on April 23, 1907, the son of Clara Moyer Erb and Harry M. Erb. He attended Secondary School in Boyertown, Pa. He obtained his A.B. degree in 1927 at the University of Pennsylvania as a member of Phi Beta Kappa and his M.D. degree at the School of Medicine in 1930 as a member of Alpha Omega Alpha. He served his internship at HUP 1930–32.

It was during his fulfillment of the Fellowship in Surgery at the Hospital of the University of Pennsylvania that he met a charming intelligent nurse, Sally Smith who became his wife. This marriage brought forth an illustrious son, Dr. William Erb, Jr., a surgeon at the Taylor Hospital, Presbyterian Hospital and Associate in Surgery in the School of Medicine at the University of Pennsylvania. He is associated with Dr. Arthur G. Baker, Jr. and Dr. Dennis N. Cronin. They continue the practice of surgery with the same decorum. Two charming daughters, Patricia Reohr, is a graduate of Cornell University and is a librarian at Strathaven High School, Suzanne Cashin, R.N., a graduate of Catholic University and the Hospital of the University of Pennsylvania. His wife Sally was his constant companion at various meetings, professional and social activities. The entire family participated together as a unit.

His professional surgical training began as a resident 1932–1933 with Dr. Frazier at HUP in charge of Thyroid and Physiology Research and as a fellow with Dr. Eliason 1933 to 1936. Later he continued studying abroad spending nine months in Germany, one month in England, one month in Scotland. He received the Pennsylvania State Board of License in 1932. He was certified by the American Board of Surgery in 1939. While he was taking the practical part in Surgery (3rd part) with Dr. Calvin Smyth as the examiner, he demonstrated his humility and honesty when he remarked to Dr. Smyth that "If you were not here to examine me, I would ask you for your personal advice in the proper management of this difficult and complicated abdominal procedure." Dr. Smyth retorted, "Bill, your doing very well, continue with your technique." Later he became affiliated with Dr. L. K. Ferguson and Dr. Lloyd Stevens at the Presbyterian Hospital and eventually in teaching at the Women's Medical College and his associate, Dr. David Cooper later became the ourstanding Professor at the Medical College of Pennsylvania. Dr. Cooper also served as President of this Academy of Surgery. Dr. Erb was appointed as Chief Surgeon by the Founders Group of the New Riddle Memoria Hospital. In that capacity, he approved the applications of all qualified surgeons to the Staff. His philosophy was that the surgeon which had the three AAA's (Ability, Availability, Affability) would succeed and remain on the staff. During the same period, he served on the staff of the old Philadelphia General Hospital from 1937 to 1974. He was chairman of surgery and in 1965 was elected President of the hospital's medical staff and in

that capacity, he was one of the leaders in the battle to keep the hospital open. He was a member of the American Board of Surgery, a fellow of the American College of Surgeons, President of the Philadelphia Academy of Surgery 1972 to 1974. President of the Delaware County Medical Society in 1960 and President of the Delaware County Cancer Society in 1958. He was a member of Phi Alpha Signa, Phi Beta Kappa and Alpha Omega Alpha.

Dr. Erb was very faithful to his Chief, Dr. Eldridge Eliason and emphasized that "It is necessary to have functional results in a fracture of a bone and not a "Cabinet Makers" reduction. When the clinical findings and the laboratory, X-Ray and other ancillary diagnostic facilities do not agree, it is necessary to repeat both of them which includes re-examination of the patient. Also, never deviate from the rountine in the management of any patient. He became very active in the Alumni of the University of Pennsylvania both in the undergraduate as well as the medical school. I had the privilege to co-author with Dr. Erb and Dr. Dyer the original by-laws, rules and regulations and periodic meetings with the various national medical and surgical conventions. The founders group of this Alumni Society was presided by Dr. Douglas Murphy and Miss Frances Houston.

