Transactions of the

Philadelphia Academy of Surgery

VOLUME XXXV

1987–1992

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NOTICE

The thirty-fifth volume of the TRANSACTIONS OF THE PHILADELPHIA ACADEMY OF SURGERY covers the six years from 1987-1992 inclusive.

Anthony J. DelRossi, M.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 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 candidate must be a Doctor of Medicine who has graduated from a reputable School of Medicine at least ten (10) years before proposal. The candidate 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 the candidate's name may be presented to the Academy as a candidate for election. The candidate 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 Fellowship by presenting at a stated meeting a communication to that effect, with the Treasurer's certificate that the Fellow 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 (2) 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 the duty of the President 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 the office. The Vice-Presidents shall assist the President in the discharge of functions, and in the absence of the President preside in the order of seniority.

SECTION V

SECRETARY

The Secretary shall keep the minutes of the meetings of the Academy, one (1) copy of which shall be sent to the Recorder. The Secretary 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, keep in 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; 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, and shall receive copies of the Annual Oration, maintain the Archives of the Academy, including copies of the Minutes, and consult with Fellows who present Annual Orations and Memoirs before the Academy in regard to publication. The Recorder 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 it 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 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 (3) 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 will have demonstrated potential for making contributions to the various programs of the Academy with a minimum of three (3) 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 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 payment of the initiation fee is made and the Constitution and By-Laws signed by the third meeting following 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 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 dues within two (2) months thereafter, shall forfeit 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 person 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 the 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.

List of Officers, 1992

President
MOREYE NUSBAUM, M.D.

First Vice-President
ROBERT D. HARWICK, M.D.

Second Vice-President
WALLACE P. RITCHIE, JR., M.D.

Secretary
STEPHEN WEISS, M.D.

Treasurer
THOMAS DENT, M.D.

Recorder
PASCHAL SPAGNA, M.D.*
ANTHONY J. DELROSSI, M.D.

Chairman, Program Committee
ANTHONY J. COMEROTA, M.D.

Member-at-Large
ANTHONY J. DELROSSI, M.D.

Samuel D. Gross Prize Fund
WARD O. GRIFFEN, JR., M.D.,
Chairman

**Deceased*

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		ELECTED	
1880	SAMUEL D. GROSS	1948	THOMAS A. SHALLOW
1884	D. HAYES AGNEW	1950	CALVIN M. SMYTH
1891	WILLIAM HUNT	1952	I. S. RAVDIN
1895	THOMAS G. MORTON	1954	L. K. FERGUSON
1898	DEFOREST WILLARD	1956	JOHN GIBBON, JR.
1902	RICHARD H. HARTE	1958	ADOLPH WALKLING
1904	HENRY R. WHARTON	1960	W. EMORY BURNETT
1906	JOHN B. ROBERTS	1962	J. MONTGOMERY DEAVER
1908	WILLIAM J. TAYLOR	1964	JONATHAN E. RHOADS
1910	ROBERT G. LECONTE	1965	GEORGE J. WILLAUER
1912	GWILYM G. DAVIS	1967	GEORGE P. ROSEMOND
1914	JOHN H. GIBBON	1970	JULIAN JOHNSON
1916	CHARLES H. FRAZIER	1972	WILLIAM H. ERB
1918	EDWARD MARTIN	1974	JOHN Y. TEMPLETON, III
1920	GEORGE G. ROSS	1976	H. TAYLOR CASWELL
1922	JOHN H. JOPSON	1978	DONALD R. COOPER
1924	EDWARD B. HODGE	1980	BROOKE ROBERTS
1926	CHARLES F. MITCHELL	1982	PAUL NEMIR, JR.
1928	ASTLEY P. C. ASHHURST	1983	R. ROBERT TYSON
1930	GEORGE P. MULLER	1984	CHARLES C. WOLFERTH, JR.
1932	JOHN SPEESE	1985	FREDERICK B. WAGNER, JR.
1934	WALTER ESTELL LEE	1986	FRANCIS E. ROSATO
1936	DAMON B. PFEIFFER	1987	WILLIS P. MAIER
1938	J. STEWART RODMAN	1988	DOMINICK A. DELAURENTIS
1940	ELDRIDGE L. ELIASON	1989	CLYDE BARKER
1942	ROBERT H. IVY	1990	RUDOLPH C. CAMISHION
1944	HUBLEY R. OWEN	1991	DAVID K. WAGNER
1946	JOHN B. FLICK	1992	MOREYE NUSBAUM

VICE-PRESIDENT

ELECTED		ELECTED	
1880	D. HAYES AGNEW	1938	ROBERT H. IVY
1880	R. J. LEVIS	1940	HUBLEY R. OWEN
1884	SAMUEL W. GROSS	1942	JOHN B. FLICK
1889	JOHN H. PACKARD	1943	THOMAS A. SHALLOW
1891	WILLIAM W. KEEN	1945	CALVIN M. SMYTH
1891	J. EWING MEARS	1948	L. KRAEER FERGUSON
1898	JOHN ASHHURST, JR.	1950	I. S. RAVDIN
1900	RICHARD H. HARTE	1952	L. K. FERGUSON
1900	HENRY R. WHARTON	1954	JOHN H. GIBBON, JR.
1902	JOHN B. DEAVER	1956	ADOLPH WALKLING
1904	JOHN B. ROBERTS	1958	W. EMORY BURNETT
1905	WILLIAM J. TAYLOR	1960	J. MONTGOMERY DEAVER
1906	ROBERT G. LECONTE	1962	JONATHAN E. RHOADS
1908	G. G. DAVIS	1964	GEORGE J. WILLAUER
1910	JOHN H. GIBBON	1965	GEORGE P. ROSEMOND
1912	CHARLES H. FRAZIER	1967	JULIAN JOHNSON
1914	EDWARD MARTIN	1976	DONALD R. COOPER
1916	GEORGE G. ROSS	1978	BROOKE ROBERTS
1918	JOHN H. JOPSON	1980	WILLIAM T. FITTS, JR.
1919	H. C. DEAVER	1981	PAUL NEMIR, JR.
1920	JOHN H. JOPSON	1982	R. ROBERT TYSON
1920	EDWARD B. HODGE	1983	CHARLES C. WOLFERTH, JR.
1922	CHARLES F. MITCHELL	1984	FREDERICK B. WAGNER, JR.
1924	ASTLEY P. C. ASHHURST	1985	FRANCIS ROSATO
1926	ASTLEY P. C. ASHHURST	1986	WILLIS P. MAIER
1926	GEORGE P. MULLER	1987	DOMINICK A. DELAURENTIS
1928	JOHN SPEESE	1988	CLYDE BARKER
1930	WALTER ESTELL LEE	1989	RUDOLPH C. CAMISHION
1932	DAMON B. PFEIFFER	1990	DAVID K. WAGNER
1934	J. STEWART RODMAN	1991	MOREYE NUSBAUM
1936	E. J. KLOPP	1992	ROBERT HARWICK
1938	ELDRIDGE L. ELIASON		

SECRETARY

ELECTED		ELECTED	
1880	J. EWING MEARS	1915	GEORGE P. MULLER
1885	J. HENRY C. SIMES	1920	J. STEWART RODMAN
1893	THOMAS R. NEILSON	1922	HUBLEY R. OWEN
1896	WILLIAM J. TAYLOR	1930	DEFOREST P. WILLARD
1905	JOHN H. GIBBON	1935	HENRY P. BROWN, JR.
1909	CHARLES F. MITCHELL	1940	JOHN B. FLICK

SECRETARY *(cont.)*

ELECTED		ELECTED	
1942	L. KRAEER FERGUSON	1982	FREDERICK B. WAGNER, JR.
1943	CALVIN M. SMYTH	1983	JAMES G. BASSETT
1945	L. KRAEER FERGUSON	1984	JAMES G. BASSETT
1948	J. MONTGOMERY DEAVER	1985	DAVID K. WAGNER
1958	WILLIAM B. FITTS	1986	DAVID K. WAGNER
1960	HENRY P. ROYSTER	1987	DAVID K. WAGNER
1964	THOMAS F. NEALON	1988	DAVID K. WAGNER
1967	DONALD R. COOPER	1989	STEPHEN WEISS
1974	PAUL NEMIR, JR.	1990	STEPHEN WEISS
1980	FREDERICK B. WAGNER, SR.	1991	STEPHEN WEISS
1981	FREDERICK B. WAGNER, JR.	1992	STEPHEN WEISS

TREASURER

ELECTED		ELECTED	
1880	WILLIAM HUNT	1981	CHARLES C. WOLFERTH, JR.
1891	WILLIAM G. PORTER	1982	WILLIS P. MAIER
1904	JAMES P. HUTCHINSON	1983	WILLIS P. MAIER
1911	EDWARD B. HODGE	1984	WILLIS P. MAIER
1920	DUNCAN L. DESPARD	1985	RUDOLPH C. CAMISHION
1922	WILLIAM B. SWARTLEY	1986	RUDOLPH C. CAMISHION
1935	L. KRAEER FERGUSON	1987	RUDOLPH C. CAMISHION
1938	HARRY E. KNOX	1988	ROBERT HARWICK
1947	S. DANA WEEDER	1989	ROBERT HARWICK
1960	ORVILLE C. KING	1990	ROBERT HARWICK
1965	EDWIN W. SHEARBURN	1991	THOMAS DENT
1974	WILLIAM T. FITTS, JR.	1992	THOMAS DENT
1980	CHARLES C. WOLFERTH, JR.		

RECORDER

ELECTED		ELECTED	
1880	JOHN B. ROBERTS	1937	ADOLPH A. WALKLING
1881	DEFOREST WILLARD	1950	JONATHAN E. RHOADS
1884	C. B. G. DENANCREDE	1952	W. EMORY BURNETT
1884	J. EWING MEARS	1956	FREDERICK A. BOTHE
1891	LEWIS W. STEINBACH	1960	H. TAYLOR CASWELL
1902	JOHN H. GIBBON	1966	WILLIAM S. BLAKEMORE
1905	JOHN H. JOPSON	1974	EDWIN W. SHEARBURN
1915	JOHN SPEESE	1976	JOSEPH W. STAYMAN
1920	HENRY P. BROWN, JR.	1980	ELMER L. GRIMES
1922	J. WILLIAM BRANSFIELD	1981	ELMER L. GRIMES
1926	CALVIN M. SMYTH, JR.	1982	ELMER L. GRIMES

RECORDER *(cont.)*

ELECTED		ELECTED	
1983	ELMER L. GRIMES	1989	WALLACE P. RITCHIE, JR.
1984	DOMINICK A. DELAURENTIS	1990	WALLACE P. RITCHIE, JR.
1985	DOMINICK A. DELAURENTIS	1991	WALLACE P. RITCHIE, JR.
1986	WALLACE P. RITCHIE, JR.	1992	PASCHAL SPAGNA*
1987	WALLACE P. RITCHIE, JR.	1992	ANTHONY J. DELROSSI
1988	WALLACE P. RITCHIE, JR.		

**Deceased*

COUNCIL

ELECTED		ELECTED	
1880	JOHN ASHHURST, JR.	1948	FRANCIS C. GRANT
1880	JOHN H. BRINTON	1950	THOMAS A. SHALLOW
1894	WILLIAM B. HOPKINS	1952	ADOLPH WALKLING
1895	HENRY R. WHARTON	1952	CALVIN M. SMYTH
1898	THOMAS R. NEILSON	1954	I. S. RAVDIN
1900	W. JOSEPH HEARN	1954	FREDERICK A. BOTHE
1902	ROBERT G. LECONTE	1956	FREDERICK ROBBINS
1906	THOMAS R. NEILSON	1956	L. KRAEER FERGUSON
1910	J. CHALMERS DE COSTA	1957	FREDERICK ROBBINS
1920	CHARLES F. MITCHELL	1958	JOHN H. GIBBON, JR.
1922	GEORGE G. ROSS	1959	ORVILLE C. KING
1922	JAMES H. BALDWIN	1960	ADOLPH WALKLING
1923	WILLIAM J. TAYLOR	1960	JONATHAN E. RHOADS
1924	JOHN H. JOPSON	1962	DONALD K. COOPER
1924	JOHN SPEESE	1962	W. EMORY BURNETT
1925	EDWARD B. HODGE	1964	J. MONTGOMERY DEAVER
1926	DAMON B. PFEIFFER	1965	JONATHAN E. RHOADS
1927	CHARLES F. MITCHELL	1967	JOHN Y. TEMPLETON
1930	ASTLEY C. ASHHURST	1967	GEORGE WILLAUER
1930	HUBLEY R. OWEN	1974	WILLIAM H. ERB
1932	GEORGE P. MULLER	1974	CHARLES C. WOLFERTH, JR.
1935	DEFOREST P. WILLARD	1974	JOSEPH W. STAYMAN, JR.
1936	WALTER ESTELL LEE	1976	JOHN Y. TEMPLETON, III
1936	ROBERT H. IVY	1976	CHARLES C. WOLFERTH, JR.
1940	J. STEWART RODMAN	1976	R. ROBERT TYSON
1940	DAMON B. PFEIFFER	1978	H. TAYLOR CASWELL
1941	EDWARD B. HODGE	1978	ELMER L. GRIMES
1942	THOMAS A. SHALLOW	1978	FREDERICK B. WAGNER, JR.
1942	EDLRIDGE L. ELIASON	1980	DONALD R. COOPER
1943	ROBERT H. IVY	1980	WILLIS B. MAIER
1946	HUBLEY R. OWEN	1980	FRANCIS E. ROSATO
1947	CHARLES F. MITCHELL	1981	DONALD COOPER

COUNCIL *(cont.)*

ELECTED		ELECTED	
1981	WILLIS P. MAIER	1986	HUNTER NEAL
1981	JAMES BASSETT	1987	GERALD MARKS
1982	BROOKE ROBERTS	1987	HUNTER NEAL
1982	JAMES BASSETT	1987	FRANCIS E. ROSATO
1982	HARRY V. ARMITAGE	1988	GERALD MARKS
1983	PAUL NEMIR, JR.	1988	THOMAS DENT
1983	HARRY V. ARMITAGE	1989	DOMINICK A. DELAURENTIS
1983	WILLIAM STAINBACK	1989	THOMAS DENT
1984	R. ROBERT TYSON	1989	GERALD MARKS
1984	WILLIAM STAINBACK	1990	CLYDE BARKER
1984	CLIFTON F. WEST, JR.	1990	PASCHAL SPAGNA
1985	CHARLES C. WOLFERTH, JR.	1990	THOMAS DENT
1985	CLIFTON F. WEST, JR.	1991	JOHN M. DALY
1985	MOREYE NUSBAUM	1991	PASCHAL SPAGNA
1986	FREDERICK B. WAGNER, JR.	1991	RUDOLPH C. CAMISHION
1986	MOREYE NUSBAUM	1992	ANTHONY J. DELROSSI

With President, Vice-President, Secretary and Treasurer

BUSINESS COMMITTEE

ELECTED		ELECTED	
1895	WILLIAM J. TAYLOR	1931	HENRY P. BROWN, JR.
1895	DEFOREST WILLARD	1932	EDWARD T. CROSSAN
1896	RICHARD H. HARTE	1935	B. FRANKLIN BUZBY
1897	ROBERT G. LECONTE	1936	JOHN B. FLICK
1900	G. G. DAVIS	1938	L. KRAEER FERGUSON
1902	JOHN H. JOPSON	1940	J. MONTGOMERY DEAVER
1905	GEORGE G. ROSS	1942	CALVIN M. SMYTH
1908	FRANCIS T. STEWART	1943	FREDERICK A. BOTHE
1914	JOHN SPEESE	1943	W. EMORY BURNETT
1916	WALTER ESTELL LEE	1944	ADOLPH A. WALKLING
1916	MORRIS BOOTH MILLER	1946	J. MONTGOMERY DEAVER
1917	DAMON B. PFEIFFER	1949	FREDERICK A. BOTHE
1917	ASTLEY P. C. ASHHURST	1950	JOHN H. GIBBON, JR.
1919	A. BRUCE GILL	1950	JONATHAN E. RHOADS
1919	J. STEWART RODMAN	1951	FRANK ALLBRITTEN, JR.
1920	ARTHUR BILLINGS	1954	EDWIN W. SHEARBURN
1922	DAMON B. PFEIFFER	1960	JOHN Y. TEMPLETON, III
1924	DEFOREST P. WILLARD	1964	BROOKE ROBERTS
1928	WALTER ESTELL LEE	1974	BROOKE ROBERTS
1930	EDWARD T. CROSSAN	1978	R. ROBERT TYSON
1930	JOHN B. FLICK		

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

1988
WARD O. GRIFFEN, JR.

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

ELECTED		DIED
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
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	1992
1989	MICHAEL DEBAKEY, Houston, Tex.	

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 Inîluenced 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.

1987 "The Hemodynamics of Post-Shunt Neurological Deterioration: Analysis and Clinical Trial." —Dr. Kaj Johansen, University of Washington School of Medicine, Seattle, Wash.

1992 "Adventitial Elastolysis and Abdominal Aortic Aneurysm Formation." —Dr. John V. White, Temple University, Philadelphia, Pa.

Fellows Who Have Delivered the Annual Oration

1881 S. D. Gross
1882 D. Hayes Agnew
1883 William Hunt
1884 John H. Brinton
1885 John H. Packard
1886 R. J. Levis
1887 J. Ewing Mears
1888 C. B. G. deNancrede
1889 John B. Roberts
1890 DeForest P. Willard
1891 William G. Porter
1892 T. G. Morton
1893 C. W. Dulles
1894 W. B. Hopkins
1895 John B. Deaver
1896 James M. Barton
1897 Thomas R. Neilson
1898 O. H. Allis
1899 William J. Taylor
1900 None
1901 H. R. Wharton
1902 J. M. Spellissy
1903 R. G. LeConte
1904 G. G. Davis
1905 J. Chalmers DaCosta
1906 Richard H. Harte
1907 Edward Martin
1908 Charles H. Frazier
1909 John H. Gibbon
1910 Astley P. C. Ashhurst
1911 John H. Jopson
1912 George C. Ross
1913 William L. Rodman
1914 Alfred C. Wood
1915 Frances T. Stewart
1916 Edward B. Hodge
1917 J. Edwin Sweet
1918 None
1919 None
1920 John G. Clark
1921 J. Torrance Rugh
1922 George P. Muller
1923 Walter Estell Lee
1924 Robert H. Ivy
1925 John Speese
1926 Damon B. Pfeiffer
1927 Emory G. Alexander
1928 Edward J. Klopp
1929 Edward T. Crossan
1930 J. Stewart Rodman
1931 Hubley R. Owen
1932 Eldridge L. Eliason
1933 George M. Dorrance
1934 DeForest P. Willard
1935 A. Bruce Gill
1936 Alexander Randall
1937 Henry P. Brown, Jr.
1938 Isidor S. Ravdin
1939 John B. Flick
1940 Francis C. Grant
1941 William Bates
1942 S. Dana Weeder
1943 Frederick A. Bothe
1944 Calvin M. Smyth
1945 Adolph A. Walkling
1946 John H. Gibbon, Jr.
1947 L. Kraeer Ferguson
1948 Jonathan E. Rhoads
1949 Francis C. Grant
1950 W. Emory Burnett
1951 J. Montgomery Deaver
1952 Herbert R. Hawthorne
1953 Julian Johnson
1954 George Rosemond
1955 William H. Erb
1956 George Willauer
1957 Irvin E. Deibert
1958 Orville C. King
1959 James R. Jaeger
1960 H. Taylor Caswell
1961 Donald R. Cooper
1962 John Y. Templeton, III
1963 Edwin W. Shearburn
1964 Henry P. Royster
1965 C. Everett Koop
1966 Kenneth E. Fry
1967 Thomas F. Nealon, Jr.
1968 R. Robert Tyson
1969 H. L. Stahlgren
1970 Brooke Roberts
1971 William T. Fitts, Jr.
1972 Joseph G. Bassett
1973 Lloyd W. Stevens
1974 Joseph W. Stayman
1975 Charles Fineberg
1976 Leonard Goldman
1978 David Wagner
1979 Frederick Wagner
1980 Clyde Barker
1981 Moreye Nusbaum
1982 Michael O'Conner
1983 Jack Kolff
1984 Simon Simonian
1985 Francis E. Rosato
1986 Bruce E. Jarrell
1987 Robert D. Harwick
1988 Rudolph C. Camishion
1989 Wallace P. Ritchie, Jr.
1990 Dominick A. DeLaurentis
1991 John M. Daly
1992 James L. Weese

Fellows of the Philadelphia Academy of Surgery

Active Fellows

	ELECTED	BORN	SPECIALTY
Alexander, James B., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	2-4-91	1954	G.S.
Asensio, Juan Hahnemann University Hospital Broad & Vine Streets, Mail Stop 414 Philadelphia, PA 19102-1192	5-6-91	1954	G.S.
Au, Francis C., M.D. 3401 N. Broad Street Philadelphia, PA 19140	1-3-83	1943	G.S.
Badosa, Francisco, M.D. 5401 Old York Road, Suite 505 Philadelphia, PA 19141	11-5-90	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 Street & 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.
Barot, Lenora R., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	12-3-90	1950	G.S.
Berkowitz, Henry D., M.D. Presbyterian Medical Center 39th & Market Streets 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	Orthopaedics
Boova, Robert S., M.D. 101 Bryn Mawr Avenue, Suite 135 Bryn Mawr, PA 19010	11-5-90	1951	Thoracic
Brooks, Clint, M.D. 2100 Keystone Avenue, Suite 305 Drexel Hill, PA 19026	11-1-82	1932	G.S., Vascular
Brown, Arthur S., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	11-5-84	1944	Plastics

	ELECTED	BORN	SPECIALTY
Bucheit, William A., M.D. 3401 N. Broad Street Philadelphia, PA 19140	1-6-75	1933	Neurosurgery
Busby, Gordon, M.D. 3400 Spruce Street Philadelphia, PA 19104	1983	1948	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	1-1-79	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., Vascular
Cossa, John P., M.D. P.O. Box 1428 Rabbitt Run Road Malvern, PA 19355	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.
DelRossi, Anthony J., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	11-6-89	1943	G.S., Thoracic, Vascular
Dempsey, Daniel T. Temple University Hospital Broad & Ontario Streets Philadelphia, PA 19140	5-6-91	1953	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.
DiSesa, Verdi J. Hospital of the University of Pennsylvania 3400 Spruce Street Philadelphia, PA 19104	11-4-91	1950	G.S., Thoracic

	ELECTED	BORN	SPECIALTY
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
Edie, Richard N., M.D. Jefferson Medical College 1025 Walnut Street, Suite 607 Philadelphia, PA 19107	12-5-84	1937	Thoracic
Ehrlich, Frank, M.D. The Graduate Hospital 1800 Lombard Street Pepper Pavilion, Suite 1101 Philadelphia, PA 19146	12-4-89	1939	G.S., Pediatric
Eisenberg, Burton L., M.D. 7701 Burholme Avenue Philadelphia, PA 19111	2-4-91	1948	G.S.
Fallahnejad, Manucher, M.D. Room 1004, The Graduate Hospital 19th & Lombard Streets Philadelphia, PA 19146	10-6-75	1936	G.S., Thoracic, Cardiovascular
Finnegan, James O., M.D. Crozer-Chester Medical Center Professional Office Building #1, Suite 301 15th & Upland Streets Upland, PA 19013-3995	11-4-74	1938	G.S., Thoracic
Frazier, Thomas G., M.D. 101 Bryn Mawr Avenue Bryn Mawr, PA 19011	2-5-79	1943	G.S.
Gain, Thomas B., M.D. 230 N. Broad Street Philadelphia, PA 19102	12-5-77	1942	G.S.
Goldman, Scott, M.D. Suite 558, Lankenau Medical Building East 100 Lancaster Avenue, West of City Line Wynnewood, PA 19096	11-5-90	1950	Thoracic
Grosh, Julieta D., M.D. Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	10-6-80	1939	G.S.
Haith, Linwood R. Crozer-Chester Medical Center Burn Care Associates, Suite 206 Upland, PA 19013-3995	12-2-91	1950	Burn
Hamilton, Ralph W., M.D. 3400 Spruce Street Philadelphia, PA 19104	11-6-78	1933	Plastics

	ELECTED	BORN	SPECIALTY
Hargrove, W. Clark, III, M.D. Presbyterian Medical Center of Philadelphia Medical Office Building, Suite 140 39th & Market Streets Philadelphia, PA 19104	1-8-90	1947	Cardiothoracic
Kerstein, Morris D., M.D. Hahnemann University Broad & Vine Streets, Mail Stop 413 Philadelphia, PA 19102-1192	12-3-90	1938	G.S., Vascular
Kolff, Jacob, M.D. Conemaugh Valley Memorial Hospital 1086 Franklin Street Johnstown, PA 15905	1-10-84	1938	Thoracic
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. 2617 Huntingdon Pike, Suite 202 Huntingdon Valley, PA 19006	1-7-74	1936	G.S., Thoracic
Levien, David H. Episcopal Hospital Front Street & Lehigh Avenue Philadelphia, PA 19125	4-6-92	1948	G.S., Colorectal
Lieber, Claude P. Suite #901, One Graduate Plaza Philadelphia, PA 19146	12-2-91	1945	G.S.
Marchildon, Michael B., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	4-2-84	1940	Pediatric
Mattson, Ronald J., M.D. 306 Bryn Mawr Avenue, 1 North Bryn Mawr, PA 19010	11-5-84	1945	G.S.
McClurken, James B., M.D. Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	10-7-91	1950	G.S., Thoracic
McCombs, Peter, M.D. 1245 Highland Avenue, Suite 600 Abington, PA 19001	4-5-82	1944	G.S.
McGrath, Lynn B., M.D. Deborah Heart & Lung Center 200 Trenton Road Browns Mills, NJ 08015	4-2-90	1951	Cardiothoracic