As a fellow of the Academy of Surgery, he was faithful to all its meetings. He was a member of various committees culminating as its President from 1972 to 1974. He stimulated debate and controversial dialogue. His discussions were well worded and concise. As President, he always recited an historical fact during the dinner preceeding the scientific meeting in which he correlated the past with the present problems in surgery. He gave the Annual Oration 12/5/55 entitled "Peptic Ulcer—Individualization in its management." "It behooves us to guard against following the fashion of the times blindly in the surgical treatment of peptic ulcer. Rather, we should base our treatment on basic physiology, pathology and past experience. Finally, a surgical procedure, no matter how well conceived and carried out, will be a failure if done on a patient in whom it is not indicated." It is gratifying that this Academy of Surgery resolved that the Annual Oration will be titled "The Jonathan E. Rhoads Annual Oration." Dr. Rhoads represents the epitome of American Surgery. Dr. Rhoads was closely associated with Dr. Erb in the Department of Surgery during their training and formative years. This was the period (1930–1940) in which emphasis was placed on making "the patient safe for surgery." The late Dr. I. S. Ravdin and Dr. Harry Vars in the Department of Harrison Research gave nutrition top priority in which Dr. Erb and Dr. Rhoads and the residents in surgery participated. Dr. Max Strumia, the Hemotologist performed the first human blood plasma transfusion in 1934. Later his plasma kit was used by the Armed Forces in World War II.

It was in the latter 19th century when great efforts were made to make "surgery safe for the patient." Prior to that time as demonstrated in the famous portraits by Thomas Eakins of Dr. Samuel D. Gross and Dr. D. Hayes Agnew Clinic, street clothes and bare hands were used in the operating room. It was the efforts of Dr. Lister, Pasteur and Semmelweis who emphasized that bacteria was ubiquitous. Dr. Halsted introduced gloves in surgery for his scrub nurse, Miss Hampden who had developed dermatitis of her hands from the use of phenol.

They realized if a nurse could perform her duties with gloves, why not the surgeon? Dr. J. William White whose name graces the HUP building at the S.W. Corner 34th & Spruce Streets practiced antiseptic and aseptic surgery. Whereas his colleagues were using the Prince Albert Frock Coat in performing the operations upon their patients I have had personal communication with his former residents in surgery namely: Dr. Francis Schumann, Dr. Manrico Troncelitti, Dr. Harry Trachtenberg, Dr. Elmer Grimes and Dr. Brooke Roberts who related the personal interest that Dr. Erb took in them. He made surgical rounds at PGH on Sunday and many times demonstrated compassion for the patients who were very seriously ill. There was no chicanery in his lifestyle. He took the responsibility of his subordinates including the medical and nursing staff. Moreover, his wife Sally often entertained the House Staff of the Hospitals at their private home.

Immediately following the attack of the Japanese on Pearl Harbor, the Surgeon General of the United States mobilized the practicing physicians up to 45 years of age. The University of Pennsylvania organized Base Hospital #20 under the leadership of Dr. I. S. Ravdin and Dr. Thomas FitzHugh as Commanding Officers with a large number of the teaching faculty as well as nurses and technicians to serve. Eventually, they served with distinction in the CBI (China Burma India) theatre of operation. Another hospital unit with full compliment of personnel served in the Medical Corps of the U.S. Navy under the command of Dr. Richard Kern and Dr. L. K. Ferguson. They set a precedent by functioning as a complete hospital unit aboard the U.S.S. Solace Hospital Ship. This ship was involved in the South West Pacific during all the battles from Guadacanal and all the islands which were stepping stones to the return to the Phillipine Islands and Okinawa. As a result of this mobilization, the teaching of the medical students as well as the care of the hospitals affiliated with the U of P were under the guidance of Dr. Eliason, Dr. Erb and Dr. Rhoads. Moreover, the Surgeon General of the U.S. Army organized a contingent of young surgeons to take the course in the Graduate School for a period of six weeks under the leadership of Dr. Calvin Smyth, Dr. W. E. Lee, Dr. H. Hawthorne, Dr. W. Bates and the residual Medical School Faculty including Surgical Anatomy and Basic Science Studies. These men, despite their heavy burden, served with pride on the "Homefront."

Despite Dr. Erb's efforts to insure that the Science and the Art of Medicine adjusted to changes in Social relationship, he kept the common touch. It was a pleasure to watch a skillful and merciful surgeon examine and gently palpate the abdomen of an acutely ill patient. His graciousness, quietness and sympathetic personality won many friends. His affection, kindliness, gentleness in dealing with patients demonstrated his true intimate character. He was a great teacher an investigator of merit and a clinical surgeon with skill, sympathy and warmth toward patients. He was like a good watch: Open-face, pure gold and quietly busy with good works. He led a full life. He was devoted to his family, work and patients. He embodied the quadrat of a good balanced living, Work, Play, Love, Worship. He enjoyed the pleasures of life, good company, music, literature, sports and a good game of cards, bridge. A total of forty-one publications in the

Medical Literature recorded Dr. Erb's work in the laboratory and clinical experience in surgery.