	ELECTED	BORN	SPECIALTY
Millili, John, M.D. Presbyterian Medical Center Medical Office Building, Suite 140 39th & Market Streets Philadelphia, PA 19104	1-7-91	1954	G.S.
Mulholland, S. Grant, M.D. 111 S. 11th Street, Suite 6128 Philadelphia, PA 19107	2-2-76	1936	Urologic
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	Plastics
Nakhgevany, Karim B., M.D. 302 Fairview Road Narberth, PA 19072	4-2-84	1937	G.S.
Noone, R. Barrett, M.D. 888 Glenbrook Avenue Bryn Mawr, PA 19010	4-5-76	1939	Plastics
O'Connor, Michael J., M.D. One Graduate Plaza, Suite 1100 Philadelphia, PA 19146	11-3-80	1941	Neurosurgery
O'Malley, Keith F., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	10-7-91	1949	G.S.
O'Neill, James A., Jr., M.D. Children's Hospital One Children's Center Philadelphia, PA 19104	11-7-83	1933	Pediatric
Padula, Anthony, M.D. 8216 Seminole Avenue Philadelphia, PA 19118	11-1-82	1941	G.S.
Paskin, David L., M.D. Pennsylvania Hospital Eighth & Spruce Streets Philadelphia, PA 19107	12-1-75	1938	G.S.
Pavlides, Constantinos A., M.D. The William Penn Medical Building Suite 400 245 N. Broad Street Philadelphia, PA 19107	10-6-86	1940	G.S.
Pello, Mark Joel, M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	12-1-86	1949	Colorectal

	ELECTED	BORN	SPECIALTY
Perloff, Leonard Jay, M.D. 3400 Spruce Street Philadelphia, PA 19104	10-1-84	1940	Vascular
Plzak, Louis, Jr., M.D. 101 Bryn Mawr Avenue, Suite 260 Bryn Mawr, PA 19010	5-7-79	1934	Thoracic
Raezer, David M. Mercy Fitzgerald Medical Office Building Suite 301 Darby, PA 19023	2-5-90	1942	Urologic
Raviola, Carol Ann, M.D. 301 S. 8th Street, Suite 2A Philadelphia, PA 19106	10-3-88	1946	Vascular
Rhoads, Jonathan E., Jr., M.D. York Hospital, Department of Surgery 1001 S. George Street York, PA 17405	1-8-79	1938	G.S., Thoracic
Ritchie, Wallace P., Jr., M.D., Ph.D. Temple University Hospital 3401 N. Broad Street Philadelphia, PA 19140	5-7-84	1935	G.S.
Roberts, Andrew B. 3300 Henry Avenue Philadelphia, PA 19129	12-5-88	1948	G.S.
Rombeau, John L., M.D. Hospital of the University of Pennsylvania 4th Floor, Silverstein 3400 Spruce Street Philadelphia, PA 19104	11-7-83	1939	Colorectal
Rosato, Ernest F., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-4-74	1936	G.S.
Rosen, Harvey Marc, M.D. 301 S. 8th Street, Suite 3H Philadelphia, PA 19106	5-6-85	1947	Plastics
Ross, Arthur J., III, M.D. Children's Hospital of Philadelphia 34th Street & Civic Center Boulevard Philadelphia, PA 19104	11-7-88	1949	Pediatric
Ross, Steven E., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	1-7-91	1951	G.S.
Sachdeva, Ajit K., M.D. 3300 Henry Avenue Philadelphia, PA 19129	12-1-86	1951	G.S.
Sala, Luis E. 1801 Pine Street Philadelphia, PA 19103	4-6-92	1944	G.S., Vascular

	ELECTED	BORN	SPECIALTY
Sataloff, Dahlia M. 1800 Lombard Street, #901 Philadelphia, PA 19146	4-6-92	1953	G.S.
Savarese, Ronald, M.D. 700 Spruce Street, Suite 101 Philadelphia, PA 19106	12-7-81	1943	G.S.
Schwab, C. William, M.D. PENNSTAR 3400 Spruce Street, G1 Philadelphia, PA 19104	12-5-88	1946	G.S.
Schwartz, Gordon, M.D. 1015 Chestnut Street, Suite 510 Philadelphia, PA 19107	2-1-78	1935	G.S.
Shaikh, Khaleel, M.D. Division of Trauma & Critical Care Community Medical Center 1822 Mulberry Street Scranton, PA 18510	11-6-89	1940	G.S.
Sink, James D. Presbyterian Medical Center 51 N. 39th Street Philadelphia, PA 19104	4-6-92	1949	Cardiothoracic
Silver, Stephen C., M.D. 1010 West Chester Pike Havertown, PA 19083	10-3-83	1946	G.S.
Smink, Robert D., Jr., M.D. Lankenau Medical Building, Suite 233 Philadelphia, PA 19151	1-7-40	1940	G.S.
Smullens, Stanton N., M.D. 111 S. 11th Street, Suite 6255 Philadelphia, PA 19107	5-1-72	1936	G.S., Thoracic
Snyder, Howard M., III, M.D. Children's Hospital of Philadelphia Urology Division, 3rd Floor 34th Street & Civic Center Boulevard Philadelphia, PA 19104	12-1-86	1943	Urologic, Pediatric
Solit, Robert W., M.D. 111 S. 11th Street, Suite 8229 Philadelphia, PA 19107	11-4-74	1935	G.S.
Spence, Richard K., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	12-2-85	1945	Vascular
Vernick, Jerome J., M.D. 111 South 11th Street, Suite 8254 Philadelphia, PA 19107	1-3-77	1937	G.S.
Vinocur, Charles D., M.D. St. Christopher's Hospital for Children Erie Avenue at Front Street Philadelphia, PA 19134-1095	1-9-89	1947	Pediatric

	ELECTED	BORN	SPECIALTY
Weese, James L., M.D. Presbyterian Medical Center 39th & Market Streets Philadelphia, PA 19104	11-7-88	1949	Surgical Oncology
Wein, Alan J., M.D. 3400 Spruce Street Philadelphia, PA 19104	5-3-76	1941	Urologic
Weingarten, Michael S., M.D. 1212 Cherinar Lane Penn Valley, PA 19072	5-2-88	1949	G.S.
Weintraub, William, M.D. 1260 Lakemont Road Villanova, PA 19083	11-2-81	1939	Pediatric
Weiss, Stephen M., M.D. Mercy Fitzgerald Medical Office Building 1501 Lansdowne Avenue, Suite 207 Darby, PA 19023	10-1-84	1947	G.S., Surgical Oncology
Whitaker, Linton A., M.D. 3400 Spruce Street Philadelphia, PA 19104	1-6-75	1936	Plastics
Yum, Keuk Y., M.D. 100 Lancaster Avenue 334 Lankenau Medical Building West Wynnewood, PA 19096	10-7-74	1936	G.S.
Zaren, Howard A., M.D. 3300 Henry Avenue Philadelphia, PA 19129	10-6-86	1941	G.S.

Senior Fellows

	ELECTED	BORN	SPECIALTY
Armitage, Harry V., M.D. 1032 General Lafayette Boulevard West Chester, PA 19382	10-6-58	1916	G.S.
Bacharach, Benjamin, M.D. 1025 Walnut Street Philadelphia, PA 19107	5-3-71	1939	G.S.
Barker, Clyde F., M.D. 3400 Spruce Street Philadelphia, PA 19104	12-7-70	1932	G.S.
Bassett, James G., M.D. Medical College of Pennsylvania 3300 Henry Avenue Philadelphia, PA 19129	12-6-61	1919	G.S.
Bishop, Harry G., M.D. Children's Hospital One Children's Center Philadelphia, PA 19104	5-3-71	1921	G.S.

	ELECTED	BORN	SPECIALTY
Bower, Robert M., M.D. 230 N. Broad Street Philadelphia, PA 19102	1-6-67	1925	G.S.
Boyd, Robert T., III, M.D. 301 Keithwood Road Wynnewood, PA 19096	11-6-67	1925	G.S.
Brockman, Stanley K., M.D. Hahnemann University Hospital Broad & Vine Streets, Mail Stop 111 Philadelphia, PA 19102	10-7-74	1928	G.S., Thoracic
Buyers, Robert A., M.D. Box 205 – Dogwood Lane Gwynned Valley, PA 19437	10-1-56	1917	G.S.
Camishion, Rudolph C., M.D. Three Cooper Plaza, Suite 411 Camden, NJ 08103	1-4-65	1927	G.S., Thoracic
Chen, Chijen, M.D. Suite 233, Lankenau Medical Building Philadelphia, PA 19151	1-4-71	1933	G.S.
Closson, Edward W., M.D. Homestead Farm 260 North Main Street Lambertville, NJ 08530	12-5-66	1914	G.S.
Cohn, Herbert E., M.D. 111 S. 11th Street, Suite 8229 Philadelphia, PA 19107	12-6-65	1930	G.S., Thoracic
Cooper, Donald R., M.D. 8 Chamond Arbordeau Devon, PA 19333	10-6-52	1917	G.S.
D'Alonzo, Walter A., M.D. 1647 S. 15th Street Philadelphia, PA 19145	5-2-55	1914	G.S.
Davis, Richard A., M.D. 3400 Spruce Street Philadelphia, PA 19104	1-6-69	1925	Neurosurgery
DeLaurentis, Dominic A., M.D. Pennsylvania Hospital Eighth & Spruce Streets Philadelphia, PA 19107	5-4-64	1925	Cardiovascular
DiGiovanni, Alphonse, M.D. Four Martin's Run Media, PA 19063	2-4-80	1931	G.S.
Donnelly, Joseph C., Jr., M.D. 200 N. 13th Street, Suite 307 Reading, PA 19604	1-8-68	1929	G.S., Thoracic, Cardiovascular
Dorian, Alan L., M.D. 2 Woodstream Drive, Apt. G-6 Norristown, PA 19403	—	1920	G.S.

	ELECTED	BORN	SPECIALTY
Dzwonczyk, John, M.D. 45 Ashby Road Upper Darby, PA 19082	10-3-83	1927	G.S.
Edmunds, L. Henry, Jr., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-7-74	1931	Cardiovascular
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. 624 Scott Building 1020 Walnut Street Philadelphia, PA 19107	1-7-66	1918	Orthopaedics
Gonick, Paul, M.D. 227 North Broad Street Philadelphia, PA 19107	5-6-74	1930	Urologic, G.S.
Gorham, William K., III, M.D. 405 Atwater Road Broomall, PA 19008	1-6-69	1927	G.S.
Gostigian, John J., M.D. 1016 Warrior Road Drexel Hill, PA 19026	5-4-70	1929	G.S.
Gowen, George F., M.D. 1133 East High Street Pottstown, PA 19464	1-4-65	1925	G.S.
Griffen, Ward O., Jr., M.D. American Board of Surgery 1617 JFK Boulevard Philadelphia, PA 19103	1-7-85	1929	Thoracic
Hardesty, William H., M.D. 416 Bellevue Avenue Trenton, NJ 08618	10-7-68	1932	G.S.
Harris, James S. C., M.D. Roxborough Memorial Hospital 5800 Ridge Avenue Philadelphia, PA 19128	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.

	ELECTED	BORN	SPECIALTY
Holst, Hazel, M.D. 15 Morton Avenue Ridley Park, PA 19078	11-5-73	1931	G.S., Plastics
Hughes, Eugene P., Sr., M.D. 109 Sparango Lane Plymouth Meeting, PA 19462	5-4-64	1924	G.S.
Krueger, Charles S., M.D. 131 Madison Avenue Mt. Holly, NJ 08060	5-4-70	1933	G.S.
Lamp, J. Curtis, M.D. 888 Glenbrook Avenue Bryn Mawr, PA 19010	11-7-66	1918	Plastics
Lerner, Harvey J., M.D. Germantown Hospital Medical Center One Penn Boulevard 4th Floor, Wister Tower Philadelphia, PA 19144	2-3-67	1932	G.S.
Mackie, Julius A., M.D. 3400 Spruce Street Philadelphia, PA 19104	11-7-66	1927	G.S.
Maier, Willis P., M.D. 3401 N. Broad Street Philadelphia, PA 19140	5-4-70	1933	G.S.
Marks, Gerald, M.D. 1100 Walnut Street, Suite 702 Philadelphia, PA 19107	10-5-70	1925	Colorectal, G.S.
Matsumoto, Teruo, M.D. 230 N. Broad Street, Suite 7150 Philadelphia, PA 19102	2-7-72	1929	G.S.
Miller, Leonard D., M.D. 3400 Spruce Street Philadelphia, PA 19104	10-5-70	1930	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
Murtagh, Frederick, Jr., M.D. Hospital of the University of Pennsylvania 210 White Building 3400 Spruce Street Philadelphia, PA 19104	11-6-67	1917	Neurosurgery
Neal, Hunter S., M.D. Suite 334, Lankenau Medical Building Wynnewood, PA 19096	11-6-66	1923	G.S.
Nelson, Harry M., Jr., M.D. 15 Wood Street Norristown, PA 19401	5-1-72	1931	G.S.

	ELECTED	BORN	SPECIALTY
Nemir, Paul, Jr., M.D. Room 1100, 19th & Lombard Streets Philadelphia, PA 19146	1-3-55	1920	G.S., Thoracic
Nusbaum, Moreye, M.D. Presbyterian Medical Center Suite 140, Medical Office Building 39th & Market Streets Philadelphia, PA 19104	1-7-74	1929	G.S., Thoracic
Osterholm, Jewell, M.D. 1025 Walnut Street, Suite 501 Philadelphia, PA 19107	11-4-74	1929	Neurologic, G.S.
Pechin, Sergius P., M.D. 430 Sycamore Mills Road Media, PA 19063	2-4-74	1914	G.S.
Pecora, David V., M.D. 4747 Hogan Drive Wilmington, DE 19808	2-3-75	1916	G.S., Thoracic
Perlman, Morton H., M.D. 230 N. Broad Street Philadelphia, PA 19102	2-7-72	1930	G.S.
Pierucci, Louis, M.D. 455 Route #70 West Cherry Hill, NJ 08002	12-6-65	1928	G.S., Thoracic
Quill, Joseph R., M.D. 21 W. Fornance Street Norristown, PA 19401	1976	1928	G.S.
Randall, Peter, M.D. 3400 Spruce Street Philadelphia, PA 19104	4-1-62	1923	Plastics
Ranieri, Tito A., M.D. 2320 S. Broad Street Philadelphia, PA 19145	1951	1912	Orthopaedics
Reichle, Frederick A., M.D. 51 N. 39th Street Philadelphia, PA 19104	12-6-71	1935	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.
Rosato, Francis, M.D. 1025 Walnut Street, Suite 605 Philadelphia, PA 19107	12-6-71	1934	G.S.
Saris, Demetrius S., M.D. 230 N. Broad Street Philadelphia, PA 19102	2-1-65	1921	G.S.

	ELECTED	BORN	SPECIALTY
Scheuermann, Henry, M.D. 115 E. Township Line Road Upper Darby, PA 19082	11-1-82	1930	Plastics
Schumann, Francis, M.D. Five Pleasant Street Machias, ME 04654	4-2-62	1914	G.S.
Schwegman, Cletus W., M.D. 3400 Spruce Street Philadelphia, PA 19104	1951	1914	G.S.
Sherk, Henry H., M.D. 1210 Brace Road Cherry Hill, NJ 08034	2-3-69	1930	Orthopaedics
Sigel, Bernard, M.D. 3300 Henry Avenue Philadelphia, PA 19129	—	1930	Vascular
Snedden, Hal, M.D. 350 Hidden River Road Narberth, PA 19072	12-1-75	1922	Orthopaedics
Somers, Laurence A., M.D. 727 Welch Road, Suite 102 Huntingdon Valley, PA 19006	11-6-78	1931	G.S., Pediatric
Steel, Howard H., M.D. Shriners Hospital, Philadelphia Unit 8400 Roosevelt Boulevard Philadelphia, PA 19152	5-6-68	1921	Orthopaedics
Templeton, John Y., M.D. 311 Airdale Road Rosemont, PA 19010	1954	1915	G.S.
Trout, Robert, M.D. 2100 Keystone Avenue, Suite 400 Drexel Hill, PA 19026	11-3-69	1922	Thoracic
Wagner, David K., M.D. 3300 Henry Avenue Philadelphia, PA 19129	2-3-69	1931	Pediatric
Wagner, Frederick B., Jr., M.D. Room 306, Scott Building 1020 Walnut Street Philadelphia, PA 19107	1-7-52	1916	G.S.
Wallace, Herbert W., M.D. 255 Harrogate Road Wynnewood, PA 19096	11-4-74	1930	G.S., Thoracic
West, Clifton, Jr., M.D. Route 3, Box 127 Chestertown, MD 21620	2-1-60	1923	G.S.
White, Jack D., M.D. 972 South Penn Drive West Chester, PA 19380	12-4-72	1928	G.S.

	ELECTED	BORN	SPECIALTY
Williams, Kirkley R., M.D. 207 Bryn Mawr Medical Building Bryn Mawr, PA 19010	5-1-71	1931	Thoracic
Wolferth, Charles C., Jr., M.D. 1900 Lombard Street Philadelphia, PA 19146	11-1-63	1928	G.S.

Inactive Fellows

	ELECTED	BORN	SPECIALTY
Alday, Edgardo, M.D. 747 Conestoga Road Rosemont, PA 19010	2-1-82	1933	G.S.
Bernhard, Victor M., M.D. University of Arizona Health Science Center Department of Surgery Tucson, AZ 85724	4-5-82	—	—
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.
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.
Cohen, Erwin A., M.D. Medical Arts Building 60 East Township Line Road Elkins Park, PA 19117	2-4-74	1925	G.S.
Cooper, Robert A., M.D. 804 Mark 70, Route 70 Cherry Hill, NJ 08034	1-6-53	1917	G.S.
Culf, Norris K., M.D. 644 Norristown Road Horsham, PA 19044	11-3-75	1931	G.S., Plastics
Davila, Julio C., M.D. 413 Alarid Street Santa Fe, NM 87501	4-1-68	1928	Thoracic
DeTuerk, John J., M.D. 10150 N. Ocean Drive Citrus Springs, FL 32630	5-7-71	1912	G.S.
Farrell, Harry L., M.D. 135 S. 20th Street Philadelphia, PA 19103	—	1905	—

	ELECTED	BORN	SPECIALTY
Gilmour, William R., M.D. 6616 Woodland Avenue Philadelphia, PA 19142	1928	1891	G.S.
Goldsmith, Harry 75 East Newton Street Boston, MA 02118	5-1-72	1929	G.S.
Hall, John M., M.D. 604 General Scott Road Wayne, PA 19087	—	1915	G.S.
Haupt, George J., M.D. Suite 306, Lankenau Medical Building Philadelphia, PA 19151	10-5-59	1924	G.S.
Hume, H. Alan, M.D. RFD #1, Pond Road Oakland, ME 04963	2-3-64	1926	G.S.
Jones, Robert K., M.D. Lankenau Medical Building, Suite 655 Lancaster Avenue West of City Line Wynnewood, PA 19096	1965	1924	Neurosurgery
Kaplan, Louis, M.D. 1204 Greentree Lane Narberth, PA 19072	4-4-47	1904	G.S.
Langfitt, Thomas W., M.D. President & CEO The Glenmede Trust Co. 229 South 18th St. Philadelphia, PA 19103	2-7-66	1927	Neurosurgery
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. 202 Broad River Drive 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.
McKeown, John J., Jr., M.D. 935 Cedar Grove Road Wynnewood, PA 19096	4-3-61	1919	G.S., Thoracic, Vascular
McLaughlin, Edward D., M.D. 3112 Garnet Mine Road Boothwyn, PA 19061	5-5-69	1931	Thoracic
Medinger, Frederick G., M.D. The Beaumont 77 Middle Road, 163 Bryn Mawr, PA 19010	1950	1911	G.S.
Murphy, John J., M.D. 1501 Lansdowne Avenue, Suite 304 Darby, PA 19023	—	—	Urologic

	ELECTED	BORN	SPECIALTY
O'Neill, James F., M.D. 8116 Bustleton Avenue Philadelphia, PA 19152	1954	1910	G.S.
Pitt, Leldon P., M.D. 919 Club House Boulevard New Smyrna Beach, FL 32069	12-5-60	1920	G.S.
Roberts, John M. 234 West Allens Lane Philadelphia, PA 19119	5-4-64	1926	G.S.
Rosemond, George P., M.D. 3401 N. Broad Street Philadelphia, PA 19140	1945	1910	G.S.
Royster, Henry P., M.D. 1507 Canterbury Road Raleigh, NC 27608	1950	1909	Plastics
Sensenig, David M. 436 A State Street Bangor, ME 04401	—	1921	Thoracic
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.
Stainbach, William C., M.D. 2221 Buttonwood Road Berwyn, PA 19312	4-1-57	1916	G.S.
Tobias, Gordon L., M.D. Vice President, Medical Affairs Delaware Valley HMO Greater Valley Health Care, Inc. 9 LaCrue Street P.O. Box 1111 Concordville, PA 19331	12-4-78	1927	Urologic
Troncilleti, Manrico, M.D. 1282 Round Hill Road Bryn Mawr, PA 19010	5-4-70	1915	G.S.
Tyson, R. Robert, M.D. 3401 N. Broad Street Philadelphia, PA 19104	12-6-54	1920	G.S.
Weber, Edgar H., M.D. 3008 E. Powell Avenue Evansville, IN 47714	—	—	G.S.
White, Jack, M.D. 972 South Penn Drive West Chester, PA 19380	12-4-72	1928	G.S.

Non-Resident Fellows

	ELECTED	BORN	SPECIALTY
Allbritten, Frank F., Jr., M.D. P.O. Box 177 Cunningham, KS 67035	—	1914	G.S., Thoracic
Austin, George, M.D. 933 Alston Road Santa Barbara, CA 93108	—	—	Neurosurgery
Bailey, Charles P., M.D. 114 Prospect Avenue Neptune, NJ 07753	—	—	G.S., Thoracic
Beljan, John R., M.D. 6490 Saddle Drive Long Beach, CA 90815	11-5-84	1930	G.S.
Boland, James P., M.D. 1939 Parkwood Road Charleston, WV 25314	10-7-68	1931	G.S., Thoracic
Cohen, Max, M.D. 4567 East Ninth Avenue Denver, Colorado 80220	—	—	—
Crichlow, Robert W., M.D. Dartmouth Medical School Hanover, NH 03755	—	1932	G.S.
Deutsch, Joel, M.D. 13 Finch Run Avon, CT 06001	12-7-70	1926	G.S.
Dudrick, Stanley J., M.D. Hermann Hospital 6411 Fannin, 6th Floor—Cullen Building Houston, TX 77030	1971	1935	G.S.
Fry, Kenneth E., M.D. 621 University Avenue Walla Walla, WA 99362	—	1902	G.S.
Goldman, Leonard, M.D. 3013 Nottingham Drive Shreveport, LA 71115	—	1930	G.S.
Gross, Richard H., M.D. 11 College Street P.O. Box 459 Littleton, NC 27850	5-6-74	1935	G.S.
Hartford, Charles E., M.D. 7269 South Andes Court Aurora, CO 80016	12-4-78	1932	G.S.
Jarrell, Bruce E., M.D. Professor & Head, Department of Surgery University of Arizona 1501 N. Campbell Avenue Tucson, AZ 85724	10-6-86	1947	G.S., Vascular

	ELECTED	BORN	SPECIALTY
Kholoussy, A. Mohsen, M.D. 1851 West End Avenue Pottsville, PA 17901	2-4-85	1947	G.S.
Koop, C. Everett, M.D. 5924 Maplewood Park Place Bethesda, MD 20814	—	1916	Pediatric
Law, F. Dana, M.D. 125 W. Market Street Lewistown, PA 17044	2-3-64	1924	G.S.
Manges, Lewis D., M.D. Seven Palm Lane, Box 176 Tangerine, FL 32777	—	1906	G.S.
Masson, Newton L., M.D. 3564 Montclair Circle Shingle Springs, CA 95682	—	—	—
McNamara, Marian F., M.D. 3990 John R Street Detroit, MI 48201	1-4-82	1946	Vascular
Morris, Robert S., M.D. 938 Strangler Fig Lane Sanibel, FL 33957	1958	1915	—
Nealon, Thomas F., Jr., M.D. St. Vincent's Hospital 170 W. 12th Street New York, NY 10011	—	1920	G.S.
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. 5920 Forest Park Road, Suite 530 Dallas, TX 75235	11-4-76	1932	Orthopaedics
Schmidek, Henry H., M.D. 166 Ridge Road Grosse Pointe, MI 48230	2-7-77	1937	Neurosurgery
Schwartz, Gordon, M.D. *(no address available)*	2-1-78	1935	G.S.
Sears, Henry F., M.D. c/o Copley Venture Partners 600 Atlantic Avenue, 13th Floor Boston, MA 02210	4-6-81	1940	Surgical Oncology
Sencindiver, P. Victor, M.D. 220 Centre Street Beach Haven, NJ 08008	2-1-65	1927	G.S.
Simonian, Simon J., M.D. The Vein Institute 3301 Woodburn Road, Suite 102 Annandale, VA 22003	12-6-82	1932	Vascular

	ELECTED	BORN	SPECIALTY
Stahlgren, LeRoy H., M.D. Director of Surgery St. Joseph Hospital 1835 Franklin Street Denver, CO 80218-1191	11-7-60	1924	G.S.
Stayman, Joseph W., Jr., M.D. 322 Hearthstone Ridge Landrum, SC 29356-9602	1950	1915	G.S.
Stephenson, Larry W., M.D. Harper Grace Hospital 3990 John R Street Detroit, MI 43236	10-5-81	1944	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
Thompson, James C., M.D. Department of Surgery University of Texas Galveston, TX 77550	—	—	G.S.
Ziegler, Moritz M., M.D. Children's Hospital Medical Center 240 Bethesda Avenue Cincinnati, OH 45229	11-7-83	1942	Pediatric

Annual Oration for 1987

Treatment of Parotid Salivary Gland Tumors

ROBERT D. HARWICK, M.D.

The possibility of a salivary gland neoplasm must be strongly considered in a patient with a swelling in the area of the parotid gland even if the swelling has been present for years. When the swelling is intermittent, associated with discomfort, and there is a purulent discharge from Stenson's Duct, a much rarer inflammatory process is most likely. In adults, 80% of parotid tumors are benign. In children with a parotid swelling not due to mumps, hemangiomas and embryonal rhabdomyosarcomas are more common than salivary gland neoplasms. In children only 50% of parotid tumors are benign and the most common malignant tumor is muceopidermoid carcinoma.