His greatest hobby was horticulture, especially flowers, but hated weeding. During his years of retirement, he visited the Taylor Hospital daily in order to meet old patients and old friends. He brought newspapers and magazine clippings to them. He received the title of "Newsboy" which he thoroughly enjoyed.

On August 7, 1986, he had open heart surgery by Dr. Edmunds, from which he made a satisfactory recovery. He returned to his participation in several groups of retirees including attendance to the monthly meetings of the Academy of Surgery. As he was preparing to attend a session in Bridge, he paused at the landing of his steps in his home and his heart came to rest on January 5, 1987. He died as a swinging gate and not as a rusty one. He left us the heritage: Live and be prepared as to die tomorrow, work and study as to live forever—T. A. Ranieri, M.D.

HERBERT REID HAWTHORNE, M.D.
1894–1981

In the early morning of Sunday, October 4, 1981, Herbert Reid Hawthorne, a Fellow of the College of Physicians for 34 years, passed away at the age of 87 at his home in Bryn Mawr. Dr. Hawthorne was born in Wahoo, Nebraska, in 1894. He attended Central High School in Philadelphia, and in 1919 received his medical degree from the University of Pennsylvania. His postgraduate training in surgery was at the Polyclinic Hospital (later to become the Graduate Hospital), at the Graduate School of Medicine, and at the University of Edinburgh.

Following his return from Edinburgh he was made Instructor in Surgery in the Graduate School of Medicine of the University of Pennsylvania. He was promoted to Professor in 1946 and the following year he succeeded Dr. Walter Estell Lee as chief of a surgical service at the Graduate Hospital. In 1954 he was made Chairman of the Department of Surgery in the Graduate School of Medicine and at the Graduate Hospital, a position he held until his retirement at the age of 65. These were particularly happy and productive years for him.

After retiring as Chairman of the Department of Surgery in the Graduate School of Medicine he accepted a position as consultant to the Veterans Administration. This second phase of his professional career occupied about eight years; even though it necessitated considerable travel up and down the Eastern seaboard and areas in the Western United States, he derived great satisfaction from his continued association with young surgeons and his continued endeavors in teaching. He was not only instrumental in helping to set up programs which assisted in the association between Veterans Administration Hospitals and local medical schools, but he also often taught in the surgical amphitheater.

At the age of 75 he began a third phase of his professional career. He accepted a position as consultant on the University of Pennsylvania Surgical Service at the Veterans Administration Hospital in Philadelphia and he continued as a consultant at the Graduate Hospital. He remained active in these endeavors until just a few months before his death. He received the greatest satisfaction in working with the medical students and residents and this was reciprocated by them.

Dr. Hawthorne was a master surgeon and had a special interest in esophageal and gastrointestinal surgery. He made many contributions to the medical literature and was the author or co-author of more than 80 scientific publications covering a wide variety of subjects. He contributed chapters to numerous textbooks of surgery and gastroenterology and was co-editor of three textbooks concerned with surgery on the gastrointestinal tract and of the vascular system. He was responsible for initiating a strong surgical research program at the Graduate Hospital, and indeed, in the early stages personally supported the effort. He contributed significantly to the educational and training experience of hundreds of

young surgeons from across the United States and from many foreign countries. Because of his warmth, charm, and easy accessibility he was held in great affection by all his students who attended the Graduate School of Medicine and he was usually the first person asked about whenever an alumnus was encountered.

His qualities as a teacher were unusual. He was not a dynamic or electrifying lecturer. He was generally shy and reserved and not given to ad-libbing or to commenting spontaneously. However, he had a special capacity to stimulate the students to ask questions and to seek answers that might easily have been overlooked. He had a special quality of gentleness which applied to his teaching, his attitude and conduct in the operating room and his relationship with colleagues. His greatest legacy was his unfailing optimism and faith in people.

Many honors were bestowed upon him. He was invited to give the Annual Oration at the Philadelphia Academy of Surgery in 1952. The Hawthorne Surgical Society was created in 1955 by his students in the Graduate School of Medicine and his residents at the Graduate Hospital. He was presented an award in recognition of his contributions to surgical education and research by the Association of Veterans Administration Surgeons during the 1974 meeting of the American College of Surgeons. He received the Strittmatter Award of the Philadelphia County Medical Society in 1972. In 1979, at the age of 85, he was awarded the Centennial Medal of the Academy of Surgery of Philadelphia in honor of his devotion to the ideals of that organization.

He was a member of many surgical organizations, among them the American College of Surgeons, the Society for Surgery of the Alimentary Tract, the American Surgical Association, and the International Surgical Society. He was a member of the Sons of the American Revolution, the St. Andrews Society, and the Philadelphia Country Club. He is survived by his wife, the former Grace Briles, his daughter Jessica Hawthorne Contosta, and three grandchildren, all of whom were sources of such great joy and contentment to him. His stately and kindly presence will be greatly missed by this College—Paul Nemir, Jr., M.D.

WILLIAM Y. INOUYE, M.D.
1920–1985

William Y. Inouye was born October 26, 1920 in San Francisco of Japanese parents. He grew up in Sacramento attending the public schools there. At the outset of World War II, because people of Japanese decent living on the West coast were considered a threat to national security, he was interned with his family in Tule, California. A year later he became a student at Swarthmore College graduating in 1944. His plans to attend medical school were delayed by the economic losses suffered by his family during imprisonment. He sought employment which would allow him to save money for medical school and for several years worked in the asbestos industry. This period of enforced delay in his education had far reaching implications, but by 1949 he was able to enter the School of Medicine at the University of Pennsylvania. During his time as a medical student he began working in the laboratory of Dr. Louis Blumele on development of the artificial kidney.

Following graduation from medical school Dr. Inouye interned at the Hospital of the University of Pennsylvania. He then spent two years as Chief of Surgery at Aufrede Hospital in La Rochelle, France. After completion of his military service he returned to residency training at the Hospital of the University of Pennsylvania. During this time he did laboratory research in the Harrison Department of Surgical Research. He published work on a variety of his interests, including the localization of chemotherapeutic agents, intestinal absorption of insulin, localized cerebral hypothermia for protection of the brain during circulatory arrest and cutaneous peritoneal conduits for intermittent peritoneal dialysis. Most significant was his pioneering work on the artificial kidney, which was recognized to be of lasting importance. Dr. Blumele later described him as an investigative genius. Working with Jay Engelberg, Dr. Inouye constructed the first concentrically wrapped coil dialyzer. He made this from screening material and cellulose tubing purchased from a hardware store. The device was enclosed in a standard Presto Pressure Cooker for distribution of the dialyzing fluid and to provide a closed system allowing for ultrafiltration by using negative pressure. Two papers on the device were published, the first in the Surgical Forum in 1953 and the second in the Transactions of the Society for Artificial Internal Organs in 1954. The device was used clinically at the Hospital of the University of Pennsylvania in one of the earliest successful dialyses. During this time Dr. William Kolf visited Dr. Blumele's laboratory and was shown Dr. Inouye's dialysis machine. He became very interested in the device and soon after perfected the widely used twin coil dialyzer, which he is credited with developing. Nevertheless, because the seminal importance of Dr. Inouye's machine was recognized he was awarded the Dialysis Pioneering Award from the National Kidney Foundation. The original machine which was sought by the Smithsonian Institute and is now on loan to the Travenol Traveling Museum on the history of dialysis.

Following his residency training, Dr. Inouye became a member of the staff of the Jeanes Hospital, the American Oncologic Hospital, the Hospital of the University of Pennsylvania and the Philadelphia General Hospital. At the latter institution he was especially active in teaching and following Dr. William Erb's retirement he became the Chief of Surgery for the University of Pennsylvania's service. He held that position until the Philadelphia General Hospital closed in 1975. Following this he became active in teaching at the Philadelphia Veterans Hospital Medical Center and was made the chief of the residency teaching service at the Hospital of the University of Pennsylvania. He rose through the faculty ranks at the School of Medicine at the University of Pennsylvania becoming a full professor of surgery in 1983.