In the painting by Robert Hinckley of Dr. Morton giving ether anaesthesia at Massachusetts General Hospital, Dr. John Collins Warren is depicted removing a tumor from the left parotid area. This was reported in a letter to the *Boston Medical and Surgical Journal* in December of 1846. The operation took 30 minutes and although Dr. Warren called the lesion a tumor of the neck it may well have been a parotid salivary gland tumor.

The etiology of parotid tumors is usually unknown. However, a history of radiation is found occasionally. In a well known article in *Lancet,* February 1974 11,000 children had immigrated to Israel from North Africa and other countries of the Mideast. All received irradiation to the scalp because of a high incidence of ringworm. The children were followed for 12 to 23 years, a marked increase incidence of brain, thyroid and parotid tumors was found. The incidence of salivary gland tumors was markedly increased in survivors of Hiroshima and Nagasaki 12 to 25 years later as reported by the Atomic Bomb Casualty Commission in *Cancer,* February 1975. In the same journal in October 1963 a report of parotid tumors in Eskimos noted a 30-time increase in incidence as compared with that of the neighboring white population. These tumors were often poorly differentiated. Repeated dental x-rays in children and young adults have been suspect but I am unaware of any study that documents a relationship to development of salivary gland tumors.

The parotid salivary gland is the site of salivary neoplasms in 70% of cases with the submandibular, sublingual, minor salivary glands and lachrymal glands being the primary site in the remaining cases.

The pathologic classification of salivary gland tumors is unique and in no other organ system does the pathologic type so influence prognosis and treatment. The most frequent types are listed below.

Histologic Classification of Salivary Gland Neoplasms

Benign

- Pleomorphic Adenoma (Benign Mixed Tumor)
- Warthin's Tumor — Papillary Cystadenoma Lymphmatosium
- Oncocytoma
- Benign Lymphoepithelial Lesion
- Monomorphic Adenoma
  - Sebaceous Gland or Basal Cell Adenomas

Malignant

- Malignant Mixed Tumor
- Mucoepidermoid Carcinomas
- Adenoid Cystic Carcinoma
- Acinic Cell Carcinoma
- Squamous Cell Carcinoma
- Adenocarcinoma

Pleomorphic Adenomas account for about 90% of benign tumors. These lesions have a pseudocapsule and should not be enucleated as growth occurs by pseudopods extending into the false capsule. If enucleated, recurrence rates of 40% or more can be expected. A recurrence rate of less than 4% occurs when the tumors are removed with normal parotid tissue on all sides of the lesion.

Almost all Warthin's Tumors occur in the parotid gland. The typical presentation is an asymptomatic mass in the region of the tail of the parotid in an elderly male. 15% of these lesions are multicentric and 10-15% may be bilateral. All other salivary gland tumors are unicentric and never bilateral.

Benign Lymphoepithelial Lesions are seen in Sicca Syndrome, rheumatoid arthritis and in AIDS patients.

Monomorphic Adenomas have been recognized recently, but they have not been followed long enough to determine histologic implications. It is thought that their action will be the same as pleomorphic adenomas.

The Malignant Mixed Tumor is the malignant variant of the pleomorphic adenoma. Its clinical presentation is often that of a benign tumor and it usually is not high grade or high stage.

Mucoepidermoid Carcinomas comprise about 20% of parotid tumors and may be a low, intermediate or high grade tumor. The grade correlates well with clinical behavior. Low grade tumors are almost 100% curable while high grade tumors have a 5-year cure rate of only 10-15% and are usually high stage as well.

Adenoid Cystic Carcinoma accounts for about 12% of malignant parotid tumors. It was first described by Theodore Billroth in 1859 and called a cylindroma. It is an insidious tumor in that it is histologically benign-appearing, but rarely curable when followed for 10 to 20 years. There is frequent nerve invasion and perineural spread well beyond its gross margins. Vascular permeation may be seen and hematogenous metastases are eventually common. The tumor rarely metastasizes to lymph nodes.

Acinic Cell Carcinoma occurs almost exclusively in the parotid gland where it accounts for 10-15% of malignant tumors. A clear cell variant could be mistaken

for a metastatic renal cell carcinoma. Prognosis is usually good but there is a rare papillocystic variety that is very virulent with a 5-year survival of only 20% and a 10-year survival of 0%.

Squamous Cell Carcinomas are very rare and usually present with a high stage and have a poor prognosis.

Adenocarcinoma comprises about 10% of malignant salivary gland tumors that cannot be classified in a more defineable way. They are often of high stage when first diagnosed.

The following is a breakdown of the distribution of 1,968 consecutive parotid tumors seen at Memorial Sloan-Kettering.

Parotid Tumors

Benign	1,342
Malignant	626
Mucoepidermoid Ca	272
Malignant Mixed Tumor	107
Acinic Cell Ca	75
Adenocarcinoma	62
Adenoid Cystic Ca	54
Squamous Cell Ca	48
Anaplastic	8

Benign tumors are often of long duration with no appreciable change. A malignant salivary gland tumor will often have a period of rapid growth in a previously quiescent tumor. One should consider that the tumor is very likely to be malignant if there is associated pain, tenderness, and/or weakness or paralysis of the facial nerve or one of its branches. Malignant tumors may be stony hard, relatively fixed and may infiltrate the overlying skin. However, some malignant tumors can be a well circumscribed, asymptomatic, freely moveable mass that has been present without change for many months. This is often the presentation of low grade mucoepidermoid tumors, acinic cell carcinomas, adenoid cystic carcinomas and malignant mixed tumors.

Sialography is more useful in showing duct stenosis or sialectasis in a case of chronic parotitis than it is in evaluating the benign or malignant nature of a parotid tumor. CT scans may be helpful in evaluating advanced tumors or the rare parotid tumor that arises from the deep portion of the gland and presents as a large submucosal oro-pharyngeal tumor. A Warthin's Tumor will show up as a hot nodule when radioisotopes scanning with Technetium 99M is done. Fine needle aspiration biopsy is done by some but not advised because it is difficult for the pathologist to give a definitive diagnosis from the scant material. Needle biopsy material can be definitive for a Warthin's Tumor.

Benign lesions and most T1 or T2 lesions can be removed with a conventional superficial parotidectomy with facial nerve presentation; 80% or more of the tumors are in a plane above the nerve trunk and its branches. The operation should begin with posterior exposure of the facial nerve trunk as it exits the stylomastoid foramen. The surgeon may in rare instances have to identify the main trunk by tracing a

peripheral branch posteriorly. A superficial parotidectomy is similar to doing a thyroid lobectomy for removal of a cold nodule. An incisional biopsy is not done. The tumor with normal parotid tissue on all its aspects is submitted for frozen section. The superficial lobectomy becomes the definitive procedure for benign lesions and a near total parotidectomy with nerve preservation is usually completed when a malignant diagnosis is reported. The chances for neuropraxia can be lessened by avoiding the use of a cautery near the facial nerve trunk or its branches and minimizing stretching of the nerve as much as possible with gentle traction. If neuropraxia occurs postoperatively, functional return is assured if the nerve trunk is intact. If the main trunk or one or more of the branches of the facial nerve is found to be infiltrated by the tumor at the time of operation, do not hesitate to sacrifice all or part of the nerve to obtain a safe margin of excision. Many times a nerve graft is feasible when this is done. The concept of a radical operation when indicated by histologic type or operative findings should dwell less on routine sacrifice of the facial nerve, which often can be avoided in part by preserving the branches to the eyelids, for example, and more on a wide removal of the involved tissues such as the masseter muscle, a portion of the mandible, or external ear canal. A radical neck dissection is added for obvious metastases, to facilitate resection of large primaries or recurrent tumors and for high grade mucoepidermoid and squamous cell carcinomas. Postoperative x-ray therapy is given for high grade and high stage tumors and for most adenoid cystic carcinomas because of extensive perineural spread.

Adjuvant chemotherapy has not been helpful and responses of recurrent and metastatic salivary gland tumors to various chemotherapy agents has been anecdotal at best. Complete surgical resection is essential and there is no salvage possible with x-ray therapy with inadequate resections. For all malignant salivary gland tumors, stage determines prognosis. Follow-up for all tumors, benign and malignant, should be at least 10 years and 20 years for adenoid cystic carcinoma.

A small number of parotid tumors arise from the deep portion of the gland and extend through the gap between the ramus of the mandible and the stylomandibular ligament to enter the parapharyngeal space and appear as large submucosal oropharyngeal tumors that displace the soft palate and tonsillar area toward the midline. These dumbell tumors should not be removed by the oral route as certain rupture will occur and injury to the facial nerve is very possible. They are readily removed with the usual parotidectomy incision and with division of the stylomandibular ligament after the facial nerve trunk and branches are identified and preserved. Fortunately most of these tumors are benign pleomorphic adenomas.

An overall 20-year survival rate of malignant parotid salivary gland tumor is listed below.

Acinic Cell Ca	78%	Malignant Mixed Tumor	40%
Mucoepidermoid Ca	68%	Adenoid Cystic Ca	33%
Adenocarcinoma	45%	Squamous Cell Ca	33%

Although these overall percentages for a 20-year follow-up are quite acceptable, lesions that were of high stage (i.e. Stage III and Stage IV) and/or high grade have a uniformly poor survival whatever the histologic classification may be. The exception is Adenoid Cystic carcinoma where the stage is important in predicting survival,

but where all histologic grades have the same 20-year survival.

The clinical staging of salivary gland malignant lesions is unique in that each T classification is subdivided into T1-4A or T1-4B. The (A) denotes "without significant local extension" and the (B) denotes "significant local extension" defined as clinical or macroscopic evidence of tumor involvement of skin, soft tissues, bone or the facial nerve. Lesions with the (B) subset are in a higher stage than the same sized tumor without significant local extension. This is most important for prognosis of salivary gland tumors since Stage is the single most predictive element.

I hope that I have been able to remind you of the long tradition general surgeons have had in treating tumors of the parotid salivary gland and I hope we continue to pass this on to our residents and not allow the treatment of these lesions to become a victim of further fragmentation of general surgery.

Annual Oration for 1988

Odysseus

RUDOLPH C. CAMISHION, M.D.

Odysseus, during one of his voyages as recounted in Homer's *Odyssey,* had to travel through the narrow passage between Italy and Sicily now known as the Strait of Messina. On one shore lived the monster Scylla and on the other was Charybdis. Scylla, daughter of Phorceys and Cratias had twelve feet and six long, snake-like necks each bearing a hideous head with three rows of pointed teeth. Her loins were protected by heads of vicious dogs growing from it. She lived in a cave in a large rock on the shore and whenever anyone ventured within her reach she struck out and devoured the intruder. She was able to seize six of Odysseus' crew. Charybdis dwelled under a large fig tree directly across on the opposite shore. This monster sucked in and belched forth the sea water creating a whirlpool three times daily. Anything floating nearby was engulfed. Odysseus was able to escape being devoured by Charybdis by clinging to a limb of the fig tree after the raft on which he had been riding, was pulled down. He later dropped down on the raft when it floated back to the surface.

These mythical beasts are represented today by the hill called Scilla on the Italian shore and whirlpools near the Sicilian shore. Ships still thread their way between these hazards. In surgery these days, it seems that we encounter a Scylla and Charybdis on every venture we undertake.

I meet every Wednesday afternoon with a group of our junior medical students who are on the surgery rotation. From time to time, the discussions veer into non-clinical topics and the students express some of their concerns. At these sessions, they mention problems which did not exist when I was in medical school, such as their tremendous indebtedness for tuition and living expenses, payment for malpractice insurance upon opening an office and finally, the surplus of physicians. In essence they asked, "Are we going to be able to practice and live in the same style as our predecessors? Spurred by these and other important issues I began to realize that my 30 years of professional life spanned an era of interesting and sometimes disturbing changes in medicine in this country. It is my belief that the present and future course of medicine is replete with hazards and like Odysseus must be negotiated with care if we are to escape unscathed.

As I thought about early times interesting and sometimes amusing similarities between then and now came to me. Since the beginning of recorded history, the physician has played an important role in society. In most cultures, the medicine man or his equivalent, was closely allied with and on the same level as the priests and magicians. These individuals were held in awe by all the others and were deeply

respected. In primitive tribes, the armamentaria of the medicine man were rattles, beads, bones, and other items of magic. Hundreds of years later, our house staff can be seen wearing stethoscopes draped around their necks. Hanging from their coats are reflex hammers, tongue blades, rubber tubing, safety pins, metric rules, — badges attesting to their importance in this world of mystique. Following completion of training, all of these items are removed from the white jacket and placed in a small, black, leather bag. Is the bag a more powerful symbol or is it just to preserve the fit of his *Hickey Freeman* suit?

Years ago, physicians were distinguished not only by their native intelligence but by their cultural education. Knowledge of Latin and Greek was a very desirable credential for an applicant to have when applying to medical school. After all, these two languages make up much of the jargon of physicians and even today many prescriptions and orders are written in Latin. It occurs to me that medical language is as unintelligible to our patients as were the incantations and verbal mutterings of the medicine man many years ago. It still smacks of magic and sorcery and befuddles the patient who assumes that the omnipotent person caring for him is able to speak in strange tongues that are understandable only to the gods. The medicine man gradually evolved into the barber surgeon, the phlebotomist physician and then to his present role as a physician with a solid background in science.

Now, to reach the exalted status of physician, there is a rigorous selection process beginning as early as high school. Students with promise and considerable intelligence are selected to attend college. There begins an active competitive existence as a pre-medical student. From this large group, a relative few are selected to attend medical school. From the graduates, a few are selected for the choice residencies and an even smaller group emerges as leaders of American medicine. Thus we can see that from a large initial pool only the cream of the crop become enlisted into the respected fraternity of physicians and only a very few are at the top of the profession.

Prior to World War II, after the completion of medical school and one or two-year internship, most physicians elected to enter general practice. After the war, there was a marked expansion in the number and types of residencies and many chose to specialize. Most commonly, no stipend was given to the resident, but uniforms, room and board were provided by the hospital. In other words, the house staff became a ward of the hospital and benevolently, had no concern other than the care of his patient. Married applicants were not considered by some programs because it was felt that the resident would then have divided loyalties and might even feel an urgent need to occasionally go home. It was not unusual to remain in the hospital for as long as six or eight weeks without a day off. Some surgical residencies were as long as eight years. However, there was no lack of applicants for these positions, and as a matter of fact, the competition for residency slots in the good university programs was as competitive then as it is today. It is apparent that physicians entering these residencies had a deep love of their profession and an intense desire to be well-trained. Salary and work hours were never a consideration.

University hospitals admitted significant numbers of indigent patients who were cared for by attending physicians and house staff without renumeration. Ambulatory medical care was provided in specialty clinics. There were the long wooden benches

in the clinics where people by the hundreds sat for hours waiting to visit their physicians who were invariably a team of an attending, a resident, and a student. There was a certain glamour to that era. Clinic day became a social event for some patients and many brought lunches with them which they shared with each other. Often patients without an appointment came just to sit and visit with their friends. Patients were grateful for our efforts and malpractice suits were a rarity, The average cost of malpractice insurance for surgeons in those days was a few hundred dollars.

It was smooth sailing up to this point but beware — troubled water was ahead. In the late fifties and early sixties, the Federal government infused vast sums of money into the hospitals and medical schools. The era of reasonably salaried house staff positions had begun. Cost of living also increased but it is hard to believe that it increased sufficiently to offset the zero income of the older residents. With research heavily funded by the NIH, there was an explosion of medical knowledge. The next twenty years saw more progress in the control and understanding of disease than probably in all of recorded medical history that preceded it. The plentiful research grants included salaries for medical school faculty and a generous overhead to fill the coffers of the medical school. Tenure for faculty was often determined by the ability to obtain grants and certainly was dependent upon the number of papers published. An academic medical career became attractive but there was very little incentive to become a skillful, clinical physician and to practice medicine in this environment. A phenomenon arose, in some instances, where surgery was taught by faculty who rarely or never operated on patients.

In 1966, Federal subsidization of health care by way of Medicare and Medicaid began. The number of "free" patients diminished markedly. Income to hospitals and to physicians increased. This was perceived by hospital administration and by physicians as a bonanza but the sad fact was that we had been bought. Most of us didn't realize it, nor cared. Those were the days of increasing affluence.

With these doles from the government came the inevitable sequelae, "those who pay the piper call the tune." The noose has tightened slowly. Now, in 1988, the Federal government, through its agencies, exerts close surveillance and control over the practice of medicine. Professional review organizations determine if the admission of your patient is appropriate. The entire hospital stay is predetermined by the diagnosis. If the patient stay exceeds the alloted number of days, payment to the hospital for the extra days will be denied. If this happens to your patients a number of times you have become a loser to the hospital and they will no longer throw out the welcome mat to you or your patients. The time has come when a nurse from quality assurance calls to ask if a rectal examination has been done on the patient that you wish to admit for an inguinal herniorrhaphy. She wants to be sure that you know that patients with a hernia may have a rectal tumor. There are diagnostic related groups (DRG) to standardize hospital costs. Can a DRG system to reimburse physicians be far behind? The bottom line of bureaucratic decisions seems to be cost rather than quality.

Decreased hospital stay is an effective way to contain costs. As a result, patients needing an elective operation have all consultations and testing done before admission to the hospital. These patients are often seen for the first time by our residents and students either in the anesthesia holding area or in the operating room. There

is virtually no opportunity for them to do a history or physical examination or participate in the development of a diagnosis. This loss of a significant segment of our patient population for medical education is a serious problem. Will we return to the preceptorship system where students are with a particular surgeon all day long and residents become assistants in practice for many years before being allowed to go out on their own?

Administration, or as they prefer to be called, Management, has seized power in the hospital. The bright boys from Harvard Business School and Wharton have seen that a large industry has been run by amateurs and they have rushed in to correct this void with their bottom line fiscal approach. The hospital is a hotel in which physicians are permitted to work. The hotel must be filled at all times and the physician who reliably fills the beds has the most power. The quality of care really does not matter too much unless it causes bad publicity. More and more, hospital managements are hiring physicians to work full-time in their "shop."

The teaching hospital is struggling today to maintain financial solvency. The salaries of residents and fellows and many intangible costs associated with a large house staff and medical students are passed on to the patient. That raises the per diem costs for patient care far above that of the non-teaching hospital. HMO's and similar, for-profit organizations, contract with hospitals that will provide medical care for their constituents at the lowest cost. The University hospital is pricing itself out of the market since the Federal government and state are no longer willing to subsidize the cost of training doctors. They feel, with some justification, that physicians themselves must bear the cost of training since they earn quite adequate incomes when they begin to practice. Interestingly, salaried physicians in some teaching hospitals are expected to help defray some educational costs from their patient service income. The teaching hospital is poorly suited to compete financially with the community non-teaching hospital and another hazard is being approached.

We are now members of the "Health Care Industry!" We speak of consumer and providers. I sometimes wonder if we are not involved in a supermarket operation. The health maintenance organization, with its multiple variants, is now controlling primary care through paid physicians who refer their patients to specialists who offer their services at a discount. Would any surgeon over 40 years of age believe that the day would come when we would be actually bidding for the right to provide our services at the lowest cost? As the number of HMO's increase, the competition increases and physicians offer increasingly larger discounts. It is estimated that in 1995, 50 percent of all physicians will be salaried so there will be less need to negotiate fees.

Who would have thought 20 years ago that hospitals would advertise on radio and television? We are now inundated by promotions in the media emanating from the hospital's public relations offices and even by groups of physicians or individual physicians. Plastic surgeons regularly advertise in *Philadelphia Magazine* showing voluptuous women who have been said to be sculpted by these artists.

Medical care now consumes 10% of the gross national product. The cost for medical benefits for an autoworker now exceeds the cost of steel used in the manufacture of a car. Something has to give. Hospitals are being built or bought by management corporations. As profit making institutions, will they be concerned

with your need to lock in a diagnosis by multiple tests? Will they provide more than the minimal necessary equipment to take care of your patients? Yet, the threat of malpractice requires that every diagnosis be considered and ruled out. Beware, Scylla is lurking on the shore. A whole new tier of lawyers now lives on the filing of malpractice suits. Many of the allegations are frivolous but the shotgun approach may hit a target or as frequently happens, the malpractice insurer will settle rather than undergo expensive litigation. What more can be said about the present state of affairs other than the recent episode in Philadelphia, where a patient sued for loss of psychic powers after a brain CAT scan and received an award by the jury for this disability in excess of a million dollars. The decision was later overturned on appeal but the message is there.

In heading a clinical department, the legal course to plot is indeed fraught with danger. Recently, the estate of a deceased Head of the Department of Medicine in a New Jersey community hospital was successfully sued because while he was living, a member of his staff produced an alleged untoward result. Recently, I was sued by a surgeon whose request for vascular surgical privileges was denied by me. At the same time, I was named in a suit by a patient who contended that I negligently granted privileges in vascular surgery to another surgeon on my staff. I sure wished I had that fig tree to grab onto.

This brings me to consider the problems inherent in the post of a Chairman of a Department of Surgery in 1988. Not too many years ago, Dr. Jonathan Rhoads resigned as Provost of the University of Pennsylvania in order to accept the appointment as Chairman of the Department of Surgery at the medical school. He felt that the surgical chairmanship was a more prestigious and important position. The chairman in that day administered his domain with power and usually with wisdom. He had the time to be a skillful surgeon and because of this, maintained the respect of his colleagues. His decisions were unquestioned. Who could tell an 800-pound gorilla where he could sit? There is no doubt, in some instance, this power was abused and a virtual dictatorship existed. Sometimes the dictatorship was benevolent, sometimes not. Today, a chairman's role is gradually being eroded to the role of administrator hemmed in by rule books and bylaws. After all, if the chairman is in the operating room, he may miss an important meeting and find that important issues have been voted away during his absence. But one must ask the question, can an effective Chairman of Surgery not be a practicing, skillful surgeon? And the answer is still absolutely no.

The practice income of a reasonably large, full-time, Department of Surgery at the average medical school is more than the income of many businesses. Departments now have business and personnel managers and a multitude of support personnel. In our department at Robert Wood Johnson Medical School in Camden, there are 25 full-time surgeons, a department manager with an MBA, a personnel manager, a billing manager with an MBA and over 100 non-professional employees. The majority of these individuals are paid through practice funds. Alex Walt recently published a paper in the *Archives of Surgery* entitled, "The Surgical Chairmanship in a Corporate World." Would-be Chairmen should read it. The post of Chairman of Surgery is still the best job in any medical school, but don't dare fire an attending or even a resident without a foot-high file documenting his deficiencies.

This year, in the state of New Jersey, funded house staff positions have been frozen at the number that existed in 1985. As stated, the Federal and state governments and third party payors are reluctant to subsidize graduate medical education so it is very likely that the salary of the residents has peaked and may even decline. Fellows will be either paid nothing at all, be paid by the faculty with whom they work, or will be paid by direct billing for services rendered to the patient by the fellow. Federal grants to finance medical research have dried up. Surgical research is increasingly being funded either by grants from private foundations, corporations and in many instances by practice income. Hard money for faculty salaries is limited despite the fact there is pressure to increase the number of faculty in developing programs like Robert Wood Johnson Medical School at Camden. Clinical medical school faculty throughout the country are being told that they must practice to subsidize their income. The difference between the private practicing physician and the full-time academic faculty is rapidly diminishing.

The pendulum is swinging back from its outermost reach. In many ways, we are returning again to conditions similar to those that existed in the World War II era. Clinical medical school faculty will receive only a token stipend from the university unless they are in full-time research positions. Compensation will not be as necessary because most clinical teachers will be practicing physicians who are chosen for prestigious faculty positions because of excellence in patient care and clinical research. These individuals will of necessity have an intrinsic love of medicine and a real desire to teach and will be happy to perform this task with minimal payment. Incidentally, people who love to teach are usually the best teachers. It is probable that house staff positions will be subsidized only to the level of being able to take care of living expenses. That's really not too much different than before. Lesser numbers of people will find medicine as a financially attractive career and only men and women who truly love people, who receive much satisfaction taking care of them and enjoy the privilege of being a special and revered person in society will aspire to attend medical school. Service rendered with income as a major consideration is not a scholarly professional activity but a business. If we are to continue the historic traditions of medicine then altruism has to be preserved.

These days, patients are being hospitalized only with absolute indication. Patients who formerly were in the hospital for two weeks are now being discharged after two days without ill effect. Operations of borderline effectiveness are not now being performed or require a second opinion. Medical research and researchers are increasingly being subsidized by private industry, which in turn profits from discoveries made. Certainly one cannot find fault with these end results.

What about physician surplus? Each day, I receive letters offering positions to my residents. The fact is, in my experience, that while there is a surplus of physicians in certain metropolitan areas, less populated regions are hurting for surgical care. He or she may be occasionally paid with a chicken and some vegetables, but regardless of where one practices I have never seen a financially destitute surgeon — have you?

It is up to us not to allow the quality of surgical care to diminish because we still are in charge of that. The excesses in medicine are being corrected as always happens in every field of endeavor if we take the time to study history. Let us try

not to be trapped by Charybdis while trying to avoid Scylla. Let us get back to the basics of medicine which are to care for people, to make them well, to enjoy our way of life and revel in the adulation of our patients and society.

If in the end, the patient is the winner, that's what it's all about and the monsters won't get us because this course is straight through the perils.

Annual Oration for 1989

Reflections on Reflux

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

I have been an unabashed admirer of the sphincters of the upper gastrointestinal tract for many years because, despite their diminutive size, they perform prodigious service: they render *succus entericus* good, in a moral sense, by keeping it in the right place and prevent it from becoming bad by preventing it from gaining access to the wrong place. On occasion these sphincters will fail spontaneously; almost as often, we surgeons will oblate or bypass them with something akin to cavalier abandon. Under either circumstances, patients may suffer.