Dr. Inouye was certified by the American Board of Surgery in 1963 and became a Fellow of the American College of Surgeons in 1964 and was a member of the American College of Surgeons, the Philadelphia Academy of Surgery, the Philadelphia County Medical Society, the American Medical Society, the American Society for Artificial Internal Organs, the American Heart Association, Sigma Xi and the American Society of Nephrology.

He continued to be academically active throughout his career and currently has a publication in press with Dr. Addonizio which was delivered at the Philadelphia Academy of Surgery within the last year. He was a superb clinical surgeon conducting his private practice primarily at the Jeanes Hospital where he was also very active in residency training. He also served as the medical director of Friends Hall, a nursing unit for long term illness on the grounds of Jeanes Hospital.

Although outstanding in his early years as a scientist and later as a clinician, it was as an educator that Dr. Inouye particularly excelled. Because of his ability and dedication as a teacher the residents dedicated two teaching awards to him. One of these is the William Y. Inouye Award for Excellence in Teaching which is awarded annually to a surgical resident deemed the best teacher. In addition, an annual award which bears his name is "presented by the chief surgical residents to that member of the surgical faculty whose leadership, caring, attitude and surgical judgement has been an exemplary model for the surgeon in training and with whom a bond of mutual respect and friendship has been established." Dr. Inouye himself won this award twice. The students at Penn were equally responsive to Dr. Inouye's worth as a teacher. They elected him to honorary membership in AOA and dedicated to him a yearbook.

In the fall of 1982, while at the peak of his powers as a surgeon and educator Dr. Inouye developed pleural effusion. This soon led to the diagnosis of mesothelioma which seems certaintly to have stemmed from his exposure to asbestos during the time he was working to earn money for medical school. As his disease progressed his activities were more and more limited. His courage and dedication to teaching allowed him to continue some professional activity and fortunately he considerably outlived predictions based on the usual national history of his disease. Although in considerable pain he continued to make teaching rounds with the residents until a few months of his death. When his physical limitations

precluded this activity he turned his attention to raising money for the residents fund, pursuing this activity to within a few days of his death.

His forced semiretirement had one beneficial effect. He was at last able to devote significant time to his family with whom he traveled extensively, including a long postponed trip to Japan. He also pursued his hobbies which included enjoyment of the solar house (which he and his wife Eleanor had built shortly before his illness) and of their extensive garden and bee hives. He was also able to spend time with his three sons in whose achievements he took great pride, including David who is in the Department of Zoology at the University of Maryland, Robert who practices law in Yacama, Washington and Richard who is an ecologist at the University of Minnesota. His death occurred on July 20, 1985.

Shortly after Bill's death I was surprised to receive a brief note from him, obviously written in anticipation of his impending death, and in it he said ". . . by the time you receive this note I will have passed on to wherever one goes on the great adventure. Life foreshortened by asbestos problems has denied some of the anticipated joys and luxuries of an extended post retirement phase. . . . However I feel it has been my privilege to pursue the active life of a teaching surgeon, working with residents who seemed to grow younger each year, to watch them develop and share with them the ever expanding knowledge of our profession. . . . My best wishes to you, William Y. Inouye."—Clyde F. Barker, M.D.

DAVID Y. P. LIN, M.D.
1919–1983

David Yao Pei Lin has had a very unusual life in a very unusual period of time in human history.

His father and grandfather lived under the feudal rule of CH'ING-MAN-CHUS. Also, they experienced the "Open Door Policy," the Boxer Rebellion as well as the Russo-Japanese War with the Naval Battle at Port Arthur in which the entire sea turned red with human blood shed. Later the period of SUN YAT-SEN in which China became a Republic. Then came World War #1 followed by the Sino-Japanese War with the Rape of Nanking and the subjugation of the Chinese People. They experienced the trials, tribulations and ravages of war. CHIANG KAI-SHEK moved to CHUNGKING as the New Capital. This was the period of the "Flying Tigers" with American Volunteers. Then came the Civil War (1946–1949) with MAO TSE-TUNG as the leader of the PEOPLE'S REPUBLIC OF CHINA. (RED CHINA) on the Main Land with PEIKING as the Capital and CHIANG KAI'-SHEK retreating to TAIWAN (FORMOSA) as the CHINESE NATIONALIST REPUBLIC OF CHINA with TAIPEI as its Capital.

David was born on October 8, 1919 in Nanking, China and was given the Chinese name of Yao Pei Lin which means Far, Cultured, Forest and the Christian name David. He was given that name because his father Peter Lin was far away when he was born being a student in Commerce and Finance in the United State. His mother Priscella Wong was a gracious lady with charm of the Eastern Culture. He has two brothers, Ben Lin, a master of the Culimary Art and an accomplished pianist as well as a respected realtor. Henry Lin is also an accomplished musician with the cello. He is employed by Bell Telephone Company in Wilmington, Delaware. Thus, music is the best tranquilizer that one can offer to the people.

Dr. Lin received a B.S. degree from St. John's University in Shanghai. This University was established by the University of Pennsylvania and the Dean of the School was Dr. Joshia E. McCracken.

During the occupation of Shanghai by the Japanese the Chinese students including David Lin removed many books and vital documents to Chengtu in the interior of China. The journey of many miles was made by foot.

He attended the West China Union Medical College and received his M.D. degree in 1946. This is the same insitution where Dr. Carl F. Schmidt, Professor of Pharmacology at the University of Pennsylvania performed research on the native herbal plants used in Chinese medical practice. In 1922 he discovered that MaHuang had sympathomimetic qualities and the name of Ephedrine was coined to the by-product of this natural herb.

In 1946 Dr. Lin emmigrated to Philadelphia and attended the University of Pennsylvania Graduate School of Medicine. Later he earned a Master of Medical

Science for a thesis on non-calculous cholecystitis. Dr. Calvin Smyth, Dr. Edward Closson and myself sponsored him for the Internship at the Methodist Hospital as well as Resident-in-Surgery 1947–1951. The year of 1952 he was Chief Resident. At that time, the State of Pennsylvania did not recognize any student from foreign medical schools. Therefore, he was unable to qualify for the State Medical Boards despite his post-graduate training. Also, he went to Canada and passed its National Boards. Later he passed the American National Boards and also became a Diplomate of the American Board of Surgery.

Dr. Lin served with the Medical Corps of the U.S. Army with the rank of Captain during the Korean War. In 1953, he was Chief of Surgery of the 21st Evacuation Hospital, Seoul, Korea. For his undefatigable work, he received the Bronze Star and the Korean Presidential Citation. He jeopardized his life as a Native of China he would be severely punished if he fell in the hands of the enemy. He demonstrated courage and demonstrated his love for his adopted country USA. In 1954, he served at Fort Stewart Army Hospital and 1955 at Fort Dix Hospital.

Following his honorable discharge from the Military Service, Governor George Leader and Dr. Max Strumia as a member of the Pennsylvania State Boards of Medical Education and Licensure were able to change the rules and regulations so that fair high standards were outlined in order to meet the qualifications for foreign graduates to take the examinations. Dr. Lin became the first foreigner to qualify and pass the State Boards.

With this milestone, he was appointed to the Surgical Staff of the Methodist Hospital. Also, he became the Anesthesiologist for Thoracic Surgery performed weekly by Dr. George Willauer at Eagleville Sanatorium. He quickly demonstrated his skill in surgery and rendered unselfish service to his patients and to the community. He participated in the teaching program of the Jefferson Medical Students as well as the Nursing Classes. He was so endeared to the students that the Nurses' Year Book was dedicated to him.

Also he participated in organized medicine becoming a member of the Philadelphia County Medical Society, Pennsylvania Medical Society, American Medical Association, Fellow of American College of Surgeons, Philadelphia College of Physicians, Philadelphia Academy of Surgery (1961), President of the Phila. County Medical Society South Branch 1963–1964, Board of Directors of Phila. County Medical Society 1964–1966, member of the Lion's Club.

Dr. Robert E. Berry became associated with Dr. Lin in the practice of Surgery at the Methodist Hospital. Eventually Dr. Berry, his wife Margy and family went as Medical Missionaries to Katmandu, the Capital of Nepal. He set up a modern hospital and performed the first open-heart surgery in that area. He had the privilege to render medical assistance to the conquerors of Mt. Everest. Following his mission in Nepal-Tibet, Dr. Berry returned to Roanoke Memorial Hospital as Director of Surgical Education and Professor of Surgery at the University of Virginia. He has the same mannerism inspired by Dr. Lin.

Dr. Ronald S. L. Jan, a native of Taiwan well trained in surgery became associated with Dr. Lin. He is pursuing with the same paintaking care of his patients.