For the past twenty years, I have had an interest in the influence of reflux of upper intestinal content on the form and function of the stomach. I believe this has merit because there are at least three clinical disorders of the gastric mucosa which are potentially related to excessive enterogastric reflux. They include: posttraumatic stress ulceration, type I benign gastric ulcer, and the putative postgastrectomy syndrome, postoperative alkaline reflux gastritis. Despite the differing presentations and treatments of each of these disorders, they do share two common clinical features. First, there is no evidence of hypersecretion of hydrochloric acid in any of them; and second, when the parameter is measured, they demonstrate an impaired ability to maintain hydrochloric acid within the gastric lumen, that is, the normal *barrier* to *acid back diffusion* is impaired. The original conceptualization of that barrier by the great Michigan physiologist, Horace Davenport, has never been improved upon: under ordinary circumstances, in response to a variety of stimuli, the parietal cells secrete hydrochloric acid which travels up the gastric pit to arrive in the gastric lumen in enormous concentration relative to subjacent tissue. In any other semipermeable membrane of the body, the concentration gradient (10^7M) would be sufficient to drive enormous amounts of acid from lumen toward blood. Yet, in the presence of a normal barrier, only small amounts of hydrochloric acid are diffused down this concentration gradient. Thus, a *barrier* to *acid back diffusion* exists. That barrier can be impaired, however, by a number of topically applied noxious agents with the consequence that large amounts of acid diffuse back into the tissue with a variety of postulated consequences, including the appearance in large quantities in luminal solutions of an ultrafiltrate of plasma. Those consequences can be measured under rigidly controlled experimental conditions as a marked net luminal loss of H^+ ion associated with a marked net luminal gain of Na^+ ion.

A variety of other resistance factors of importance also exists. They include the secretion of mucus which is rich in bicarbonate, an intact monolayer of the cells

at greatest risk, the surface epithelial cells, the ability of the gastric mucosa to modulate mucosal blood flow, ion exchange mechanisms (H^+ for Na^+, HCO_3^- for C_1^-) the capacity of the gastric mucosa to produce a HCO_3^- ion for every H^+ ion secreted, and the mysterious property of the gastric mucosa to be non-wettable, referred to as surface hydrophobicity.

What factors in upper intestinal content can affect mucosal defenses? There are three likely candidates. The first, pancreatic enzymes, have been studied only to a limited extent and, in general, have been found to be innocuous, probably because they are inactivated at low pH. On the other hand, lysolethicin may play a major role in that it has been demonstrated to increase mucosal permeability *in vitro,* to enhance H^+ *back diffusion in vivo,* and to produce acute damage to surface epithelial cells *in vivo.* My own interest has focused almost exclusively on the role of the bile acids in this regard.

The topical application of bile acids to the gastric mucosa have been found to have major effects on the resistance mechanisms of the gastric mucosa. First, they induce excessive H *back diffusion.* This effect is not only concentration-dependent but is also species-dependent: the dihydroxy, secondary, bile acids are more damaging than are the trihydroxy, primary bile acids. It is also pKa and therefore pH dependent: taurine conjugates are more damaging at low pH, glycine conjugates are more damaging at high pH. Bile acids also inhibit HCO_3 production *in vitro* and *in vivo,* inhibit carbonic anhydrase activity *in vivo,* inhibit sodium absorption, and impair lysosomal stability, particularly of the antrum. On the other hand, they do not appear to have any effect on mucus structure, thickness, or pH barrier properties.

The purposes of the studies to be described were three-fold: first, to develop a model of bile acid-induced acute physiologic and morphologic gastric mucosal damage; secondly, to define the factors responsible for modulating the severity of that injury; and, finally, to use the model to assess the "cytoprotective" potential of various pharmacologic agents.

Methods

The experimental preparation used has been well described elsewhere. Briefly, healthy adult mongrel dogs were employed. Under general anesthesia, through a midline abdominal incision, a full thickness wedge of proximal gastric wall pedicled on the left gastroepiploic artery and vein was excised, everted, and mounted between lucite rings. The upper ring served as a reservoir for the gastric mucosa into which artificial gastric juices could be introduced. In several studies, the superior branch of the splenic artery was cannulated for the infusion of organ-specific pharmacologic agents. A splenectomy *in situ* was performed to reduce the distribution volume of the drugs. The chamber and its contents were then secured in place using ring clamps close to the anterior abdominal wall.

The artificial gastric juices employed included a neutral test solution (160 mM NaC_1 containing C_{14} PEG and carrier PEG, adjusted to pH 7), an acid test solution (60 mM NaC_1, 100 mM HC_1, also containing PEG, pH = 1.2) and either an acid or a neutral test solution containing 2.5 mM or 5 mM sodium taurocholate. Parameters evaluated included net ion fluxes (a positive prefix indicated net luminal gain, a negative prefix net luminal loss), mucosal blood flow, initially using the aminopyrine clearance technique, subsequently using radio labelled microspheres, and the lesion index, a measure of the severity of gross mucosal injury induced.

Studies and Results

One of the first studies undertaken sought to investigate the pathogenesis of stress ulcer disease. It asked the question: What is the role of intraluminal acid, mucosal ischemia, and reflux (i.e., bile acids) either alone or in combination, in the production of acute gross mucosal injury?

Two groups of experimental animals were studied during three consecutive 30-minute study periods. The mucosa of group A was subjected during period one to topical acid test solution, during period two to acid test solution during organ-specific infusion of a low dose of vasopressin, and during period three to acid, bile acid, and vasopressin infusion. The mucosa of group B was subjected to acid test solution during period one, to acid test solution and bile acid during period two, and to the combination of all three factors, acid, ischemia and bile acid during period three. In each control group, the mucosa was exposed to two but never all three of the factors under consideration.

The results: in mucosa exposed to ATS alone, there was a small net loss of hydrogen ion associated with a mucosal blood flow (assessed using aminopyrine clearance) of approximately 50 ml/30 minutes. In mucosa exposed to acid and bile acid, there was a marked increase in net hydrogen ion loss but this was associated with an almost doubling of mucosal blood flow. In mucosa exposed to acid and ischemia, there was again a small net loss of hydrogen ion (not significantly different than in mucosa exposed to acid alone) but mucosal blood flow was markedly reduced. In mucosa exposed to all three factors, a marked increase in net hydrogen ion loss was noted (not different than that seen in nonischemic mucosa under the same circumstances). However, mucosal blood flow was markedly depressed. It was possible to express the relationship of hydrogen ion loss to mucosal blood flow as a ratio. Under the first three circumstances, the ratio was invariably low. Under the last circumstance, however, the ratio was extremely high and only under these circumstances was a severe degree of mucosal injury apparent. It was clear from these studies that the increase in mucosal blood flow noted when topical acid and bile acid are applied to the gastric mucosa is an important protective event because, when it and it alone was blunted (as with ATS + VP + TC), severe gross mucosal injury resulted.

We next asked the question: What is the approximate cause of this protective mucosal blood flow response? Our hypothesis was that mucosal acidification was involved. The assumption was made that differing masses of luminal H^+ loss would be reflected in differing intramucosal pH's, as this last parameter could not be measured at the time. The experiment involved producing differing amounts of luminal H^+ loss by varying the hydrogen ion concentration of the acid test solution at constant concentration of bile acid and by varying the concentration of bile acid at constant concentration of acid. Mucosal blood flow was then measured using microspheres. The results demonstrate a significant increase in mucosal blood flow (the dependent variable) with increasing H^+ loss from the lumen (the independent variable). This

relationship was significant with a very high correlation coefficient. These data indicated that the proximate cause of the protective increase in mucosal blood flow seen following the topical application of acidified bile acids to the gastric mucosa was H^+ loss, per se.

The next question to be addressed was: What are the endogenous mediators of this protective mucosal blood flow response? Our hypothesis was that endogenous prostaglandins generation was involved in some fashion. Therefore, we undertook a study, the purpose of which was to assess their role in the blood flow response by assessing the effect on it of cyclooxygenase inihibition with indomethacin and of cyclooxygenase inihibition plus added organ-specific influsion of prostacyclin (PGI_2).

Four groups of dogs were studied during three sequential 30-minute study periods. The mucosa of Group A was subjected, first, to ATS, then to ATS containing 2.5 mM TC, and finally to acid test solution containing 5 TC. The mucosa of Group B was treated similarly except that all animals were pretreated with indomethacin, 10 mg/kg i.v., 20 minutes prior to the initiation of period one. Group C animals were treated in exactly the same fashion as Group B except that PGI_2, 0.05 mcg/kg/min, was infused directly into the blood supply of the wedge beginning five minutes prior to period one. The mucosa of Group D was exposed, during period one, to neutral test solution, during period two, to NTS + 2.5 TC, and, during period three, to NTS + 5 TC.

The results indicated that, in mucosae exposed to neutral test solution, there is no net movement of H^+ irrespective of bile acid concentration and, concomitantly, mucosal blood flow remains stable at approximately 20 ml/min/100 gms of tissue. In mucosae exposed to acid test solution, however, there was a stepwise increase in H^+ loss with increasing concentrations of bile acid. This was associated with a stepwise increase in mucosal blood flow. This response was specific for the mucosa as no alterations were noted in muscularis blood flow.

Pretreatment with indomethacin increased net H^+ loss slightly in the presence of acid and bile acid. Despite this fact, however, resting mucosal blood flow was significantly reduced (to approximately 10 ml/min/100 gms of tissue) and, more importantly, the mucosal blood flow response seen in indomethacin-pretreated animals exposed to acid and bile acid was significantly blunted. As a consequence, a stepwise increase in gross mucosal injury was observed at all bile acid concentrations.

The simultaneous infusion of prostacyclin to indomethacin-pretreated animals did not alter net H^+ loss relative to indomethacin alone. However, a marked increase in mucosal blood flow (greater than 150 ml/min/100 gms of tissue) was observed with the result that total protection was provided.

These data suggest that generation of endogenous prostaglandins may be responsible, at least in part, for the protective increase in gastric mucosal blood flow observed after the topical application of bile acids at low pH.

We next asked the question of whether or not, in the absence of ischemia, reflux was innocuous? Our hypothesis was that it was not, especially not to surface epithelial cells. We therefore designed a study whose purpose it was to assess surface epithelial cell injury using scanning and transmission electron microscopy in mucosae exposed to neutral test solution, neutral test solution plus bile acid,

acid test solution, and acid test solution plus bile acid. We also attempted to quantitate the magnitude of SEC injury by determining the amount of DNA appearing in the lumen under the same conditions. Three modes of mucus release were identified in mucosae exposed to NTS, NTS + TC, and ATS. They were: exocytosis (individual mucous granules gaining access to the lumen through the apical membrane of the surface epithelial cell); apical expulsion (a fused plug of mucous granules discharged en mass into the lumen leaving a defect in the apical portion of the cell which, however, still had a limiting membrane and was quite viable); and exfoliation (individual, presumably older, surface cells gradually extruded into the lumen, usually from the apices of the intrafaveolar ridges).

On the other hand, mucosae exposed to ATS + bile acid demonstrated severe damage to the surface epithelial cells: extensive exfoliation of entire sheets with exposure of the underlying basement lamina. This injury could be quantitated by measuring the appearance of DNA in the luminal solution bathing the chambered mucosa. DNA appearance was minimal in the presence of NTS, irrespective of bile acid concentration (approximately 50 micrograms per 30 minutes) but increased dramatically in the presence of ATS at both 2.5 mM TC (approximately 180 micrograms per 30 minutes) and ATS + 5 TC (approximately 450 micrograms per 30 minutes). Furthermore, significant linear collelations were noted between DNA efflux into the lumen and net H^+ loss ($R = 0.95$) and net sodium gain ($R = 0.93$).

These data indicate that, when exposed to NTS, NTS + TC, and ATS, surface epithelial cells demonstrate exocytosis, apical expulsion, and single cell exfoliation but are viable. However, when exposed to ATS + TC, surface epithelial cells demonstrate massive exfoliation which is measurable using DNA efflux calculations. DNA efflux correlates directly with luminal H^+ loss suggesting that an intact monolayer of SECs may be the anatomic site of the "gastric mucosal barrier."

We have also attempted to assess the effect of potentially protective pharmacologic agents on many of the parameters already discussed. In particular, we recently completed a study in which we assessed the effect of topically applied 16 16 dimethyl PGE_2 (10 micrograms) on bile acid induced injury using a double chambered technique. During a 30-minute equilibration period both the test chamber and control chamber were exposed to neutral test solution. This was followed by a 15-minute pretreatment period with $DMPGE_2$ in the test chamber, with vehicle in the control chamber. The mucosae of both chambers were then exposed for 15 minutes to acid test solution and ion fluxes and DNAE were determined. The entire cycle was then repeated, concluding with a 15-minute flux period in which mucosae were exposed to acid test solution containing 5 TC.

The results indicated that, in mucosae exposed to acid test solution alone, there were no differences in Δ H^+ or in DNAE. However, a significant increment in sodium appearance and volume gain were noted in the $DMPGE_2$ pretreated chamber. This has been termed the "teardrop effect" which might serve to dilute the concentration of noxious agents in contact with the apical surfaces of the cells at greatest risk, the SECs.

In mucosae exposed to ATS + 5 TC, pretreatment with $DMPGE_2$ resulted in a significant reduction in both net H^+ loss and DNA appearance in the lumen. Changes in volume and net sodium flux were in the same direction as those observed

in mucosae exposed to ATS alone but did not reach the level of statistical significance. We concluded that, in mucosae exposed to ATS + TC, 16 16 $DMPGE_2$ reduced H^+ "loss" and efflux of surface cell DNA into the lumen. Thus, it appears to be "cytoprotective."

I appreciate more than I can say the opportunity which Dr. Barker has provided me of simultaneously reviewing a lifetime of modest but very pleasurable scientific effort and, at the same time, of paying homage to Dr. Rhoads. I believe that we have made considerable strides in improving our understanding of the mechanisms by which the gastric mucosa protects itself and are likely to increase that understanding substantially in the future because, as my former mentor, Oewn H. Wangensteen, M.D., who was also a good friend of Dr. Rhoads', was fond of saying, "Disease is very old; nothing about it has changed. It is we who change as we learn to recognize what was formerly imperceptable."

Annual Oration for 1990

A Venous View of Abdominal Aortic Aneurysm Surgery

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In the United States and in most industrialized nations, abdominal aortic aneurysm is being diagnosed and treated frequently because of the ease of diagnosis and the increase in longevity of populations. Abdominal ultrasonography and CAT Scanning have greatly increased our ability to diagnose this disease; abdominal aortic aneurysm surgery is now a common vascular operation. Despite improvements in diagnosis and in the surgical management of these patients, abdominal aortic aneurysm represents the thirteenth most common cause of death in the United States and accounts for 15,000 deaths per year. In addition to a real increase in incidence, there is also an increased mortality rate for these lesions.[1,2]

When the diagnosis of abdominal aortic aneurysm is made, the surgeon typically follows a rather standard process of evaluation. First, he must consider the cardiac, pulmonary, renal, and cerebral condition of the patient. If the patient is found to be a reasonable risk, the second step is to consider various technical aspects (i.e. a transperitoneal vs a retroperitoneal approach, prevention of colonic ischemia, prevention of prosthetic infection, exclusion and remote bypass, use of blood and its products, etc). The third hurdle for the surgeon is to consider the associated diseases, and these can include renal abnormalities such as the horseshoe kidney, the inflammatory aneurysm, occlusive disease of renal arteries, intra-abdominal visceral disease, occlusive disease of the visceral arteries, sexual disorders, associated aneurysms in the femoral and popliteal area, and occlusive disease of the lower extremity. Most of these associated diseases can be diagnosed preoperatively and plans made accordingly. The fourth and final set of circumstances that the surgeon faces is the management of the so-called "complicated aneurysm." This group includes the ruptured aneurysm, primary aorto-duodenal fistula, infected aneurysm, juxtarenal aneurysm, and a group listed as "aneurysms with venous problems." It is this last group of problems that is the focus of this presentation.

When one considers the factors above, it is apparent that surgery for abdominal aortic aneurysm is rarely straightforward; it does not lend itself to a "cookbook-type" approach. A study of the venous side of abdominal aortic aneurysm surgery is interesting, but more importantly as most surgeons know, when massive and uncontrolled bleeding occurs it is often due to major venous injury. The venous aspects of abdominal aortic aneurysm surgery can be classified and will be discussed as follows:

1. The Major Venous Anomalies
2. Aorto-Caval Fistulas and Aorto-Venous Fistulas
3. Left Renal Vein Division
4. Portal Hypertension in the Presence of the AAA

The Major Venous Anomalies

Iatrogenic injury to a major venous anomaly (MVA) during abdominal aortic surgery can result in massive and often lethal hemorrhage.[3-7] Until recently these venous anomalies were rarely diagnosed preoperatively and were usually discovered intraoperatively or after venous injury had occured. With the advent of ultrasonography and CT scans, detailed knowledge of MVA's is now possible preoperatively.[7,8]

Embryology. There are four major clinical anomalies that involve the inferior vena cava (IVC) and left renal vein (LRV). These include a retroaortic LRV, a circumaortic renal collar, duplication of the IVC and transposition of the IVC (or left-sided IVC). There are other variations of the venous system in this part of the body that involve the lumbar, azygos, and ascending lumbar venis, but they are not of any clinical signficance to the vascular surgeon since they are located behind the iliopsoas muscles and are not encountered during routine vascular reconstruction of the abdominal aorta.[3,7,8]

The IVC and renal veins develop from modification of three parallel pairs of embryologic veins: the postcardinal, the subcardinal and the supracardinal veins. These transformations occur during the fourth to eighth week of embryologic development. The postcardinal veins develop in association with the mesonephros dorsal to the aorta and anastomose with the subcardinal veins. The postcardinal veins persist only as the iliac vein bifurcation. The right subcardinal system eventually develops into the suprarenal IVC and a segment of the infrarenal IVC. The supracardinal veins form in a plane dorsal to the aorta and anastomose with the ventral subcardinal system to form a collar of veins that surrounds the aorta. The right supracardinal vein normally persists and the left supracardinal vein regresses resulting in a normal infrarenal IVC to the right of the aorta. The ventral part of the circumaortic ring persists and the dorsal segment regresses to form the normal LRV found anterior to the aorta.

Anomalies occur when the left supracardinal vein persists resulting in either a double IVC, or if the right supracardinal vein regresses, a single transposed (left-sided) IVC. In the presence of a double IVC, the LRV drains into the left IVC which then continues cephalad to drain into the right IVC. Also, the entire circumaortic ring may persist and form a circumaortic renal collar, or if the ventral portion regresses, a retroaortic LRV.

Materials and Results

Between January 1967 and July 1990, 605 patients were operated upon for an abdominal aortic aneurysm and 508 patients underwent aortic reconstruction for aortoiliac occlusive disease. During this period, 22 (2.0%) MVA's were found intraoperatively.

Retroaortic LRV (13 patients): In one patient a retroaortic LRV was not at first recognized during proximal dissection of a ruptured abdominal aortic aneurysm. The vein was lacerated which led to massive hemorrhage and death. Another patient with a ruptured abdominal aortic aneurysm was found to have a fistula between the aneurysm and a retroaortic LRV. A great deal of venous bleeding was found after the aneurysm was entered. The venous defect was oversewn from within the aneurysm and the patient did well. The emergent need for surgery in these patients prohibited obtaining preoperative CT scans. In the other 11 patients, all retroaortic LRV's were recognized either intraoperatively or with preoperative CT scans and inadvertent injury was avoided.

Duplication of the IVC (3 patients): One patient had a small left-sided portion of the IVC which passed anterior to an abdominal aortic aneurysm to join the LRV. The left-sided duplicated vein was ligated below its junction with the LRV. In the second patient the left-sided segment joined the LRV more laterally and thus ligation of the left IVC was unnecessary for proximal aortic control. The third patient had a large abdominal aortic aneurysm and during mobilization of the aneurysm to gain proximal control, a duplicate IVC located posterior and to the left of the aorta was injured. The laceration was controlled and the aneurysm was resected without further complications. An ultrasound had been obtained preoperatively in this last case and had not visualized the MVA.

Circumaortic renal collar (4 patients): One patient who did not have a preoperative CT scan sustained an injury to the retroaortic component of a renal vein collar which led to massive hemorrhage. This was eventually controlled and the patient survived. The other three anomalies were recognized and required no special treatment.

Transposition of the IVC (2 patients): In both patients these MVA's were recognized at the time of surgery and no injury occurred. Division of these left-sided cavae was not necessary and reconstruction was carried out by gentle retraction.

Management

When an MVA is identified either pre- or intraoperatively, we suggest the following guidelines to avoid massive venous hemorrhage.

Transposition of the IVC: If a left-sided IVC is retroaortic, then injury can be avoided by careful mobilization of the proximal aorta and iliac vessels. In the presence of the more common *pre-aortic* left-sided IVC[4], retraction of the vein to the left and cephalad retraction of the LRV may provide sufficient exposure of the infrarenal aorta. If this maneuver does not produce adequate exposure, the IVC may be transected below the LRV and then the proximal portion mobilized cephalad. However, this may lead to massive bilateral lower extremity venous thrombosis and chronic edema. A third techniuqe would be to transect the *right* renal vein close to its junction with the left-sided IVC. The right renal vein in the presence of transposition of the vena cava usually has the same collateral venous drainage found in the normal LRV system. A left-sided IVC can often be detected with a preoperative CT scan.

Duplication of the IVC: If the left side of a double IVC is posterior, careful dissection and avoidance of the posterior portion is warranted. If the left side of the system is anterior, the left IVC can be transected below the confluence with the LRV. The portion of the left IVC that crosses the aorta to join the right-sided IVC can then be reflected superiorly. Another technique to manage this anomaly would be to transect the left-sided IVC where it crosses the aorta proximal to its junction with the suprarenal IVC. With this maneuver, the LRV can drain into the distal left IVC.

Retroaortic left renal vein: During exposure of the proximal infrarenal aorta, one should carry the dissection cephalad until the LRV is identified crossing the aorta anteriorly. Preoperative CT scans can usually document this normal course of the LRV. If the LRV cannot be found intraoperatively, the surgeon should identify the IVC in its normal position to the right of the aorta, and if present assume that the LRV lies behind the aorta. A retroaortic LRV usually has an oblique course caudally to join the IVC and proximal aortic control can usually be obtained above the visualized retroaortic LRV. Although in most circumstances we favor circumferential control of the proximal infrarenal aorta, vertical placement of an aortic clamp proximal to a retroaortic LRV may be preferable.

Circumaortic renal collar: In the presence of an anterior LRV, the surgeon must still be alert for a circumaortic renal collar. For example, occasionally we divide and ligate the LRV in order to get good aortic control of an aneurysm. Prior to doing this we routinely measure LRV stump pressures.[10] In one patient who required LRV division, the stump pressure did not increase and equalled the central venous pressure. After division of the vein and further dissection we discovered that the patient had a circumaortic renal collar with a large retroaortic component which had decompressed the anterior part of the venous collar resulting in no rise in stump pressure. A left renal vein that is very adherent to the anterior aorta or a small, anterior LRV should alert the surgeon to the possibility of a circumaortic renal collar. Control of the aorta in the presence of this anomaly may be obtained above or below the vein.[5] If the collar is very tight and does not allow safe mobilization for proximal aortic control, the anterior segment of the collar may be divided at the confluence with the IVC. The posterior component of the collar courses caudad and obliquely before joining the IVC (similar to a retroaortic LRV). A preoperative CT scan can identify a circumaortic renal collar, but it is a most difficult anomaly to detect even on CT scan because the anterior component appears as a normal LRV and the posterior component is found several centimeters lower and rarely at the same level.

Discussion

MVA's in the vicinity of the abdominal aorta are unusual intraoperative findings. In Baldridge's review of clinical and autopsy series, the incidence of circumaortic renal collars was 1.5-8.7%, duplication of the IVC 2-3%, retroaortic LRV's 2%, and transposition of the IVC less than .5%.[4] There is a wide variation in the reported prevalence of these anomalies depending on the fastidiousness of the surgeon performing the dissection and whether the figures are based on intraoperative or postmortem findings. With CT scanning the actual incidence of venous anomalies

will be better documented. Our incidence of MVA's of 2.0% (22/1,113) based on intraoperative findings is very similar to other clinical reports.[5]

In our experience, these major anomalies were encountered more frequently during surgery for abdominal aortic aneurysm (15/605 [2.5%]) than with occlusive aortic disease (7/508 [1.4%]). We suspect that this difference reflects the more extensive dissection carried out during abdominal aortic aneurysm surgery, especially around the renal vein. Therefore, some anomalies are probably not detected during surgery for occlusive aortic disease because of the limited dissection in these patients.

Preoperative identification of MVA's is now possible with ultrasonography, nuclear magnetic resonance imaging and CT scanning.[8,9,11] The latter shows detailed anatomy of these anomalies. Giordano and Trout point out that preoperative identification of these anomalies is important not only prior to aortic surgery but also before inferior vena cava interruption, lumbar sympathetomy, portosystemic shunts, and venous sampling.[4] MVA's can be mistaken for retroperitoneal or intraabdominal tumors but the use of dynamic bolus CT scanning can usually accurately identify the lesion. The advantage of nuclear magnetic resonance imaging is in the patient with borderline renal function where these venous anomalies can be detected without using contrast material.[11]

In the present era of cost containment and high litigation rates, a controversial aspect in the management of patients about to undergo abdominal aortic aneurysm surgery is the role of routine aortography and CT scanning. Many competent vascular surgeons have performed abdominal aortic aneurysm surgery for years without the use of these adjunctive diagnostic tests. Although the less experienced surgeon may feel a greater need for these preoperative studies, we recommend the following general guidelines. Ultrasonography is a well-accepted modality to diagnose and follow small abdominal aortic aneurysms, but this method is not as accurate as CT scanning to identify MVA's.[8,9] We believe that the poor-risk or otherwise complicated patient with an aortic aneurysm should undergo both aortography and CT scanning preoperatively to identify any unexpected intra-abdominal anomalies so that the surgery may be carefully planned.[12] Presently, abdominal ultrasonography is one of the first diagnostic tests for almost any patient with a serious abdominal complaint or sign. Consequently, many incidental abdominal aortic aneurysms are found and are followed at intervals until they reach a size of 4.5 to 5 cm, at which time resection is recommended. We have been recommending one routine CT scan of the abdomen in lieu of regular interval ultrasounds in order to acquire more anatomic knowledge (i.e. venous anomalies, iliac aneurysms, suprarenal extension, dissection).