Dr. Lin was tireless in his efforts to provide for his patients. He was a Master Surgeon, dedicated physician, a remarkable teacher making learning fun. He was highly motivated and energetic. Often he cared more for others than for himself. While at the peak of life and his practice of surgery, he became interested in a religious sect in the late "60's," known as the "Body of Christ" and decided to join its work. In 1973, he retired from practice and moved to the Sect's "log cabin" community in the Providence of British Columbia in Canada. They cleared the wilderness in the cold, bearing sub-freezing temperatures on their own.

In giving David a "farewell" dinner with a donation of a Power-Saw, his medical colleagues urged him not to give up his practice. However, he was not influenced. He felt it was more rewarding "to heal the soul than to cure the body"; realizing the mortality of man. He combined knowledge and wisdom.

His entire family moved West along with a handful of people to begin their new life from scratch, clearing the wilderness to build their community. Thus, they began to reap nature's reward with their own hands.

In 1977, David returned to Philadelphia in order to give personal care to his aged father inflicted with Alzheimer's Disease, while his family remained with the community. The philosophy of the oriental is that an old individual is venerable and it is an honor to respect and care for the aged. Following his father's death in 1980, he severed his "Philadelphia Roots" forever and returned to the Community in Canada where his two sons, Lester and Alan are continuing the Lord's work in that natural environment. His daughter Vanessa and her husband are Christian Missionaries in Columbia, South America.

His wife Susan Hsueh Lin who is a most hospitable, gracious, cultured lady and an accomplished concert pianist joined him in Canada. Later both of them went to the Far East to do ministry work. They traveled and preached in Malaysia, Indonesia, Singapore, Honk-Kong and Taiwan. David was following his grandfather's footsteps as he was one of China's earliest Christian Ministers.

Dr. Lin informed his wife that "Medical Knowledge is given by God as part of his great mercy towards mankind in reducing suffering." Through his preaching and interpreting the Bible, he was able to demonstrate the mending and patching of one's body and soul. His parable "Like a woman with her mending basket, the work never ends."

On his last journey in HongKong, he was informed of a desperate need for a Surgeon in a Christian Mission Hospital in Southern Taiwan. As a man with compassion in time of adversity, he volunteered to serve; however, he was unable to fulfill that assignment as he met his Maker on January 10, 1983 with an Acute Coronary Attack and its sequela. From Taipei, Taiwan, Dr. Lin's body was transferred to British Columbia in Canada for interment in the soil of his community where he lies in peace amongst his co-workers.

Dr. Lin practiced what he preached. His life was a quadrate and well-balanced: Work, Play, Love, Worship. His fulfilment in life was the legacy of St. Augustine: "Thou hast created us O God and our hearts are restless till they rest in Thee." As a believer of body and soul, he left us the heritage: Live and be prepared to die tomorrow, work and learn as to live forever—T.A. Ranieri, M.D.

ALEXANDER ULIN, M.D.
1913–1980

Dr. Alexander Ulin died Monday, December 15, 1980 following an attack of coronary thrombosis. He was born and grew up in Philadelphia. He graduated from Central High School and both the Undergraduate and Medical Schools of the University of Pennsylvania. After interning at the Hospital of the University of Pennsylvania he started his residency at the University of Cincinnati under Mont Reed. His residency was interrupted by service in the Army during the Second World War which was largely spent in India. Following this period of military duty he completed his residency at Hahnemann Hospital where he was subsequently appointed to the faculty and rose in the ensuing years to the rank of Professor. His service at Hahnemann Medical College was marked by outstanding achievement as a teacher and investigator. As founder and advisor to the Undergraduate Research Group many students were influenced to undertake academic careers, and he was awarded the Lindback Award for excellence in teaching. During his lifetime, he published over one hundred scientific papers on diverse subjects. From 1956 to 1959 he was Chairman of the Department of Surgery at the Southern Division of the Einstein Medical Center and in 1955 was appointed Consultant in Surgery to the Philadelphia Veterans Hospital. At the time of his death he was Chief of Surgery at Rolling Hill Hospital. He was a member of many societies including Phi Beta Kappa, Alpha Omega Alpha, College of Surgeons, College of Physicians, The Philadelphia Academy of Surgery. He was a devoted husband and father and was survived by his wife and three sons—Paul Grotzinger, M.D.

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