Summary

Our experience with surgery of the abdominal aorta indicates that MVA's of the IVC and LRV are uncommon and occur about 2% of the time. CT scanning will be able to better determine the incidence of these anomalies. We recommend that any poor-risk patient about to undergo surgery for an abdominal aortic aneurysm or aortoiliac occlusive disease have a preoperative CT scan. If not recognized preoperatively, the likelihood of inadvertent injury to an MVA is increased and can

lead to massive hemorrhage and possibly death. Vascular surgeons must be aware of the normal and anomalous anatomy of major veins near the abdominal aorta to avoid these devastating complications. The most common lesion encountered in our series was a retroaortic LRV. Most MVA's are still best recognized by meticulous dissection of the proximal infrarenal aorta and an increased awareness of their presence.

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Aorto-Caval Fistulas and Aorto-Venous Fistulas

Spontaneous aortovenous fistula (AoVF) between an abdominal aortic aneurysm (AAA) and an adjacent posterior vein is a rare and dangerous complication, especially if it is not suspected preoperatively. When an AoVF is present in a patient with a ruptured AAA, preoperative testing to confirm the diagnosis of an AoVF is rarely possible. The incidence of an aortocaval fistula (AoCF) secondary to AAA's is approximately 1-2% but this incidence increases to 2-4% in the presence of ruptured AAA's.[1-13] Aortorenal vein fistula (AoRF) and aortoiliac vein fistula (AoIF)[3,6,19-22] are much less common.

The purpose of this section is to describe three patients with extremely large AAA's that spontaneously ruptured into the retroperitoneum *and* an adjacent posterior vein; to report several unusual characteristics of these AoVF's, which involved the left renal vein, the iliac vein and the inferior vena cava (IVC); and to review the pathophysiology and operative management of these complications. A review of the English-language literature (Medline access through BRS) of patients taken to surgery with spontaneous abdominal AoVF's through 1989 is included. Fistulas forming as a result of trauma or between the iliac artery and adjacent veins are not included in this review.

Between January 1982 and July 1989, 221 patients were operated on for AAA's at Pennsylvania Hospital. Three of 16 patients with aneurysms that ruptured into the retroperitoneum also had an AoVF. An AoVF was not found in the remaining 205 patients.

CASE 1. A 66-year-old white male collapsed at home after acute onset of low back pain. On physical examination at an emergency room a pulsatile abdominal mass was palpated and the patient was transferred 80 miles to our hospital with a diagnosis of a ruptured AAA. The patient had a mildly tender pulsatile epigastric mass but no abdominal bruits, thrills or distended superficial veins. He had palpable bilateral femoral artery aneurysms. There was no evidence of congestive heart failure or an elevated pulse pressure to suggest a major arteriovenous fistula. The patient was taken immediately to the operating room for laparotomy at which time a 13 cm AAA and a large retroperitoneal hematoma was found. The autotransfusion cell-saving technique was used throughout the operation. After proximal and distal control of the aorta, the aneurysm was entered and an aortocaval fistula with massive venous hemorrhage was discovered. A 2 cm erosion in the right posterior aortic wall was oversewn with a continous 3-0 Prolene suture from within the aneurysm followed by placement of an aortobifemoral graft. Except for development of a urinary tract infection which required prolonged intravenous antibiotics the patient recovered and was discharged on the 25th postoperative day. He is doing well eight years later.

CASE 2. A 72-year-old white male complained of lower back and abdominal pain for two days' duration before seeing his private physician. A large, pulsatile, periumbilical abdominal mass was palpated but there were no other abnormal physical findings. The patient's blood urea nitrogen was 26 mg/dl, serum creatinine was 1.8 mg/dl and a urinalysis showed many bacteria with a few white and red

blood cells. A CT scan documented a 14 cm AAA with a questionable small posterior leak. The patient was transferred to Pennsylvania Hospital and immediately taken to the operating room. At laparotomy a small retroperitoneal hematoma was found. The absence of an anterior left renal vein was noted. After opening the aneurysm, massive venous hemorrhage was encountered due to a large fistula involving a retroaortic left renal vein. Direct finger pressure and repair from within the opened aneurysm staunched the bleeding. The cell-saver was used to restore the blood volume. An aortobifemoral graft was placed because the aneurysm occupied most of the pelvis. The patient recovered uneventfully and was discharged on the tenth postoperative day.

CASE 3. A 70-year-old, 340-pound white male was transferred to our emergency room from another medical center 60 miles away after he developed acute back pain and an abdominal x-ray showed a 12 cm AAA. At laparotomy an inflammatory aneurysm was found that had ruptured into the retroperitoneum. An aortobifemoral graft was placed due to the presence of bilateral iliac aneurysms and stenoses. A great deal of venous bleeding was suddenly noted welling up from the distal, posterior aortic aneurysm wall. A fistula to the proximal left iliac vein was identified and oversewn with a running 3-0 Prolene suture. Presumably thrombus had temporarily occluded the fistula. The cell-saver was used throughout the procedure. Other than mild respiratory insufficiency, the patient did well postoperatively until the twentieth postoperative day when he developed a left groin infection with involvement of the distal anastomosis of the left limb of the graft. This was treated with excision of the distal limb of the graft, oversewing of the proximal uninvolved part of the graft and the common femoral artery, and a bypass from the left axillary artery to the above-knee popliteal artery dissected via a lateral approach. The patient later developed multiple complications including pneumonia and renal failure and expired on the 95th postoperative day.

Discussion

Pathogenesis: In the English-language literature, there are reports on 159 patients with an AoCF, 17 patients with an AoRF and 8 patients with an AoIF who underwent surgery after spontaneously developing these complications. Pressure and tension related necrosis of the posterior wall of large AAA's has been postulated to cause an intense adventitial inflammatory reaction resulting in adherence of adjacent posterior veins.[2] Fistulas developing between the aorta and IVC, iliac vein and retroaortic left renal vein may then develop.

A striking finding in our three patients was the extremely large size of the AAA's with an average diameter of 13 cm. When we include our cases, a review of the English-language literature reveals that the average diameter of AAA's complicated by an AoVF is 11 cm (range = 4-20 cm). The vast majority were greater than 6 cm in diameter. Similarly, the average diameter of *iliac artery aneurysms* complicated by venous fistulas as reported in a review by Morrow was 9 cm.[23] AoVF's forming secondary to very large AAA's supports the premise that pressure-related necrosis of the posterior aneurysm wall is the underlying etiology of these complications.

There have been four other reported cases of an AoVF forming in association with an inflammatory AAA.[5,13,24] Although the intense perioaortic inflammatory reaction of this entity would seem to be protective, we and others have shown that inflammatory aneurysms have a thickened "peel" of fibrous tissue only *anteriorly* and can and do rupture posteriorly.[25] Since AoVF's are almost always located posteriorly, one would therefore expect patients with inflammatory AAA's to also be susceptible to develop these fistulas.

Clinical characteristics. When an AoCF forms, a continuous abdominal bruit is present in 71% of patients, low-back or abdominal pain in 83%, and because of the very large size of the AAA's found with fistulas, a pulsatile abdominal mass in 89% (Table 1). Shunting of large amounts of blood into the low resistance venous system results in clinical evidence of high-output cardiac failure, pulmonary edema, mottling or swelling of the lower extremities, and dilated neck or superficial abdominal wall and leg veins in 58% of patients. AoIF's can present with similar manifestations (Table 2).[3,6,19-22]

A *retroaortic* left renal vein is present in approximately 3% of the population and usually courses in a caudal direction from left to right where it joins the IVC.[26] If we include our patient all but one of the AoRF's were to a *retroaortic* left renal vein and all 17 patients were *male* (Table 3). The sole patient with an anterior left renal vein had a fistula form between the proximal, overhanging part of a very large aneurysm and the underlying vein. Hematuria occurred in 94% (16/17) of patients with an AoRF, but overt signs of high-output cardiac failure are less likely because of the smaller size of the renal vein compared to the IVC and iliac vein.[14,18]

Table 1: **AORTOCAVAL FISTULA***

	Percentage	**#**	**Average** (range)
Number of patients		159	
Age		142	65 y.o. (14-91)
Males	97%	144/148	
Females	3%	4/148	
Palpable mass	89%	127/143	
Aneurysm size		62	11 cm (4-20)
Pain	83%	119/143	
Bruit	71%	96/136	
Shunt	58%	84/144	
Hematuria**	46%	37/80	
Retroperitoneal rupture	20%	27/132	
Inflammatory aneurysm	3%	4/144	
Survival	72%	107/149	

*Data based on 79 references and this report (159 total patients). Each patient characteristic was not reported by all authors accounting for the denominator of the ratio used to calculate the percentages being less than the total number of patients.

**Micro- or gross hematuria.

Table 2: **AORTOILIAC VEIN FISTULA***

	Percentage	**#**	**Average** (range)
Number of patients		8	
Age		8	68 y.o. (60-75)
Males	63%	5/8	
Females	37%	3/8	
Right iliac vein	63%	5/8	
Left iliac vein	37%	3/8	
Palpable mass	100%	7/7	
Aneurysm size		5	11 cm (7-15)
Pain	86%	6/7	
Bruit	57%	4/7	
Retroperitoneal rupture	43%	3/7	
Shunt	38%	3/8	
Inflammatory aneurysm	14%	1/7	
Hematuria**	13%	1/8	
Survival	71%	5/7	

*Data based on 6 references and this report (8 total patients). Each patient characteristic was not reported by all authors accounting for the denominator of the ratio used to calculate the percentages being less than the total number of patients.

**Micro- or gross hematuria (13% incidence may not reflect an accurate value since only one case report specifically mentioned the presence or absence of this finding).

Table 3: **AORTORENAL VEIN FISTULA***

	Percentage	**#**	**Average** (range)
Number of patients		17	
Age		17	64 y.o. (55-77)
Males	100%	17/17	
Non-filling of left kidney (IVP pre-op)	100%	13/13	
Hematuria**	94%	16/17	
Retroperitoneal left renal vein	94%	16/17	
Pain	82%	14/17	
Palpable mass	70%	12/17	
Aneurysm size		12	10 cm (6-15)
Bruit	63%	10/17	
Retroperitoneal rupture	12%	2/17	
Shunt	6%	1/17	
Survival	88%	14/16	

*Data based on 15 references and this report (17 total patients). Each patient characteristic was not reported by all authors accounting for the denominator of the ratio used to calculate the percentages being less than the total number of patients.

**Micro- or gross hematuria.

Sufficient information was available to judge if a shunt was present or absent in 21 patients with an AoCF associated with a *ruptured* AAA. Of these patients, 12 (43%) did not show signs suggestive of a fistula. Each of our patients had preoperative clinical evidence of a ruptured AAA (abdominal pain, pulsatile mass) but no findings suggestive of an AoVF. We believe this is due to aneurysmal clot occluding the fistula or hypotension which would mask the above-mentioned signs and symptoms.[21]

Diagnostic Methods

A suspicion of a patent (not occluded by clot) AoVF in the case of an intact AAA can usually be confirmed preoperatively. Various diagnostic techniques include B-mode ultrasound and doppler ultrasonography,[6] CT scan,[8] and passage of a balloon-tipped catheter into the IVC through the femoral vein to document elevated venous pressures and oxygen levels.[9] An intravenous pyelogram documented a non-functioning left kidney in all thirteen patients with an AoRF who had this diagnostic test obtained preoperatively.[14-18] Interestingly, intravenous pyelograms obtained postoperatively in six of these patients documented return of left kidney function after closure of the fistula in all six cases. We have reported that elevated left renal vein pressures greater than 50-60 cm water will usually cause marked deterioration in left kidney function.[27] Increased pressure as a result of an AoRF may account for lack of left renal function in these patients. Although angiography remains the best method to document the presence of AoVF's, the diagnosis may not be apparent if clot occludes the fistula.[21] The need for emergent surgery in the patient with a ruptured AAA often precludes obtaining these tests. The CT scans that were obtained in two of our cases did not suggest the presence of an AoVF.

Mortality Rate in Aortovenous Fistulas: Seventy-two percent (112/156) of patients with AoCF's or AoIF's and 88% (14/16) of patients with AoRF's survived surgery (Tables 1-3).[14-18] The better prognosis with AoRF's is most likely due to an associated lower incidence of ruptured aneurysms since only 12% (2/17) of patients with an AoRF had a separate retroperitoneal rupture compared to 22% (30/139) of patients with an AoCF and AoIF who also had a retroperitoneal rupture.

Surgical Management: There are at least five essential steps to the successful repair of an AoVF secondary to a ruptured AAA. 1) Proximal and distal control of the infrarenal aorta is obtained. Supraceliac control is often necessary in the presence of these large aneurysms. In the case of an AoRF, dissection of the infrarenal aorta should be performed under direct vision to avoid inadvertent injury to a retroaortic left renal vein. In most circumstances, an aortic clamp can be applied proximal to the vein and distal to the renal arteries since retroaortic left renal veins usually course caudally.[26] 2) Proximal compression of the IVC can be performed to avoid air and blood emboli. 3) Strategy to apply compression to control massive venous hemorrhage from the fistula should be developed before the aneurysm is opened. In the case of an AoRF, proximal and distal control of a retroaortic left renal vein is treacherous and should *not* be attempted. In this situation, direct compression of the left renal vein from within the aneurysm lumen should be carried out. In fact,

circumferential control of any involved vein, including the IVC, should *not* be attempted since these thin-walled vessels are easily torn. Spongesticks applied proximally and distally to the IVC or iliac vein, insertion of balloon-tipped catheters proximally and distally through the fistula opening into the involved vein or direct finger pressure are the best maneuvers to obtain hemostasis of a bleeding AoVF.[6] 4) The fistula defect should be *oversewn from within the aneurysm cavity* after all clot is removed. A large needle and 3-0 Prolene suture is desirable. 5) Routine use of an *autotransfusing cell-saving device* is essential since replacements with 10-20 units of packed red blood cells are often necessary in these cases.[11]

A heightened awareness of AoVF's is essential to improve the high mortality rates associated with these complications. Routine auscultation of the abdomen for bruits, inspection of the abdomen and lower extremities for distended superficial veins and clinical evalution of high-output congestive failure should be done in all patients with either intact or ruptured AAA's, in spite of the fact that these measures often prove unrewarding in making the diagnosis of an AoVF associated with a ruptured AAA. An extremely *large, ruptured AAA* should heighten the clinician's suspicion of an AoVF. A urinalysis demonstrating *hematuria* in a *male* patient with a *ruptured* AAA, a preoperative CT scan documenting a retroaortic left renal vein, or an intraoperative finding of an absent left renal vein anterior to the aorta and especially a thrill in a retroaortic left renal vein can confirm the suspicion of an AoRF. With a very large ruptured AAA, the surgeon should routinely palpate the IVC for the presence of a thrill. Improved survival can potentially be achieved with these suggested preoperative and intraoperative maneuvers.

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Division of the Left Renal Vein

Ligation and division of the left renal vein (LRV) can facilitate exposure and control of the juxtarenal abdominal aorta.[1-10] Clark[11] first reported division of the LRV during surgery for a retroperitoneal tumor, and Simeone[12] and Erlik[13] found this maneuver helpful during shunt procedures for portal hypertension. The presence of multiple collaterals, including the adrenal, phrenic, gonadal, ascending lumbar and capsular veins, usually allows LRV division to be performed without significant left kidney dysfunction. However, the LRV must be transected near its junction with the inferior vena cava to preserve these collaterals.[2,3,13-15]

Complications of LRV division such as retroperitoneal bleeding, elevation in serum creatinine, renal venous congestion, hemorrhage, renal infarction, and rupture of the left kidney have been reported.[3,4,11,16-22] To avoid these complications during LRV division, we have recommended that a left renal vein stump pressure (LRVSP) be taken and if greater than 60 cm water LRV division should not be performed.[1] We noted marked venous distention above this pressure in our original series of 23 patients undergoing LRV division.[1] Arterial blood flow to an organ is known to be impaired when venous pressure approaches diastolic arterial pressure (i.e., $\geq$ 60 cm water).[23] A distended, tense LRV after test clamping has been proposed as a simple method to determine the safety of LRV division.[24] Others have recommended routine re-anastomosis of the transected LRV to prevent complications.[5] Often LRV reconstruction is not feasible due to retraction and subsequent tension on the vein. In order not to rely solely on a subjective impression of LRV swelling after test clamping, we attempted to confirm our previous clinical impression that a LRVSP greater than 60 cm water should serve as a contraindication to LRV division.[1] The purpose of this section is to 1) report experimental data correlating LRVSP with left kidney function in the canine model, 2) report our results of a large series of patients undergoing LRV division during abdominal aortic surgery, 3) suggest guidelines for LRV division and 4) recommend when re-establishment of LRV flow should be performed.

Materials and Methods

Experimental model: LRV collaterals in the dog are not present as in humans.[25] *Ligation* of the LRV near the hilum results in severe venous congestion of the kidney with LRVSP's rising to 135 cm water.[26,27] We developed an experimental model to determine the relationship of renal function and *incremental increases* in LRV pressure. Nine mongrel dogs were anesthetized with intravenous sodium pentobarbitol (25 mg/kg), intubated and ventilated with room air on a volume-cycled respirator. Anaesthesia was maintained with supplemental sodium pentobarbitol as needed. Ringer's lactacte was inflused at a rate of 5 cc'c/kg throughout the operation. Continuous central venous pressure monitoring was established by inserting an 18-gauge catheter into the right internal jugular vein. Both ureters were cannulated and the right kidney in each dog served as a control with the central venous pressure reflecting the right renal vein pressure. The LRV was mobilized and cannulated for pressure monitoring. A screwdown vascular clamp was applied to the LRV to produce gradual, incremental elevations in LRVSP. Thirty minutes following induction of anaesthesia baseline pressures of each renal vein were measured. Every 15 minutes thereafter the LRVSP was increased approximately 10 cm water using the screwdown clamp and the following measurements of each kidney were made: urine output, creatinine clearance (CL-cr), urinary sodium concentration ([U-na+]), and urinary/plasma osmolality ratios (U/PL-osm). Plasma sodium concentration ([PL-na+]) was also measured at 15 minute intervals. Animal care complied with the "Principles of Animal Laboratory Care" and the "Guide for the Care and Use of Laboratory Animals" (NIH Publication No. 80-23, revised 1985).

Clinical series: Between January 1967 and December 1989, 71 of 1,095 patients undergoing abdominal aortic surgery were considered for LRV division because of difficulty obtaining perirenal aortic exposure (Table 1). The LRV was divided in 64 of these patients: 10% (57/589) of patients with an abdominal aortic aneurysm (AAA) and 1% (7/506) of patients with aortoiliac occlusive disease.

Table 1: **LEFT RENAL VEIN DIVISION**

LRV DIVISION CONSIDERED 71 PATIENTS		
LRV DIVIDED 64 PATIENTS		**LRV NOT DIVIDED** 7 PATIENTS
LRVSP (Not Measured*) 20 PATIENTS	**LRVSP** 44 PATIENTS	**LRVSP** 7 PATIENTS
	43 PTS $\leq$ 60 CM WATER	50 CM WATER
	1 PT = 85 CM WATER	55 CM WATER
		66 CM WATER
		66 CM WATER
		66 CM WATER
		70 CM WATER
		80 CM WATER
		84 CM WATER

*LRVSP was not measured 1) in the early part of this series and 2) due to the urgent need to gain proximal aortic control in the case of a ruptured abdominal aortic aneurysm.

LRV = left renal vein

LRVSP = left renal vein stump pressure

PT(S) = patient(s)

Re-anastomosis of the LRV was performed in only one patient. Any swelling or increased tension of the LRV was noted after application of a vascular clamp across the vein near its confluence with the inferior vena cava. Stump pressures were obtained by using a water-filled manometer held at the level of the kidneys

after insertion of a 21-gauge needle into the LRV. The vein was transected and oversewn approximately 1 cm from its junction with the cava.

Stump pressures were not measured in 20 patients undergoing LRV division because of the emergent need to obtain proximal aortic control in the case of ruptured AAA's or during the early part of this series when pressures were not routinely measured. The LRV was divided in 44 other patients undergoing elective surgery when only moderate swelling of the vein was noted and LRVSP's were found to be ≤ 60 cm water except in one case. The exception was a patient who had only moderate swelling of the LRV after clamping, a LRVSP of 85 cm water, a normally functioning right kidney based on a preoperative intravenous pyelogram, and an extremely large AAA that began just distal to the renal arteries. In seven other cases, the LRV was not divided because of elevated LRVSP's ($\geq$ 50-60 cm water) and LRV distention.

All surviving patients that underwent LRV division had serial serum creatinines and at least one postoperative intravenous pyelogram, renal scan or renal arteriogram to demonstrate left renal function.

Results

Experimental model: Left kidney urine output, CL-cr and ability to conserve sodium gradually declined with incremental elevations in LRVSP. A marked drop in these parameters occurred when the stump pressure rose above 50-60 cm water. The average urine output in the nine dogs dropped to 0.17 ml/min (86% below baseline) and the [U-na+] approximated [PL-na+] when the LRVSP equalled 60 cm water. The same parameters reflecting right kidney function remained constant as the LRVSP increased. Additionally, on the right side the U/P-osm rose with increasing LRVSP's as the control kidney excreted progressively more concentrated urine, while the left U/P-osm steadily decreased until a precipitous drop was noted when the LRVSP was greater than 50 cm water. It is important to note that the left renal vein in the dog does not have collaterals, unlike humans.

Clinical series: Ten of the 64 patients who underwent LRV division died but none as a direct result of this maneuver. The deaths were due to a ruptured AAA (4 cases), ischemic sigmoid colitis (2 cases), myocardial infarction (1 case), massive pulmonary embolism (1 case), postoperative bleeding (1 case), and respiratory failure (1 case).

Although several of the surviving patients were noted to have a transient rise in serum creatinine postoperatively, these values returned to baseline by the time of discharge in all but two patients. One of these patients underwent temporary suprarenal aortic clamping and the other had a ruptured AAA. Only one of the surviving patients who underwent LRV division was found to have a non-functioning left kidney as diagnosed by intravenous pyelography. This was also the only patient who had re-anastomosis of the LRV (after having undergone a left renal artery bypass). Of note is that two patients underwent left renal artery bypass, and based on a LRVSP $\leq$ 50 cm water, did not undergo LRV reconstruction. Postoperative angiograms demonstrated functioning left kidneys in both patients.

Comments

The need to divide the LRV to gain adequate exposure of the juxtarenal aorta during aortic surgery can be as high as 37% but in most reports is less than 5%.[1,5,9,19,20,24] In the present series LRV division was necessary in 10% of patients undergoing AAA surgery. This figure may be higher than other series because difficult, juxtarenal aortic aneurysms are frequently transferred to our medical center and LRV division may be required more in these cases.

LRV division can frequently be avoided by dividing its collaterals. However, if LRV division is necessary despite sacrifice of these branches, then left renal dysfunction is much more likely to occur.

Complications occurring as a direct result of LRV division were extremely rare in our series. Besides using a LRVSP of 50-60 cm water as a contraindication to LRV division, higher complication rates in other series may be secondary to a larger percentage of patients subjected to temporary *renal arterial hypoperfusion* (due to suprarenal aortic clamping or hypotension from a ruptured aneurysm) rather than due to obstruction of venous outflow. In only two of the 64 patients undergoing LRV division was an aortic clamp temporarily applied *above* the level of the renal arteries. Rastad reported a statistically significant increase in serum creatinine in 31 patients with abdominal aortic surgery who had LRV division compared to 25 patients where LRV division was not necessary, and that six patients who had undergone LRV division had sustained increase in serum creatinine (one of whom had loss of left kidney function), two patients had total or partial left kidney infarction, and two patients required a left nephrectomy for bleeding.[19] However, apparently more patients who underwent LRV division required suprarenal or renal artery clamping than the control group, and three of the six patients with a sustained increase in creatinine had *ruptured* AAA's.

Harris noted a significantly higher postoperative serum creatinine in patients operated on for *elective* AAA's who underwent LRV division but a four times greater percentage of these patients required suprarenal clamping than patients without vein division.[20] He noted a similar finding in patients operated for *ruptured* AAA's but the percentage of patients requiring suprarenal clamping was not reported.

Stenstrom reported a higher mortality in patients after *ruptured* AAA's who required LRV division.[21] However, as in the previous series concerning ruptured aneurysms, this result may reflect a greater degree of prolonged, severe hypotension resulting in relatively diminished arterial inflow in patients who had LRV division compared to patients not requiring this maneuver.

In a review of literature in 1986, Anderson reported that 11% (10/89) of patients suffered significant postoperative renal morbidity secondary to LRV division but notably in three cases collateral veins were damaged at the time of surgery.[22] Although it is difficult to determine if left kidney dysfunction is due to venous outflow obstruction secondary to LRV division rather than diminished arterial inflow associated with suprarenal aortic clamping or ruptured aneurysms, we recommend that sacrifice of the LRV be very cautiously performed in the face of these latter two situations.

However, many authors have reported minimal left kidney morbidity associated with LRV division.[1-10] Neal was one of the first to report favorable results with this maneuver in 11 patients with AAA's.[4] No deaths or renal dysfunction were attributable to LRV division. James noted that only one of 10 patients who underwent LRV division during aortic surgery developed moderate renal insufficiency but collateral venous channels were damaged in this patient.[7] Babu has recently reported a series of 25 patients who underwent LRV division for juxtarenal aortic aneurysms without any patient developing renal failure or requiring nephrectomy.[8] Reports by Adar[9] and Dearing[10] included a total of 34 patients that underwent aortic surgery and LRV division without re-anastomosis and documented no difference in postoperative serum blood urea nitrogen and creatinine compared to a control group or compared to preoperative levels by multi-variate analysis, respectively. Patients in this series had a transient rise in serum creatinine 24-48 hours postoperatively but radiologic studies of the left kidney 5-14 days after surgery were normal except in the patient mentioned above.

We do not favor routine re-anastomosis of the LRV after division for several reasons: the extra time that is required in these frequently poor-risk patients, the transected ends of the LRV often retract and render reconstruction difficult, and a LRVSP less than 50-60 cm generally provides adequate assurance that significant left kidney dysfunction will not result. We believe that contraindications to LRV division include a LRVSP $\geq$ 60 cm water, a very distended and tense LRV after test clamping (regardless of the pressure) and unknown function of the right kidney.

In one patient, placement of a clamp across the LRV did not result in any increase in pressure and a posterior segment of a left renal vein collar was identified. Awareness of this venous anomaly can avoid inadvertent injury and massive venous hemorrhage.

If LRV division is deemed essential to gain juxtarenal aortic exposure, and if left kidney venous congestion then develops, we recommend 1) re-anastomosis of the LRV if this can be accomplished without undue tension or 2) suturing an interposition polytetraflouroethylene graft between the transected ends of the LRV.[24]

In conclusion, an experimental canine model has confirmed a LRVSP $\geq$ 50-60 cm water as a relative contraindication to LRV division and a clinical series of 64 patients has established the safety of LRV division during aortic surgery without re-anastomosis anastomosis of the LRV if the LRVSP is $\leq$ 50-60 cm water. Only one patient developed a non-functioning left kidney after this maneuver. A more liberal use of LRV division in cases of difficult exposure of the juxtarenal aorta is indicated provided the above guidelines are followed. Reconstruction of the LRV is rarely indicated.

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Portal Hypertension Associated with Abdominal Aortic Aneurysms

The combination of a large abdominal aortic aneurysm and severe portal hypertension with or without massive gastrointestinal bleeding presents a challenging surgical problem. This combination is not common because most alcoholic cirrhotics with portal hypertension succumb to their liver disease at an early age before abdominal aortic aneurysms develop. The older patient with cryptogenic cirrhosis and portal hypertension, however, can have an associated abdominal aortic aneurysm and these patients require careful evaluation and surgical planning. In the patient with a nonruptured abdominal aortic aneurysm, if there is esophageal bleeding, this should be controlled with sclerotherapy. If there is no bleeding or controlled bleeding, it is my opinion that a laparotomy should be carried out with the surgeon prepared to do a shunt, a shunt and resection of the abdominal aortic aneurysm, or resection of the aortic aneurysm alone. The choice depends on the condition of the retroperitoneum and the degree of portal hypertension. The shunts in these cases should be portacaval or mesocaval with immediate decompression of the entire portal system. I would not recommend a selective splenorenal shunt (Warren) if one expects to carry out a concomitant retroperitoneal resection and abdominal aortic aneurysm resection — the bleeding in this situation can be horrendous as exemplified by one of our cases. A 55-year-old white man presented with a history of three episodes of upper gastrointestinal bleeding secondary to esophageal varices due to alcoholic cirrhosis. He also had a large abdominal aortic aneurysm. We thought that he probably could not withstand portal decompression and resection of the abdominal aortic aneurysm at the same time. Our initial plan was to perform axillobifemoral bypass first, and ligate the aneurysm at the time of splenorenal or portacaval shunt. Arteriography, however, demonstrated the possibility of a mycotic aneurysm. Therefore, it was decided to attempt a portosystemic shunt and resection of the aneurysm at the same time. On October 17, 1980, the patient successfully underwent a Warren splenorenal shunt, after which the retroperitoneum over the aneurysm was opened and mobilization was attempted. Because of marked portosystemic collateral vessels in the retroperitoneum, torrential bleeding was encountered. We decided to terminate the procedure and deal with the aneurysm later. Therefore, both internal iliac arteries were ligated and the abdomen was closed.

The patient did well postoperatively, and on October 22 he was returned to the operating room and right axillobifemoral bypass and ligation of both external iliac arteries were performed. Even with both internal and external iliac arteries ligated, there was not complete thrombosis of the aneurysm and, on October 25, the patient underwent embolization of the aneurysm through a left transaxillary approach. Multiple coils and Gelfoam were embolized into the common iliac arteries, the distal aorta and the aneurysm. There was a great deal of turbulence within the aneurysm, making complete thrombosis difficult. Repeat arteriography on November 18, 1980, revealed markedly reduced flow through the aneurysm.

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Annual Oration for 1991

In Defense of the Surgical Patient

JOHN M. DALY, M.D.

Injury, malnutrition and malignant disease all lead to depression of host immune defenses. This immunosuppression in surgical patients can result in sepsis and increased mortality. In the cancer patient, immunosuppression leads to the further development of metastatic disease and a diminished duration of survival. Thus, the patient with malignancy undergoing major operation may suffer from a background of malnutrition due to the tumor and anti-neoplastic therapies. In addition, the cancer patient who undergoes a major surgical procedure with or without blood transfusions may develop further suppression of host defense mechanisms resulting in increased morbidity, the potential for increasing metastatic disease and increased mortality. The immunologic consequences of trauma effect both cell-mediated (T-cell) and B-cell functions as well as non-specific immune defenses such as neutrophil and macrophage functions. Classically, there is a decrease in the delayed hypersensivity response, B-cell number and lgG synthesis, neutrophil antimicrobial activity, natural killer cell cytotoxicity and T-cell responsiveness to allo antigen. In addition, there is a significant alteration in host macrophage function. For example, patients who undergo major operation on the first postoperative day have a significant decrease in peripheral T-cell blastogenesis to the mitogen phytohemagglutinin. In addition, when one co-cultures normal T-cells with small amounts of IL-2, there is an increase in mitogenesis. However, in the postoperative patient, there is no increase in the mitogen stimulation index after co-culturing peripheral lymphocytes with IL-2. The mechanism of this immunosuppression is unclear. Various mediators of post-injury immunosuppression have been described. These include wound factors, endotoxin/bacterial products, immunosuppressive peptides, prostaglandins, the neuroendocrine response and other therapeutic measures which are used.

It is well known, for example, that following major operation there is a substantial neuroendocrine response to the surgery. This results in markedly elevated urinary cortisol levels as well as increases in plasma catecholamines and glucagon levels. In an effort to study the relationship of cortisol secretion following injury to immunosuppression, we have used the drug, etomidate. This is a substitute imidazole which is an intravenous anesthetic. Etomidate inhibits 11-beta hydroxylase which inhibits the secretion of cortisol in humands and corticosterone in rodents. In animals undergoing major injury, postoperative serum corticosterone levels were significantly diminished at and below normal levels when injury was accompanied using Etomidate in contrast to injury alone when corticosterone levels were at

400 ng/ml at six hours and 24 hours following injury. In this same animal model, natural killer cell cytotoxicity was significantly decreased at 24 and 72 hours following injury. However, in the group receiving etomidate at the time of injury, natural killer cell cytotoxicity remained normal. In other models of injury, animals bearing Lewis Lung carcinoma, injury resulted in significant growth of the tumor. *In vitro* studies demonstrated a significant decrease in the lysis of Lewis Lung carcinoma cells by splenocytes. This *in vitro* data correlates with increased tumor growth *in vivo* in other animal-tumor models.

Another mediator of post-injury immunosuppression is prostaglandin E-2. PGE_2 is associated with diminished T-lymphocyte mitogenesis, and CTL development. In addition, decreased natural killer cell activity and cytokine production of IL-1 and IL-2 are also exhibited. Finally, there is an increase in suppressor T-cell development. In animal models of injury, perioperative secretion of PGE_2 by peritoneal macrophage occurs within two hours of injury increasing from levels of 15 ng/ml to 38 ng/ml. These increased levels persist for approximately 24 hours. The use of prostaglandin E_2 blocking agents such as indocin abrogates not only the increased secretion of PGE_2 but decreases the immunosuppression occuring following injury. Another mediator of postoperative immunosuppression is that of nitric oxide. Nitric oxide is produced through the arginine deiminase pathway whereby arginine goes directly to citrilline with the release of nitric oxide in mitrochrondria. Concommitant with decreases in mitogenesis in animal models following injury is an increase in splenocyte nitric oxide production. Other *in vitro* studies have demonstrated that PGE_2 stimulates nitric oxide production in splenic macrophages. In addition, *in vitro* studies have demonstrated decreased con A mitogenesis after addition of PGE_2 in coculture systems. Thus, the etiologies of postoperative immunosuppression are multi-factoral. Opertive injury, anesthesia, blood transfusion, the presence of malignancy and concommitant malnutrition all effect the immune system. In the latter instance, macrophage superoxide production is markedly diminished in animals given a protein-free diet for one week. In addition, candida albicans phagocytosis and killing were also significantly depressed in protein calorie malnourished animals. Interestingly, both PGE_2 production and serum cortisol levels were increased in malnourished animals after one week of protein-free diet. Macrophage nitric oxide production, however, was diminished.

The clinical applicability of these findings relates to the incidence of postoperative infectious complications. Windsor and Hill demonstrated that normal individuals after upper gastrointestinal surgery have a rate of atelectasis of approximately 50%. However, only 15% (3/20 patients) developed postoperative pneumonia. In malnourished patients undergoing the same operative procedures, again 50% of these individuals (16/39) developed atelectasis. However, eight of these 16 patients went on to develop pneumonia with an increase in the mean length of hospital stay from 13 to 17 days. The consequences of immunosuppression secondary to malnutrition are those of increased infectious complications.

Because of this, we have sought to identify specific nutrients which may improve immunologic function following injury. Arginine is a non-specific amino acid which is part of the urea cycle. In addition, arginine through arginase gives rise to ornithine and the polyamines spermine, spermadine and putrescine. Finally, it gives

rise to reactive nitrogen intermediates through the arginine deiminase pathway. Bolus administration of arginine results in increases in plasma levels of growth hormone, insulin, glucagon, prolactin and somatostatin. We have also noted in injured and septic animal models that hepatic fibrinogen synthesis is markedly and significantly increased with arginine administration *in vivo*. Other immunologic effects of arginine administration are demonstrated by increased thymic size and cellularity. In addition, lymphocyte mitogenic and allogeneic responses are increased as well as macrophage tumor cytotoxicity and natural killer cell cytotoxicity. Finally, there is an increase in IL-2 production and IL-2 receptor activity. These cellular effects result in immunologic effects demonstrated by increased rejection of allogeneic grafts and decreased tumor growth with increased host survival. In prior studies using C1300 and TBJ neuroblastoma models, supplemental arginine administration to a regular diet resulted in markedly diminished tumor growth and improvement in survival. Further studies demonstrated increases in lymphokine activated killer cell cytotoxicity in culture with increasing amounts of arginine in the culture. Finally, natural killer cell cytotoxicity was also enhanced in animal models in which tumor-bearing animals received either arginine or glycine plus interleukin-2. Animals which received arginine plus interleukin-2 had a 50% survival in comparison with a 25% survival in animals receiving glycine plus IL-2 or arginine alone. None of the animals who received glycine alone survived beyond 34 days.

It is critically important that these studies using cultured cells and animal models be able to be replicated in man. For this reason, we turned to patients with upper gastrointestinal malignancies affecting the esophagus, stomach and pancreas in order to demonstrate the potential benefit of nutrient supplementation. These patients undergo major operative procedures lasting four-six hours and at least one third of these patients suffer significant malnutrition when first presenting to the surgeon's office. Thus, they often have lost more than 10% of their body weight at the time when they are first seen. Because of the potential for significant morbidity and mortality, the use of postoperative enteral feeding has been initiated in order to improve outcome. The first prospective randomized trial compared patients receiving postoperative enteral jejunostomy feedings using a modular enteral diet with and without arginine. One group received L-arginine at 25 grams per day while the second group received L-glycine at 43 grams per day. The patients had a jejunostomy placed at operation and began to receive a full-strength formula at 25 cc/hour on the first postoperative day. The feedings continued for at least eight days; they were then allowed to eat by mouth depending upon their clinical situation. Mean daily nitrogen intake was nearly identical in the two groups of patients. Thirty patients were studied for the biologic effects of this nutrient supplementation. In both groups, peripheral T-lymphocyte activation was markedly diminished from the preoperative to the first postoperative day. However, only in the arginine group was there a rapid and significant rise to normal levels of T-lymphocyte activation by the fourth and seventh postoperative day, whereas those individuals who received glycine supplementation had a continued suppression of T-lymphocyte activation.

Other nutrients such as RNA and omega-3 fatty acids can also improve immunologic function. Dietary nucleotide restriction has been shown to decrease T-lymphocyte mitogenesis, decrease response to allogeneic antigen and result in

suppression of delayed type hypersentivity. The provision of omega-3 fatty acids to the diet has been shown to increase the production of eicosapentaenoic acid which results in increased levels of trianoic prostaglandins such as PGE_3, PGI_3, and TXA_3 rather than the dianoic prostaglandins which are so immunosuppresive. A randomized prospective clinical trial was then carried out to look at biologic endpoints such as serum protein levels, nitrogen balance, and lymphocyte mitogenesis. In addition, clinical endpoints such as tolerance, morbidity, mortality and hospital length of stay were assessed. Eighty-five patients with esophageal, gastric and pancreatic tumors were entered into this trial. Forty-one patients received the diet supplemented with arginine, RNA and omega-fatty acids while 44 patients received a standard enteral product. Weight loss $\geq$ 10% body weight occurred in approximately one-third of these patients and was equally distributed across groups. Mean ages were 60 and 65 years respectively. Operative procedures such as esophagogastrectomy, gastrectomy and pancreatectomy were equally distributed across groups. Lymphocyte proliferation following PHA stimulation decreased significantly in both groups following the operation. However, by the second postoperative day it had returned to normal levels in the supplemented group compared with the standard fed group. In addition, eicosopentioic acid levels increased dramatically by seven days in the plasma and in peripheral monocytes and further increased at weeks two, four and twelve. In addition, PGE_2 production was markedly diminished over this period of time. Of the 85 patients who entered the trial, four patients were unable to be fed and four were ineligible. Thus, there were 77 eligible patients were analyzed. Cardiopulmonary complications such as electrocardiographic changes, pulmonary embolus and atelectasis occurred in equally similar rates in both groups of patients. However, infectious and wound complications were significantly increased in the standard fed group (38%) versus 12% in the supplementally fed groups when analyzed for all 85 patients or for the 77 eligible patients. The 77 eligible patients had a mean length of stay of 20.2 days in comparison with 15.8 days in the group fed the supplemental diet ($p < .01$).

Similar findings were found in a study by Gottschlich et al. They fed patients who had suffered major body burns with a modular feeding formula containing increased amounts of arginine and omega-3 fatty acids. They compared this group of 17 patients with 14 patients who received Osmolite HN and 19 patients who received Traumacal. The number of wound infections, instances of pneumonia and total infections were significantly decreased in the modular tube feeding group compared with either of the other two groups. In addition, hospital length of stay per percent burn was significantly decreased in the modular tube feeding group. Finally, a recent multi-center trial in patients in the Intensive Care Unit evaluated an arginine supplemented diet compared with a standard formula and found that hospital length of stay was significantly decreased at 21 days compared with 27 days median length of stay in the standardly fed group ($p = .01$).

Thus, potential applications for specific nutrient feeding include those with malignancy, injury, and protein-calorie malnutrition. Current studies are evaluating the use of standard and supplemental feedings not only during the hospital stay but in the outpatient period as well. It is clear that immunologic deficits in patients are multi-factoral including advanced age, presence of cancer and malnutrition,

the occurrence of diabetes as well as injury and other metabolic abnormalities. The use of certain cytokines and monoclonal antibodies to cytokines markedly improve cellular function and result in less immunologic deficiency following major injury. The use of certain nutrients may also be beneficial in groups of patients who may suffer protein-calorie malnutrition following their injury.

The Jonathan Rhoads Annual Oration 1992

Multidisciplinary Approaches to the Future Treatment of Cancer

JAMES L. WEESE, M.D., F.A.C.S.

This presentation will review what I consider to be important issues in the care of patients with cancer, discuss some interesting changes brought about by the multidisciplinary management of cancer patients, and finally, consider possible solutions to what is perceived as the "healthcare problem." As we address the changes that are to come in the treatment of patients with cancer, it is important to point out that more patients with cancer have been cured by surgery alone than by any other modality.

Multiple Therapeutic Modalities

As we look toward the future, we must examine the interactions of multiple therapeutic modalities. In anticipation of this, it becomes critical to understand anatomic principles of surgery which are often ignored and unfortunately, I believe, result in compromised results for patients with cancer. A classic example is the role of hysterectomy in patients with rectal cancer. Everybody here understands the specifics of a Miles abdominal perineal resection, but I would like to review some studies published years ago by Block and Enquist.[1] Specifically, they took sky blue dye and injected it into the submucosa of the rectum at the dentate line and every two inches above the dentate line. They found that tumors of the rectum at the dentate line have lymphatic drainage through the posterior vagina. Those two inches above the anal verge drained through the posterior vagina, the cervix, the corpus of the uterus, the broad ligaments, the tubes, the ovaries and the cul de sac. Those lesions four inches above the anal verge had lymphatic drainage going through the broad ligaments and the cul de sac.

Another study published by Veazey and McBride[2] showed that in women with transmural tumors of the rectum, 20 percent who had their pelvic organs left *in situ* had local recurrences compared to none in those patients who either had a concomitant or prior hysterectomy. In patients with Dukes' C lesions, 48 percent of women without a prior or concomitant hysterectomy recurred after abdominal perineal resection, compared to 11 percent of those who had undergone hysterectomy. These data suggest that hysterectomy is an important adjunct in these women and may provide better local control of their disease.

Role of the Omentum

Another critical anatomic issue concerns the role of the omentum in intra-abdominal tumor spread and small bowel obstruction. As surgeons, we have all been faced with the problem of the "omental cake" where patients with cancers present with life-threatening, intractable bowel obstructions. I believe that many of these bowel obstructions are caused when tumor cells are picked up by the omentum and implant and grow into the small intestine ultimately resulting in a bowel obstruction. We investigated this problem by using a transplantable n-Methyl-N-Nitrososourea (NMU), induced rat colon cancer carried in Fisher 344 (F344) rats. Tumors were removed from the subcutaneous tissue and separated by sharp dissection and passage through stainless steel screens to generate viable single cell suspensions. These were reconstituted in RPMI 1640 with HEPES buffer at 10^8 cells per ml. One-tenth ml (10^7 cells) was injected into the peritoneal cavity of each of three groups of F344 rats. The first group underwent tumor injection alone. The second group underwent tumor injection after a sham laparotomy. The third group underwent tumor injection after laparotomy with omentectomy. Results are shown below:

Group	No. of Animals	Operation	Number with Omental Tumor (%)	Number with SBO (%)
1	43	None	19 (44%)	13 (30%)
2	47	Sham	35 (75%) *	40 (85%) *
3	43	Omentectomy	11 (26%†) *	7 (16%) *

*$p<0.001$ by Chi-Square Analysis
†Tumor in lesser omentum or omental remnant

Those animals undergoing sham laparotomy had a significantly increased rate of omental tumor and of small bowel obstruction. Omentectomy significantly reduced the rate of small bowel obstruction.[3]

Multidisciplinary Therapy

Although I define a Surgical Oncologist as a Surgeon who knows when not to operate on cancer, it is important that we not confuse our residents and students as they explore multidisciplinary approaches to cancer.

Recent acceptance of multidisciplinary therapy can perhaps best be seen in patients with breast cancer. When many of us were residents the treatment for breast cancer prescribed most often was a classic Halsted radical mastectomy. Over the last 15-20 years, the trend has been towards much lesser surgery. Studies have shown that a modified radical mastectomy gives the same survival and local control advantages of a classic Halsted. Most recently, lumpectomy with lymph node dissection and radiation therapy has been shown to offer comparable local recurrence and long-term survival rates when compared to modified radical mastectomy for many patients with breast cancer. When we teach our residents the concepts of multidisciplinary therapy, we must stress that lesser operations alone may not be the best treatment for all patients with cancer. If therapy is not designed

with a complete understanding of the disease process, we will see higher rates of recurrences in the future.

Neoadjuvant Treatment

I would like to present some of our studies using neoadjuvant treatment in patients with pancreatic cancer. Neoadjuvant therapy in general describes treatment with a combination of modalities prior to definitive local-regional therapy. Pancreatic cancer is a particularly difficult problem which we felt was amenable to neoadjuvant therapy. There are approximately 27,000 new cases per year, of which 24,500 patients die annually. The five-year survival rate in most series runs 0-3%. Gudjonsson in 1987 examined 22,319 cases published in the literature. He found 92 patients (0.41%) who survived five years.[4] Part of the problem is that less than one in four patients is a candidate for resection at the time of diagnosis. Once diagnosed, less than one in five individuals explored is a candidate for resection. Other studies suggest that lesions over two centimeters have a very high rate of metastatic disease in the lymph nodes. Statistics for palliative procedures are quite poor. The median survival of patients undergoing bypass is 4.3 months, compared to 16.5 months for those patients undergoing Whipple procedures. Tsuchiya of Japan reviewed 100 of his patients with cancers of two centimeters or less. He found that 99 were resectable. However, it was disheartening to find that even in these best risk patients, only 30% survived five years.[5]

Treatment of these patients is particularly difficult because pancreatic cancer spreads through lymphatic, hematogenous, and perineural pathways. However, direct extension is the most important problem in local control of the disease. The pancreas is rich in lymphatic channels, being surrounded by nodes and densely adherent lymphatic tissue surrounding the mesenteric vessels, the aorta, the vena cava and other critical structures that cannot easily be removed. In addition, pancreatic cancer stays localized to the region for long periods of time prior to dissemination. In fact, two thirds of patients with pancreatic cancer die from complications of local disease.

Pilepich and Miller in 1980 looked at 17 patients who were explored at Tufts University who were found to have unresectable or borderline resectable pancreatic cancers. After operative exploration these patients were given 4600 cGy over four-and-a-half to five weeks. Eleven patients came to reexploration at six weeks, of which six of the 11 were thought to be resectable for cure. Two of the six patients had tumor at their resection margin. Another two of the six survived for over five years.[6] The Gastro-Intestinal Tumor Study Group (GITSG) looked at a multi-institutional comparative trial of radiation therapy alone or in combination with 5-FU for unresectable pancreatic cancer patients. They found that patients treated with 6,000 rads alone did quite poorly with a median survival of just over 20 weeks. When 5-FU was used as a radiation sensitizer with either 4,000 or 6,000 rads, they found that the median survival could be nearly doubled with overall survival also being doubled.[7]

Because of the extremely low cure rate for either surgery or radiation alone, a high failure rate with both local and metastatic disease, the risk of dissemination of viable tumor cells during surgery, and limited margins even with very radical

operations, I, along with two colleagues, the late Dr. Anthony Paul (a medical oncologist) and Dr. Larry Solin (a radiation oncologist), developed a program looking at neoadjuvant chemoradiotherapy in pancreatic cancer. Patients were staged with CT scans of the head, chest, abdomen and pelvis as well as a bone scan. They were given 5-FU, one gram per square meter per day for four days as a continuous i.v. infusion, starting days two to five and 29 to 32. They were given Mitomycin-C 10 mg. per square meter i.v. on day two. They received 5,040 cGy of radiation as 180 cGy fractions daily, five days per week; 3960 cGy were given to a wide field of the tumor and head of the pancreas with an additional 1080 cGy given to a coned-down field. Patients with extrapancreatic disease were excluded. Three to six weeks after completion of radiation therapy, they were restaged. We found only about 15% of patients showed a decrease in the tumor size on CT scan. Those patients free of extrapancreatic disease underwent resection. Originally, total pancreatectomy was performed because of: 1) our concern of doing an anastomosis after radiation; 2) the concern about the multifocal nature of these cancers; and, 3) the published high rate of tumor at the resection line after Whipple procedures.

In my personal series, 38 patients have been subjected to this treatment: 35 with cancers of the head of the pancreas, two with cancers of the body of the pancreas and one with a large carcinoma of the duodenum with periaortic adenopathy. Five patients were not explored: two had portal vein thrombosis, one patient had liver metastases seen on CT scan, one patient refused operation, and one patient died from cholangitis prior to completion of chemoradiotherapy. Thirty-three of these thirty-eight individuals have come to laparotomy. Twenty-two of the 33, all of whom had large tumors to begin with, were able to undergo resection. The 11 who were not resected were felt to be technically resectable but had extrapancreatic disease at the time of laparotomy. Particularly upsetting was the fact that the majority of this group of patients had tumor seeding along drain sites and in the wound after prior exploration which excluded them from potentially curative resection.

Operatively, we found that tissue planes were well-defined and enhanced by post-irradiation edema. The bulk of radiated tissue was removed and anastomoses were done in non-irradiated tissue. The only organ that we found to be grossly damaged by radiation was the duodenum.

Histologically, twenty of the 22 patients had residual tumor. Twenty patients had completely negative margins. Knowing that these were large tumors, we were quite pleased that only four of 22 had microscopic disease in regional lymph nodes. Tumor cell necrosis and hyalinization was seen on resected specimens that had not been seen on the pre-therapy specimens. Nine patients of the 22 resected are still alive, free of disease ranging up to 82 months from the time of diagnosis. In sum, we feel that aggressive neoadjuvant chemoradiotherapy can be performed safely, allows successful resection and may yield long-term survival or cure.[8,9]

Immune Modulatory Techniques

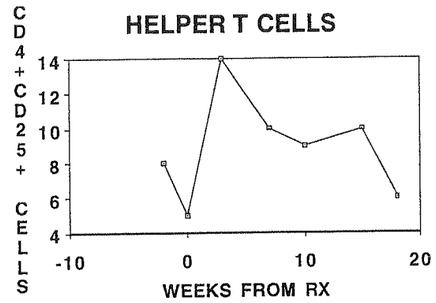
I would now like to turn my attention to the use of immune modulatory techniques, one of the other aspects of multidisciplinary therapy. I have often thought

this part of the oration should be subtitled, "Was Aunt Tillie Right After All?" How often have we as cancer surgeons been asked by a patient or their family about their concerns that when "air hits the tumor," will the cancer spread dramatically, resulting in rapid progression of the tumor and death? As an immunologist, I am concerned that acute interactions of therapy may alter the immune system to transiently favor the tumor over the host. To address this, a number of experiments were done to test the hypothesis that perioperative immunodepression is a critical factor in the implantation and growth of metastatic disease. Animal experiments in our laboratory using the previously described NMU-induced colon adenocarcinoma in F344 rats have shown that multiple operations increase the rate of tumor growth when compared to animals undergoing no operation or only one operation.[10] Additionally, numerous investigators have shown that operations and anesthesia produce transient depression of immune parameters of T-cell function.

In 1982, Windell and Bell demonstrated that levamisole given perioperatively can reduce operation-induced immunosupression in rats as measured by lymphocyte proliferation assays. Using the tumor model that we have previously described, tumor cells were injected into rat portal veins, which generated hepatic metastases in F344 rats. Laboratory studies demonstrated that the use of perioperative treatment with either levamisole or MVE-2 (Maleic-anhydride-divinyl ether-2), which are both non-specific biologic response modifiers, would decrease the incidence of liver metastases when compared to the metastatic rate in identical rats not receiving treatment with biologics.[11,12] When we tested this same hypothesis using Interleukin-2 (IL-2), we found that perioperative treatment with low-dose IL-2 significantly decreased the incidence of liver metastases; however, higher doses of IL-2 appeared to increase tumor dissemination. These studies suggested that perioperative treatment with biologics may reduce the incidence of tumor dissemination in this model.[13]

Knowing the potential changes that occur in the immune system, we recently developed a program to examine whether depressed levels of T-helper cells, specifically CD-4, CD-25 positive cells, may correlate with tumor progression. We asked whether *in vitro* stimulation of T-cells with tumor marker antigens can increase levels of T-helper cells and whether autologous *in vitro* cultured T-helper cells stimulated with tumor marker antigens would have an effect on tumor growth and on patient response when reinfused intravenously. After obtaining approval from our institutional review board and after patients signed informed consent, cells were removed from peripheral blood and placed on a ficoll-hypaque gradient. We performed *in vitro* stimulation with purified tumor marker protein. In the case of prostate cancer patients, we used PSA (Prostate Specific Antigen) to determine optimal dose. Cells were cultured with PSA to raise 10^7 helper cells. After identification by dual marker flow cytometry, these cells were infused into the patient. We used dose response curves to document in the given patient that a dose of five micrograms/ml was the optimal stimulatory dose. We subsequently studied changes in T-cell levels from the time of infusion and documented a dramatic increase of T-helper cell levels persisting for nearly four months.

We also noted a decrease in the slope of the curve of this patient's PSA increases. Although this is only one case, we were pleased that the gentleman sustained immediate bone pain relief after his T-helper cell infusion.



Global Issues

As we enter 1993 with calls for major revisions in health care, I think it would be inappropriate to deliver this address without discussion of more global issues. It is frightening to think that health care expenditures in 1990 reached approximately 11.5% of the gross national product and will reach 12.2% by 1995 at current growth rates. Although America has the best medical care in the world, the cost of providing care to indigent or uninsured people made up only 3.6% of hospital costs in 1980. This increased to 6.7% in 1990 and is projected to be 8.3% by 1995.

Recently isolated mortality statistics for individual heart surgeons were published out of context in the *Philadelphia Inquirer* several weeks ago. It is frightening to conceive that this biased presentation of data may force surgeons to turn down more technically difficult cases. This sort of limited reporting of data without concern for severity of cases may lead to reduced availability of quality care for those who need it most.

In times when rapidly increasing costs of medical care must be controlled, we as surgeons must ask what we can do. It is imperative that cost containment must *NOT* affect quality of care. I suggest that several important actions be used to help contain costs in the future.

1. Many young physicians are graduating from medical school and finishing their residencies saddled with large debts. The government should help arrange debt repayment of obligations by arranging for these physicians to take a certain percentage of Medicare, medical assistance and uninsured patients in their practice. With this commitment, their debt should be waived over a number of years.

2. Malpractice costs, claims and lawyer fees must be limited. Unfortunately, without caps on settlement awards and legal fees, the great majority of malpractice settlements are going to lawyers rather than the harmed parties. In days of cost containment, it is inappropriate for our legal colleagues to be making fees of millions of dollars. While physicians' fees are rapidly being reduced and controlled, how can an attorney justify such outrageous personal rewards for the amount of work they perform on a given case? Without malpractice costs being controlled, defensive medicine will continue to be practiced and expenses will continue to rise.

3. We physicians must be prepared to standardize therapy. Not every surgeon does the same operation for a given problem. In cancer therapy, many patients are subjected to chemotherapy which varies greatly from published drug regimens which have been shown to be most effective. Certainly future cancer treatment should be done by established protocol which will result in more standardized and presumably more effective and cost efficient therapy options.

4. The new administration must be prepared to commit funding to research and development of new therapy. American medicine has always set the standard for the modern world in innovative medical care. We cannot allow this competitive advantage to fall behind others in the future.

5. Resources must continue to be committed to discover the causes of cancer. This commitment will enhance our chance for curing cancer in the future.

It is truly an honor to have been able to deliver the Jonathan Rhoads Annual Oration. I have appreciated Dr. Rhoads' support from the time that I first moved to Philadelphia and became Chairman of Surgical Oncology at Fox Chase during the time he was a member of their Board of Directors. His commitment to surgical education and research has made a tremendous impact on many generations of surgeons. The principles he has taught and the surgeons he has trained will allow many of these goals to be accomplished. I greatly appreciate the opportunity to present this oration in his honor.

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Transactions of the Philadelphia Academy of Surgery

Regular Meeting

January 15, 1987

The meeting was called to order by President Francis Rosato, M.D.

Scientific Session

"Intraoperative Laser Arteroplasty For Ischemic Leg" was presented by Teruo Matsumoto, M.D., and discussed by Drs. Rudolph C. Camishion, Brooke Roberts and Charles C. Wolferth.

"Impedance Cardiology in the 1980's" was presented by Joseph M. VanDeWater, M.D. (by invitation). There was no discussion.

"Initial Results — Liver Transplant Program," presented by Bruce E. Jarrell, M.D., was discussed by Dr. Ira J. Fox.

Regular Meeting

February 2, 1987

Willis P. Maier, M.D., President, called the meeting to order.

Scientific Session

"Blunt Trauma" was presented by Charles Vinocur, M.D. (by invitation). Drs. Ward O. Griffen, Moreye Nusbaum, and David Wagner discussed.

"Fewer Hepatic Metastases (METS) by Perioperative Treatment with Recombitant Human Interleukin-2 (RIL-2)" was presented by James L. Weese, M.D. (by invitation). The discussers were Drs. Stephen Weiss and Henry Moss.

"Significance and Staging of Clinically Occult Breast Cancer" was presented by Anne L. Rosenberg, M.D. (by invitation). Drs. Robert Somers, Jonathan E. Rhoads, and Willis P. Maier discussed.

Conjoint Meeting with New York Surgical Society

March 11, 1987

The annual Joint Meeting with the Philadelphia Academy of Surgery was held at the University Club, New York City at 2:00 p.m.

Program

Warren Wetzel, M.D. (by invitation). "Urban Snakebites and the New York City Snakebite Center — First Four Years' Experience."

Discusser: Charles E. Hartford, M.D.

W. Moscovici, M.D. (by invitation), J. Marks, M.D. (by invitation), and C.K. McSherry, M.D. "An Experimental Model for the Combined Medical and Surgical Treatment of Gallstones."

Discusser: Charles C. Wolferth, Jr., M.D.

By invitation: A. Estabrook, M.D., S. Yemul, M.D., S. Kister, M.D., D. Seldin, M.D., M.J. Link, M.D., and P. Kramer, M.D. "Immunoimaging and Biodistribution Studies with ^{111}Indium Labeled Monoclonal Antibody to Breast Cancer in Nude Mouse Xenografts."

Discusser: Stephen M. Weiss, M.D.

Dana K. Anderson, M.D. and by invitation: F. Charles Brunicardi, M.D., Rochelle L. Chaiken, M.D., Neal E. Seymour, M.D., Harold E. Lebovitz, M.D., Ronald E. Chance, Ph.D., Ronald L. Gingerich, Ph.D., Dariush Elahi, Ph.D. "Reversal of Abnormal Glucose Production in Chronic Pancreatitis by Pancreatic Polypeptide (PP) Administration in Man."

Discusser: Wallace P. Ritchie, Jr., M.D.

Anthony J. Acinapura, M.D., Daniel M. Rose, M.D., Joseph N. Cunningham, Jr., M.D., Israel J. Jacobowitz, M.D., Marshall D. Kramer, M.D. (by invitation), and Ziv Zisbrod, M.D. (by invitation). "Internal Mammary Artery Bypass: Influence of Recurrent Angina and Survival in 2,100 Patients."

Discusser: Horace MacVaugh, III, M.D.

Jose Guillem, M.D. (by invitation), Michael R. Treat, M.D. (by invitation), and Kenneth A. Forde, M.D. "An Aggressive Approach to Rectal Bleeding Will Diagnose Colon Cancer at an Early Stage."

Discusser: Thomas L. Dent, M.D.

Regular Meeting

April 6, 1987

The meeting was called to order by President Willis P. Maier, M.D.

Scientific Session

"S(7) — The Hemodynamics of Post-Shunt Neurological Deterioration: Analysis and Clinical Trial," recipient of the Samuel D. Gross Prize, was presented by Kaj Johansen, M.D.. Ph.D.

Regular Meeting

May 4, 1987

The meeting was called to order by Willis P. Maier, M.D., President.

Scientific Session

"Radiomanometry: A Guide to Common Bile Duct Exploration," presented by Laszdo Fuzesi, M.D. (by invitation), was discussed by Drs. Edgardo S. Alday and George Gowen.

"Chronic Mesenteric Ischemia" was presented by Luis E. Sala, M.D. (by invitation) and discussed by Brooke Roberts, M.D. and Dominic A. DeLaurentis, M.D.

"Avulsion Injuries of the Pelvic Torso," presented by David S. McCloskey, M.D. (by invitation), was discussed by Dr. Steven Rossi.

Regular Meeting

October 5, 1987

Vice-President Dominic A. DeLaurentis, M.D., called the meeting to order in the absence of President Maier.

Scientific Session

"The Effect of Trauma Severity on Hospital Cost and Its Relationship to Reimbursement Under the Prospective Payment System," presented by C. William Schwab, M.D. (by invitation), was discussed by Dr. John M. Templeton.

"Salvage of Complications in the Irradiated Breast with Arterialized Flaps" was presented by Don LaRossa, M.D. Dr. Francis Rosato discussed.

"Surgical Treatment of Abdominal Aortic Aneurysms with the Rigid Intraluminal Prosthesis" was presented by Paschal M. Spagna, M.D. Drs. Gerald M. Lemole and Frederick A. Reichle discussed.

Regular Meeting

November 2, 1987

President Willis P. Maier, M.D., called the meeting to order.

Scientific Session

"Intraoperative Radiotherapy for Pancreatic Cancer — Radiation Therapy Oncology Group (RTOG) Experience" was presented by Stephen M. Weiss, M.D. and discussed by James Weiss, M.D.

"Acute Acalculous Cholecystitis as a Complication of Open-Heart Surgery," presented by Feroz Sheikh, M.D. (by invitation), was discussed by Francis Rosato, M.D.

"Surgical Correction of Facial Skeletal Growth Abnormalities" was presented by Harvey M. Rosen, M.D. Dr. Peter Randall discussed.

Regular Meeting

December 7, 1987

The meeting was called to order by Willis P. Maier, M.D., President.

Annual Oration

"Surgery of the Parotid Gland" was presented by Robert Harwick, M.D.

Annual Report of the Secretary

1987

There were eight formal meetings of the Philadelphia Academy of Surgery in 1987. Six were held in Thompson Auditorium and one in Mitchell Hall at the College of Physicians of Philadelphia. The average attendance at these scientific sessions was 125. The dinner meetings preceding these sessions were well attended with an average of 85 members.

The Conjoint Meeting with the New York Surgical Society was held in New York City on March 11th. The Scientific Session began at 2:00 p.m. There were 35 members of the Philadelphia Academy of Surgery and 92 members from the New York group present. Willis P. Maier, M.D. and Kenneth Ford, 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 New York Fellows were discussed by the members of the Philadelphia group. The New York Surgical Society hosted dinner at the University Club.

William P. Maier, M.D., appointed a Nominating Committee consisting of Chairman H. Taylor Caswell, M.D., Donald R. Cooper, M.D. and Francis Rosato, M.D. This committee submitted the following slate of officers and council members for 1988:

President — Dominic A. DeLaurentis, M.D.
1st Vice President — Clyde Barker, M.D.
2nd Vice President — Rudolph C. Camishion, M.D.
Secretary — David K. Wagner, M.D.
Treasurer — Robert Harwick, 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.
— Thomas Dent, M.D.

Chairman of the Samuel D. Gross Prize Fund — John M. Daly, M.D., Ward O. Griffen, M.D., Simon Simonian, M.D.

Robert Harwick, M.D., of Temple University presented the Jonathan E. Rhoads Annual Oration on December 7, 1987 entitled, "Surgery of the Parotid."

Regular Meeting

January 4, 1988

President Willis P. Maier, M.D., called the meeting to order.

Scientific Session

"Internal Carotid Stump Pulse, Internal Carotid Free-Back Flow. Internal Carotid Back Pressure/Systemic Pressure Index: Correlates of Internal Carotid Back Pressure," was presented by Carol A. Raviola, M.D. (by invitation). Dr. Richard K. Spence discussed.

"Stress is Good," presented by Max M. Cohen, M.D. (by invitation), was discussed by Dr. Wallace P. Ritchie, Jr.

"Surgery for Vertebral Artery Stenosis" was presented by Andrew B. Roberts, M.D. (by invitation). Dr. Richard K. Spence discussed.

Regular Meeting

February 1, 1988

President Dominic A. DeLaurentis, M.D., called the meeting to order.

Scientific Session

"Peri-Graft Seroma," Marsha Guowicz, M.D. (by invitation) was discussed by Henry Berkowitz, M.D.

"Section of the Arcuate Fasciculus in the Dominant Hemisphere of a Severe Stutterer" was presented by Robert K. Jones, M.D. and discussed by Michael J. O'Connor, M.D.

"Prevention and Management of Ureteral Injuries During Reoperative Aoaorto-iliac Surgery" was presented by John W. Francfort, M.D. (by invitation). Dr. Henry D. Berkowitz discussed.

Conjoint Meeting

March 9, 1988

The meeting was called to order at the College of Physicians in Philadelphia at 2:00 p.m.

Program

Michael J. O'Connor, M.D. "Advances in Temporal Lobe Epilepsy Surgery."
Discusser: Joseph Ransohoff, M.D. (by invitation).

John S. Kukora, M.D. "Not All Perforated Peptic Ulcers Are Alike: Prepyloric (PU) Versus Duodenal (DU) Versus Gastric (GU)."
Discusser: Michael M. Eisenberg, M.D.

John M. Daly, M.D. "Immune Stimulation Using Arginine: Correlation of In-Vivo and In-Vitro Effects."
Discusser: Stanley M. Levenson, M.D.

Gerald Marks, M.D. "Sphincter Preservation for Cancer of the Distal Rectum Utilizing Full-Dose Preoperative Radiation Therapy."
Discusser: Kenneth Eng, M.D.

Ronald P. Savarese, M.D., Dominic A. DeLaurentis, M.D. "Venous Anomalies Encountered in Abdominal Aortic Surgery."
Discusser: Michael Silane, M.D.

Herbert E. Cohn, M.D. "The Value of Non-Invasive Localization Studies in Primary Hyperparathyroidism."
Discusser: Ashok Rakaram, M.D.

Regular Meeting
April 4, 1988

President Dominic A. DeLaurentis, M.D., called the meeting to order.

Scientific Session

"Patterns of Recurrent Adenomas Discovered Endoscopically Predict the Clinical Course of Colorectal Neoplasia" was presented by George F. Gowen, M.D. Drs. John M. Daley and Ward O. Griffen discussed.

"Enders Nail Fixation in Long Bone Fracture — Experience in a Level I Trauma Center," presented by Khaleel Shaikh, M.D. (by invitation), was discussed by Dr. John R. Clarke.

"Opthalmic Artery Aneurysms" was presented by Eugene S. Flamm, M.D. (by invitation) and discussed by Dr. Michael J. O'Connor.

Regular Meeting
May 2, 1988

The meeting was called to order by Dr. Dominic A. DeLaurentis, President.

Scientific Session

"Congenital Microgastria: The Role of Aggressive Surgical Management" was presented by Alfonsa L. Velasco, M.D. (by invitation). Dr. Moritz M. Zeigler discussed.

"Laser-Aided Vascular Reconstructive Surgery: 20-Month Follow-up," presented by Teruo Matsumoto, M.D., Ph.D., was discussed by Drs. Rudoph Camishion, Brooke Roberts, and Joseph VanDeWater.

"Protrusio Acetabuli — Its Occurrence as a Constant in Marfan's Syndrome and a Surgical Approach to the Problem by Closure of the Triradiate Epiphysis" was presented by Howard H. Steel, M.D. Dr. James E. Nixon discussed.

Regular Meeting
October 3, 1988

The meeting was called to order by President Dominic A. DeLaurentis, M.D.

Scientific Session

"Use of Non-Absorbable Radioactive Material in Treatment of Carcinomas by Local Injections" was presented by Karim B. Nakhgevany, M.D. Drs. Howard A. Zaren and Henry Moss discussed.

"Surgical Management of Severe Liver Trauma: A Role for Liver Transplantation," presented by John Angstadt, M.D. (by invitation), was discussed by Drs. Ira J. Fox and Charles C. Wolferth, Jr.

"A Rational Approach to Understanding Missile Wounds" was presented by Frank Ehrlich, M.D. (by invitation) and discussed by Dr. Charles C. Wolferth.

Regular Meeting
November 7, 1988

President Dominic A. DeLaurentis, M.D., called the meeting to order.

Scientific Session

"Reconstruction of Facial Defects After Moh's Chemosurgery," presented by Kristin Steuber, M.D. (by invitation), was discussed by Robert D. Harwick, M.D.

"Subclavian Axillary Artery Thrombosis Following Radiation for Breast Carcinoma" was presented by Farouq A. Samhouri, M.D. (by invitation). Dr. Brooke Roberts discussed.

"Pre-operative Determination of the Requirement for Tricuspid Valve Replacement Versus Repair: Analysis of 412 Patients with Tricuspid Valve Disease" was presented by Lynn B. McGrath, M.D. (by invitation). Dr. Dryden Morse discussed.

Regular Meeting
December 5, 1988

Dr. Dominic A. DeLaurentis, President, called the meeting to order.

Annual Oration

"Between Scylla and Charybdis in Surgery Today" was presented by Rudolph C. Camishion, M.D.

Annual Report of the Secretary
1988

There were eight formal meetings of the Philadelphia Academy of Surgery in 1988. Seven were held in Thompson Auditorium and one in Mitchell Hall at the College of Physicians of Philadelphia. The average attendance at these scientific sessions was 110. The dinner meetings preceding these sessions were well attended with an average of 75 members.

The Conjoint Meeting with the New York Surgical Society was held in Philadelphia on March 9th. The Scientific Session began at 2:00 p.m. There were 43 members of the Philadelphia Academy of Surgery and 48 members from the New York group present. Dominic A. DeLaurentis, M.D., President of the Philadelphia Academy of Surgery, presided over the individual sessions. Two papers presented by the New York Fellows were discussed by the members of the Philadelphia group. Four papers were presented by the Philadelphia Academy. The Philadelphia Academy of Surgery hosted dinner at the Union League of Philadelphia.

The Nominating Committee consisting of Frederick B. Wagner, Jr., M.D., William Maier, M.D., and Chairman, Francis E. Rosato, M.D., submitted the following slate of officers and council members for 1989:

President — Clyde F. Barker, M.D.
1st Vice President — Rudolph C. Camishion, M.D.

2nd Vice President — David K. Wagner, M.D.
Secretary — Steven M. Weiss, M.D.
Treasurer — Robert Harwick, M.D.
Recorder — Wallace P. Ritchie, Jr., M.D., Ph.D.
Chairman, Committee on Scientific Business — Moreye Nusbaum, M.D.
Council-at-Large — Dominic A. DeLaurentis, M.D.
— Thomas Dent, M.D.
— Gerald J. Marks, M.D.
Samuel D. Gross Prize Fund — Ward O. Griffen, M.D.
— John M. Daly, M.D.
— Paschal Spagna, M.D.

Rudolph C. Camishion, M.D., of Cooper Hospital/University Medical Center, presented the Jonathan E. Rhoads, M.D., Annual Oration on December 5, 1988 entitled, "Between Scylla and Charybdis in Surgery Today."

Regular Meeting

January 9, 1989

The meeting was called to order by President Dominic A. DeLaurentis, M.D.

Scientific Session

"Perspectives in the Treatment of Carcinoma of the Esophagus: Eighteen Years of Clinical Experience" was presented by Manucher Fallahnejad, M.D. Dr. Dominic A. DeLaurentis discussed.

"Augmentation Cystoplasty," presented by David M. Raezer, M.D. (by invitation), was discussed by Dr. John J. Murphy.

"Management of Pancreatic Abscess — An Approach to Transverse Mesocolon" was presented by Donna Barbot, M.D. (by invitation). Dr. Ernest F. Rosato discussed.

Regular Meeting

February 6, 1989

President Clyde F. Barker, M.D., called the meeting to order.

Scientific Session

"Symptomatic Unilateral Vertebral Artery Stenosis: The Diagnosis and Follow-up Assisted by 123-I Iodoamphetamine Spect Imaging" was presented by Anthony J. Comerota, M.D. Dr. Andrew B. Roberts discussed.

"Hypotension and Bleeding with Various Anatomic Patterns of Adult Blunt Splenic Injury," presented by Robert F. Buckman, Jr., M.D. (by invitation), was discussed by Charles C. Wolferth, Jr., M.D.

"Carcinoma of the Lung with Chest Wall Involvement," presented by Ronald Savarese, M.D., was discussed by Dr. William Clark Hargrove, III, M.D.

Conjoint Meeting with New York Surgical Society

March 8, 1989

The annual Joint Meeting with the Philadelphia Academy of Surgery was held at the University Club, New York City at 2:00 p.m.

Program

A. Epstein, M.D. and W. Wetzel, M.D. "A New Technique for Managing Severe Liver Injury."
Discusser: John P. Clarke, M.D.

Jamal J. Hoballah, M.D., Edward H. Kim, B.A. and Allan E. Dumont, M.D. "Thrombocytopenic Purpura in Parenteral Drug Abusers: Response to Splenectomy."
Discusser: Michael S. Weingarten, M.D.

A. J. Capizzi, M.D., S. Blau, M.D., K. C. Cho, M.D., H. M. Delany, M.D. and M. L. Gliedman, M.D. "Impact of Early Radiologic Studies on the Surgical Management of Suspected Acute Sigmoid Diverticulitis."
Discusser: Bernard Sigel, M.D.

Jon R. Cohen, M.D., Noel Tenenbaum, M.D. and Leslie Wise, M.D. "Greenfield Filter (GF) As Primary Therapy for Deep Venous Thrombosis (DVT) and/or Pulmonary Embolism (PE) in Cancer Patients."
Discusser: Charles C. Wolferth, Jr., M.D.

Rene A. Khafif, M.D., Gary A. Gelbfish, M.D. Patrick Tepper, M.D. and Joseph N. Attie, M.D. "Elective Radical Neck Dissection in Epidermoid Carcinoma of the Head and Neck, a Retrospective Analysis of 853 Cases of Mouth, Pharynx and Larynx Cancer."
Discusser: Robert D. Harwick, M.D.

Hon S. Lee, M.D., Henry Lamaute, M.D., Walter F. Pizzi, M.D. and Donald Picard, M.D. "Acute Gastro-Duodenal Perforations Associated with Crack Use."
Discusser: John S. Kukora, M.D.

D. L. Abramson, M.D. J. P. Gertler, M.D., T. Lewis, M.D. and J. G. Kral, M.D. "Crack Related Perforated Gastro-Pyloric Ulcers — an Urban Epidemic."
Discusser: John S. Kukora, M.D.

Regular Meeting

April 3, 1989

The meeting was called to order by President Clyde F. Barker, M.D.

Scientific Session

"Gallbladder Rupture from Blunt Abdominal Injury" was presented by Joseph Whitlark, M.D. (by invitation). Dr. John R. Clarke discussed.

Traumatic Transections of the Thoracic Aorta: Report of 29 Cases" was presented by Anthony Del Rossi, M.D. Dr. Paschal M. Spagna discussed.

"Correlation of Preoperative Arteriographic Findings and Follow-up Results with Argon and YAG Laser Endarectomy," presented by Teruo Matsumoto, M.D., Ph.D., was discussed by Drs. Anthony J. Comerota and Rudolph C. Camishion.

Regular Meeting
May 1, 1989

Dr. Clyde F. Barker, President, called the meeting to order.

Scientific Session

"Relationship of Injury Severity Score to Hospital Resource Utilization" was presented by Keith F. O'Malley, M.D. (by invitation). Dr. C. William Schwab discussed.

"Update on Open Mitral Commissurotomy" was presented by Francis P. Sutter, D.O. (by invitation), and discussed by Dr. Dryden P. Morse.

By invitation, "Urology Research in the Soviet Union" was presented by Professor N. N. Lopatkin, and discussed by Deputy Director Anatoly Darenkov of the Urologic Research Institute of Moscow.

Regular Meeting
October 2, 1989

The meeting was called to order by President Clyde F. Barker, M.D.

Scientific Session

Dr. Michael DeBakey, newly elected Honorary Fellow, presented "Some Personal Observations on the Surgery of Atherosclerosis."

Regular Meeting
November 6, 1989

Dr. Clyde F. Barker, President, called the meeting to order.

Scientific Session

"The Benefits of High Dose Preoperative Radiation for the Unfavorable and Low-Lying Rectal Cancer: Impact upon Local Recurrence, Survival, and Extended Use of Sphincter Preservation Surgery" was presented by Gerald Marks, M.D. Dr. Harvey Lerner discussed.

"Airway Management in Severe Facial Injury," presented by Steven E. Ross, M.D. (by invitation), was discussed by Dr. David K. Wagner.

"Primary Malignant Peritoneal Mesothelioma: A Report of Seven Cases and a Review of the Literature" was presented by Juan A. Asensio, M.D. (by invitation). Dr. Paul Addonizio discussed.

Regular Meeting
December 4, 1989

President Clyde F. Barker, M.D., called the meeting to order.

Annual Oration

"Reflections on Reflux" was presented by Wallace P. Ritchie, Jr., M.D., Ph.D.

Annual Report of the Secretary
1989

There were eight formal meetings of the Philadelphia Academy of Surgery in 1989. Six were held in Thompson Auditorium and one in Mitchell Hall at the College of Physicians of Philadelphia. The average attendance at these scientific sessions was 130. The dinner meetings preceding these sessions were well attended with an average of 80 members.

The Conjoint Meeting with the New York Surgical Society was held in New York City on March 8th. The Scientific Session began at 2:00 p.m. There were 40 members of the Philadelphia Academy of Surgery and 102 members from the New York group present. Clyde F. Barker, M.D. and Melvin H. Worth, M.D., presidents of the Philadelphia Academy of Surgery and New York Surgical Society respectively, presided over the individual sessions. Seven papers presented by the New York Fellows were discussed by the members of the Philadelphia group. The New York Surgical Society hosted dinner at the University Club.

President Clyde F. Barker, M.D., submitted the following slate of officers and council members for 1990:

President — Rudolph C. Camishion, M.D.
1st Vice President — David K. Wagner, M.D.
2nd Vice President — Moreye Nusbaum, M.D.
Secretary — Steven M. Weiss, M.D.
Treasurer — Robert Harwick, M.D.
Recorder — Wallace P. Ritchie, Jr., M.D., Ph.D.
Chairman, Committee on Scientific Business — Anthony J. Comerota, M.D.
Council-at-Large — Clyde F. Barker, M.D.
— Paschal Spagna, M.D.
— Thomas Dent, M.D.

Wallace P. Ritchie, Jr., M.D., Ph.D., of Temple University Hospital presented the Jonathan E. Rhoads, M.D. Annual Oration on December 4, 1989 entitled, "Reflections on Reflux."

Regular Meeting
January 8, 1990

The meeting was called to order by President Clyde F. Barker, M.D.

Scientific Session

"Surgical Treatment of Pancreatic Abscesses" was presented by Francisco Badosa, M.D. (by invitation). Dr. Ernest F. Rosato discussed.

"Twelve Years' Experience with Intraluminal Sutureless Prosthesis Repair of

Thoracic Aortic Dissections," presented by Mehmet Oz, M.D. (by invitation), was discussed by Dr. Paschal M. Spagna.

"Delayed Gastric Emptying Post Roux-Y Anastomosis" was presented by George F. Gowen, M.D., and discussed by Dr. Moreye Nusbaum.

Regular Meeting

February 5, 1990

The meeting was called to order by Rudolph C. Camishion, M.D., President.

Scientific Session

"Fetal Cardiac Mechanics" was presented by Jeffrey Dunn, M.D. Dr. Charles Vinocur discussed.

"The timing of Surgical Intervention in the Treatment of Facial Bone Fractures" was presented by Lenora R. Barot, M.D. (by invitation), and discussed by Dr. Harvey Rosen.

"Popliteal Venous Aneurysm Causing Pulmonary Emboli: Current Diagnosis and Treatment," presented by Anthony J. Comerota, M.D., was discussed by Dr. Richard K. Spence.

Conjoint Meeting with New York Surgical Society

March 14, 1990

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

Program

Henry D. Berkowitz, M.D., Karl Engleman, M.D., Marlynne Micalizzi, R.N. "Pheochromocytoma: Diagnosis, Localization and Treatment."
Discusser: Demetrius Persemilidis, M.D.

J. P. Hoffman, M.D., J. L. Weese, M.D., B. L. Eisenberg, M.D., R. L. Krigel, M.D., K. A. Padvia-Shaller, M.D. "Continuous Intrasplenic Arterial Recombinant Interleukin-2 (RIL-2) Therapy in Patients with Hepatic Metastases."
Discusser: John B. Hodgson, M.D.

Robert D. Smink, Jr., M.D., Albert Yellin, M.D., Gilbert S. Campbell, M.D., Ph.D., Henry Buchwald, M.D., Ph.D. "Partial Ileal Bypass: Five Year Lipid Results of the Program on the Surgical Control of Hyperlipidemias (POSCH)."
Discusser: Leslie Wise, M.D.

J. M. Daly, M.D., M. A. Shinkwin, M.D., FRSCI, R. Lenkinski, Ph.D., M. Zlatkin, M.D., T. Frank, M.D., G. Holland, M.D., H. Kressel, M.D. "Integrated Magnetic Resonance Imaging and 31P-Spectroscopy of SOF Tissue Tumors."
Discusser: Norman Bloom, M.D.

R. K. Spence, M.D., J. Costabile, M.D., G. S. Young, M.D., E. D. Norcross, M.D., J. B. Alexander, M.D., M. J. Pello, M.D., U. M. Atabek, M.D., R. C. Camishion, M.D.

"Is Hemoglobin Level Alone a Reliable Predictor of Outcome in the Severely Anemic Patient?"
Discusser: James Barone, M.D.

Keith D. Calligaro, M.D., Ronald Savarese, M.D., Peter R. McCombs, M.D., Dominic DeLaurentis, M.D. "Left Renal Vein Division — Clinical and Laboratory Implications."
Discusser: William Stahl, M.D.

Regular Meeting

April 2, 1990

President Rudolph Camishion, M.D., called the meeting to order.

Scientific Session

"Arteriovenous Fistula in Distal Prosthetic Arterial Reconstructions" was presented by James B. Alexander, M.D. Dr. Anthony J. Comerota discussed.

"HLA Crossmatch, Age and Gender as Variables Determining the Outcome for Cardiac Transplantation," presented by James B. McClurken, M.D., was discussed by Dr. Bruce Jarrell.

"Preoperative Chemotherapy for Soft Tissue Sarcomas of the Extremities" was presented by Christopher M. Pezzi, M.D., and discussed by Dr. John Daly.

Regular Meeting

May 7, 1990

Dr. Rudolph Camishion, President, called the meeting to order.

Scientific Session

"Pancreatitis Following Resection of Aortic Aneurysm," presented by Peter R. McCombs, M.D., was discussed by Leonard J. Perloff, M.D.

"Recent Insights on Permanent Closure of Massive Wounds" was presented by Linwood R. Haith, M.D. Dr. John J. Gostigian discussed.

"Gastric Carcinoma — New Advances in Treatment" was presented by Burton L. Eisenberg, M.D. Dr. Stephen M. Weiss discussed.

Regular Meeting

October 1, 1990

The meeting was called to order by President Rudolph C. Camishion, M.D.

Scientific Session

"Laparoscopic Cholecystectomy at the Bryn Mawr Hospital: The First 100 Cases" was presented by John Kairys, M.D. Dr. Gordon Busby discussed.

"Surgery of the Alimentary Tract in Heart Transplant Patients," presented by Daniel T. Dempsey, M.D., was discussed by Moreye Nusbaum, M.D.

"Venous Injury: A Dilemma in Management" was presented by Morris Kerstein, M.D., and discussed by Peter McCombs, M.D.

Regular Meeting

November 5, 1990

President Rudolph C. Camishion, M.D., called the meeting to order.

Scientific Session

"Heart Transplantation in High-Risk Patients Over 55 Years" was presented by Verdi J. DiSesa, M.D. Dr. Jeffrey Alpern discussed.

"Advances in the Surgery of Ulcerative Colitis and Polyposis Coli," presented by Moreye Nusbaum, M.D., was discussed by George F. Gowen, M.D.

"S. Epidermidis Synthetic Vascular Graft Infection: The Critical Inoculum Threshold and Bacterial Morphogenesis" was presented by John V. White, M.D. Dr. Richard Spence discussed.

Regular Meeting

December 3, 1990

The meeting was called to order by President Rudolph C. Camishion, M.D.

Annual Oration

"A Venous View of Abdominal Aortic Aneurysm Surgery" was presented by Dominic DeLaurentis, M.D.

Annual Report of the Secretary

1990

There were eight formal meetings of the Philadelphia Academy of Surgery in 1990. Seven of these meetings were held in Thompson Auditorium and a Conjoint meeting with the New York Surgical Society was held in Mitchell Hall at the College of Physicians in Philadelphia. The average attendance at the regular meetings was 95. The dinner meetings preceding the regular meeting were well attended with an average of 75 members.

The Conjoint Meeting began at 2:00 p.m., at the Philadelphia Academy of Surgery on March 14. There were 65 members of the Philadelphia Academy of Surgery and 55 members of the New York group present. Six papers were presented by the Philadelphia Academy and discussed by the New York group.

Rudolph C. Camishion, M.D., President, appointed a Nominating Committee and the Committee submitted the following slate of officers and council members for 1990:

President — David K. Wagner, M.D.
1st Vice President — Moreye Nusbaum, M.D.
2nd Vice President — Robert Harwick, M.D.

Secretary — Steven M. Weiss, M.D.
Treasurer — Thomas Dent, M.D.
Recorder — Wallace P. Ritchie, Jr., M.D., Ph.D.
Chairman, Committee on Scientific Business — Anthony J. Comerota, M.D.
Council-at-Large — Paschal Spagna, M.D.
— John M. Daly, M.D.
— Rudolph C. Camishion, M.D.

Dominic A. DeLaurentis, M.D., of Pennsylvania Hospital presented the Jonathan E. Rhoads, M.D. Annual Oration on December 3, 1990 entitled, "A Venous View of Abdominal Aortic Aneurysm Surgery."

Regular Meeting

January 7, 1991

The meeting was called to order by President Rudolph C. Camishion, M.D.

Scientific Session

"The Treatment of Ischemic Ulcers with Iloprost: A Stable Prostacyclin Analogue," authored by Kent S. Haas, M.D., Barrie Ashby, M.D., Robert W. Colman, M.D., Yanina T. Wachtfogel, M.D., and Anthony J. Comerota, M.D., was discussed by Henry Berkowitz, M.D.

"Retrospective Review of Mitral Valve Repair," authored by Francis P. Sutter, D.O. and Scott M. Goldman, M.D., was discussed by Glenn Whitman, M.D.

"Blood Transfusion and Oxygen Uptake from the Microcirculation," authored by Ronald F. Sing, D.O., Paul Marino, M.D., and Michael Sawyer, M.D., was presented. Dr. Thomas Santora discussed.

Regular Meeting

February 4, 1991

President David K. Wagner, M.D., called the meeting to order.

Scientific Session

Dr. Morris Kerstein discussed "Anastomotic Infections Treated by Selective Graft Preservation," authored by Keith D. Calligaro, M.D., Carl H. Westcott, M.D., Ronald P. Savarese, M.D., Carol A. Raviola, M.D., and Dominic DeLaurentis, M.D.

"Early Metabolic Response in Isolated Severe Brain Injury," authored by Anthony J. Mure, M.D., Michael E. Wegener, M.D., Keith F. O'Malley, M.D., E. Douglas Norcross, M.D., David W. Unkle, BSN, and Steven E. Ross, M.D., was presented. Dr. John Daly discussed.

"Intraoperative Ultrasound Use During Coronary Artery Bypass Surgery," authored by Paschal M. Spagna, M.D., Toshihiko Kurohiji, M.D., Fernando Gomez-Rivas, M.D., Bernard Sigel, M.D., Junji Machi, M.D., Howard A. Zaren, M.D., Richard E. Parsons, M.D., and Issei Kodama, M.D., was presented. Dr. Glenn Whitman discussed.

Conjoint Meeting

March 13, 1991

The Annual Joint Meeting with the New York Surgical Society was called to order at the University Club in New York at 2:00 p.m.

Program

Jorge Bustamante, M.D., Juan Ros-Carretero, M.D. Akella Chendrasekhar, M.D., Jose Marti, M.D. "Ringer's Lactate Versus Hydroxyethyl Starch in the Resuscitation of Hypovolemic Elderly Patients."

Rahinder P. Gandhi, M.D., Arthur Cooper, M.D., Peter Altman, M.D. "Experience with 120 Endoscleroses for Treatment of Bleeding Esophageal Varices in Children."

B. W. Pace, M.D., J. M. Cosgrove, M.D., B. Breur, Ph.D., l. B. Margolis, M.D. "Intraoperative Cholangiography Revisited."

Polly Gazetas, M.D., Alison Estabrook, M.D. "Importance of Adequate Staging and of Hormone Receptors in Women Over Age 70 with Breast Cancer."

L. Dashow, M.D., I. K. Friedman, M.D., R. Kempner, M.D., J. Rudick, M.D., C. K. McSherry, M.D. "Laparoscopic Cholecystectomy: The Initial Experience at Beth Israel Medical Center-Doctors Hospital."

Thomas M. Scalea, M.D., Fernando Garzia, M.D. "High Frequency Ventilation in the Treatment of Right Heart Failure."

Regular Meeting

April 1, 1991

The meeting was called to order by President David K. Wagner, M.D.

Scientific Session

"Cardiac Transplantation: Testing the Limits." Jeffrey Alpern, M.D., James B. McClurken, M.D., Susan Brozena, M.D., Paul Addonizio, M.D. Dr. Verdi DiSesa discussed.

"Radiolabeled Monoclonal Antibody Detection of Primary and Recurrent Large Bowel Cancer." J. M. Daly, M.D., A. Keenan, M.D., F. Weintraub, R.N., D. Haller, M.D., L. Cannon, R.N. The discusser was James Weese, M.D.

"Gastric Outlet Obstruction Following Gastroplasty: Diagnosis and Natural History." U. L. Seinige, M.D., R. Sing, D.O., D. M. Sataloff, M.D., C. P. Lieber, M.D. Dr. Francis Rosato was the discussant.

Regular Meeting

May 6, 1991

The meeting was called to order by President David K. Wagner, M.D.

Scientific Session

"Erythropoietin Treatment Accelerates Hematocrit Recovery in Severe Anemia Following Blood Loss." Richard K. Spence, M.D., Ronald Alvarez, B.S., Mark J. Pello,

M.D., James B. Alexander, M.D., Rudolph C. Camishion, M.D. The discussant was John V. White, M.D.

"A Prospective Comparison of Trauma Severity Indices." R. C. Talucci, M.D., J. Carson, M.D., K. F. O'Malley, M.D., S. E. Ross, M.D. John Clarke, M.D., discussed.

Regular Meeting

October 7, 1991

President David K. Wagner, M.D., called the meeting to order.

Scientific Session

"The Effect of Dynamic Cardiomyoplasty on Left Ventricular Systolic and Diastolic Functions in a Canine Model of Chronic Heart Failure" was authored by James D. Sink, M.D. John D. Mannion, M.D., was the discusser.

"Microsurgical Reconstruction of Advanced Recurrent Head and Neck Carcinomas for Palliation: Is It Justified?" Christopher Tzarnas, M.D., Michael Guiffrida, M.D. Robert D. Harwick, M.D., discussed.

"Surgical Treatment of Anorectal Fistula in Patients with Crohn's Disease — Revisited." David H. Levien, M.D., James Surrell, M.D., Patrick Mazier, M.D. The discusser was Thomas L. Dent, M.D.

Regular Meeting

November 4, 1991

President David K. Wagner, M.D., called the meeting to order.

Scientific Session

"Combined Cardiac Surgery and Carotid Endarterectomy During Aortic Cross-Clamping," authored by Steven J. Weiss, M.D., Francis P. Sutter, M.D., Thomas O. Shannon, M.D., Scott M. Goldman, M.D., was discussed by Andrew Roberts, M.D.

"Management of Pelvic Arteriovenous Malformations" was authored by Keith D. Calligaro, M.D., Patricia Carneval, M.D., Carl J. Westcott, M.D., Thomas V. Sedlacek, M.D., Carolyn E. Parry, M.D., Ronald P. Savarese, M.D., Dominic DeLaurentis, M.D. Dr. R. Anthony Carabasi discussed.

"Predictive Value of Flow Cytometric Analysis," authored by Dahlia M. Sataloff, M.D., was discussed by Burton Eisenberg, M.D.

Regular Meeting

December 4, 1991

The meeting was called to order by President David K. Wagner, M.D.

Annual Oration

John M. Daly, M.D., "Host Defenses in the Surgical Patient: Mechanisms for Intervention.

Annual Report of the Secretary

1991

There were eight formal meetings of the Philadelphia Academy of Surgery in 1991. Seven were held in Thompson Auditorium at the College of Physicians in Philadelphia. The average attendance at these scientific sessions was 90. The dinner meetings preceding these sessions were well attended with an average of 60 members.

The Conjoint Meeting with the New York Surgical Society was held in New York at the University Club. The Scientific Session began at 2:00 p.m. and six papers were presented by the New York group and discussed by the Philadelphia Academy. Dinner followed at the University Club in New York.

President, David K. Wagner, M.D., submitted the following slate of officers and council members for 1992:

President — Moreye Nusbaum, M.D.
1st Vice President — Robert Harwick, M.D.
2nd Vice President — Wallace P. Ritchie, Jr., M.D.
Secretary — Steven M. Weiss, M.D.
Treasurer — Thomas Dent, M.D.
Recorder — Paschal Spagna, M.D.
Chairman, Committee on Scientific Business — Moreye Nusbaum, M.D.
Council-at-Large — Anthony J. DelRossi, M.D.

John M. Daly, M.D., of the University of Pennsylvania, presented the Jonathan E. Rhoads, M.D. Annual Oration on December 2, 1991 entitled, "Host Defenses of the Surgical Patient: Mechanisms for Intervention."

Regular Meeting

January 6, 1992

The meeting was called to order by President David K. Wagner, M.D.

Scientific Session

"An Improved Technique for Needle Localized Biopsy of Occult Breast Lesions: Clinical Results of 98 Procedures," authored by Mary Lou Patton, M.D., was discussed by James Weese, M.D.

"Safe Laparoscopic Cholecystectomy Without Intraoperative Cholangiography," authored by J. B. Morris, M.D., R. Margolis, CRPN, E. F. Rosato, M.D., was discussed by Daniel T. Dempsey, M.D.

C. William Schwab, M.D., discussed "Facial Trauma in Motor Vehicle Accident," authored by Karim B. Nakhgevany, M.D., Mark Libassi, M.D., Barbara Esposito, R.N.

Regular Meeting

February 3, 1992

The meeting was called to order by President Moreye Nusbaum, M.D.

Scientific Session

"Evidence That Leukotrienes Mediate Bile Acid Injury to the Gastric Mucosa" was presented by Daniel T. Dempsey, M.D., and discussed by Dr. George F. Gowen.

"Surgical Approaches in Disequilibrium," presented by Kenneth F. Casey, M.D., was discussed by Dr. Frederick A. Simeone.

"Nosocomial Pneumonia in Trauma Patients: A Prospective Study" was presented by Collin E. M. Brathwaite, M.D. Dr. Charles Wolferth discussed.

Conjoint Meeting

March 18, 1992

The meeting was called to order at the College of Physicians in Philadelphia at 2:00 p.m.

Program

W. N. Bothwell, M.D., R. Bleicher, M.D., T. L. Dent, M.D. "Prophylactic Ureteral Catheterization in Colon Surgery: A 5-Year Review."

G. J. R. Whitman, M.D., M. A. Schinco, M.D., T. A. Santora, M.D., D. S. Weiman, M.D., P. M. Spagna, M.D. "Superiority of Pressure Support Ventilation with Continuous Positive Airway Pressure: PSV CPAP over CPAP Alone for Weaning Trials in Post-operative Coronary Artery Bypass Patients."

Steven P. Dunn, M.D. "Is Age Less Than One Year a High Risk Category for Orthotopic Liver Transplantation?"

Gordon P. Buzby, M.D., James L. Mullen, M.D., William O. Williford, M.D. "Department of Veterans Affairs Cooperative Trial of Perioperative TPN."

Robert F. Buckman, M.D., Michael M. Badellino, M.D., Juan Asension, M.D., Julieta D. Grosh, M.D., Jennifer Gass, M.D. "Observations in 66 Patients with Penetrating Cardiac Wounds: A Two-Year Prospective Study."

F. E. Rosato, M.D., Steven E. Copit, M.D., Ernest L. Rosato, M.D., Anthony P. Furnary, M.D., Donna J. Barbot, M.D. "Hepatic Resection for Metastatic Colorectal Cancer."

Regular Meeting

April 6, 1992

President Moreye Nusbaum, M.D., called the meeting to order.

Scientific Session

"Early Experiences with Russell Taylor Interlocking Nail System," presented by William B. DeLong, M.D., was discussed by Douglas Wright, M.D.

"Surgical Management of Splenic Abscess" was presented by W. H. Ayers, M.D. Dr. David L. Paskin discussed.

"Microsurgical Free Tissue Transfer for Salvage of Diabetic Lower Extremity Ulcers" was presented by Dennis T. Monteriro, M.D., and discussed by Amitabha Mitra, M.D.

Regular Meeting
May 4, 1992

Dr. Moreye Nusbaum, President, called the meeting to order.

Scientific Session

John V. White, M.D., presented the Samuel D. Gross Award Lecture, entitled "Adventitial Elastolysis and Abdominal Aortic Aneurysm Formation."

Regular Meeting
October 5, 1992

The meeting was called to order by Dr. Moreye Nusbaum, President.

Scientific Session

"A New Device and New Method for Cardiopulmonary Resuscitation: Minimally Invasive Direct Cardiac Compression." Robert F. Buckman, Jr., M.D.

"Success of Lytic Therapy in Occluded Reversed Vein Bypass Grafts." Henry D. Berkowitz, M.D.

"Warm Continuous Retrograde Cardioplegia in 100 Patients." Francis P. Sutter, D.O., Scott M. Goldman, M.D.

Regular Meeting
November 5, 1992

The meeting was called to order by President Moreye Nusbaum, M.D.

Scientific Session

"Our Approach to Differentiated Thyroid Carcinoma," presented by Francisco Badosa, M.D., was discussed by Herbert Cohn, M.D.

"Comparison of Laparoscopic and Open Appendectomy" was presented by Umur Atabek, M.D. Dr. David Rose, M.D., discussed.

"Management of Bile Duct Strictures: An Evolving Strategy" was presented by Joel L. Roslyn, M.D. Dr. Ernest Rosato, M.D., discussed.

Regular Meeting
December 7, 1992

Dr. Moreye Nusbaum, President, called the meeting to order.

Annual Oration

"Multidisciplinary Approaches to the Future Treatment of Cancer." James M. Weese, M.D.

Annual Report of the Secretary
1992

There were eight formal meetings of the Philadelphia Academy of Surgery in 1992, seven of which were held in Thompson Auditorium at the College of Physicians of Philadelphia. The average attendance at these scientific sessions was 83. The dinner meetings preceding these sessions were well attended with an average of 55 members.

The Conjoint Meeting with the New York Surgical Society was held in Philadelphia at the College of Physicians for the scientific meeting and dinner at the Union League. Thirty members of the New York Surgical Society attended.

Dr. Moreye Nusbaum appointed a Nomination Committee consisting of Chairman Moreye Nusbaum, M.D., Rudolph Camishion, M.D. and Clyde Barker, M.D. The following slate of officers and council members were presented for 1993:

President — Dr. Robert Harwick
1st Vice President — Dr. Wallace P. Ritchie, Jr.
2nd Vice President — Dr. Stephen M. Weiss
Secretary — Dr. John Daly
Treasurer — Dr. Thomas Dent
Scientific Business — Dr. Anthony Comerota
Recorder — Dr. Anthony DelRossi
Council-at-Large — Dr. Jonathan Rhoads, Jr.
— Dr. Ernest Rosato
— Dr. Moreye Nusbaum

Dr. James Weese of Graduate Hospital presented the Jonathan E. Rhoads Annual Oration of the Academy on December 7, 1992. His talk was entitled, "Multidisciplinary Approaches to the Future Treatment of Cancer."

Memoirs

THOMAS H. AINSWORTH, JR., M.D.
1920-1989

Dr. Thomas Ainsworth was born June 16, 1920, in Schenectady, New York, and raised in Upper Darby, Pennsylvania. He received a Bachelor of Science Degree from Pennsylvania State University in June 1941, and received his medical degree from Temple University Medical School in June 1944. Following graduation, he served a Rotating Internship at The Bryn Mawr Hospital from October 1944 to June 1945 and as a Resident in Surgery from July 1945 to March 1946, at which time he entered the military service, from which he was discharged in January 1948 with the rank of Captain. He completed his residency training at the Bellevue Hospital, II Surgical Division at New York University in June 1951. He was appointed to the staff of The Bryn Mawr Hospital on July 1, 1951, and was certified by the American Board of Surgery in 1952.

For the next 20 years, Dr. Ainsworth was engaged in the private practice of Surgery at The Bryn Mawr Hospital and achieved the rank of Attending Surgeon in 1963. He was appointed Clinical Associate Professor of Surgery and Anatomy at Temple University Medical School in 1967 and continued with this appointment until 1970.

The Bryn Mawr Hospital, and particularly the Department of Surgery, is indebted to Dr. Ainsworth for his role in helping us develop one of the first Intensive Care Units in the country in 1958. He was the first chairman of the unit at Bryn Mawr. During his years at The Bryn Mawr Hospital, he continued his interest in developing special units at the Hospital. He was instrumental in the planning and designing of the Home Care Program, the Patient Care Committee and our first and present Utilization Review Program, which later became the Quality Assurance Program (QAP). While at Bryn Mawr, he served as chairman of the Council on Extended Care for the Hospital Association of Pennsylvania for three years, and served on many committees, advisory panels, and the Council on Professional Services of the American Hospital Association, thanking us for making it possible for Dr. Ainsworth to participate as a member of the Committee on Nursing Services, the subcommittee on Patient Care and the Workshop on Organization of Hospital Inpatient Services, and for the valuable contribution he made to these committees.

During his tenure on the staff of The Bryn Mawr Hospital, he served as Secretary and as Vice-President of the Medical Staff and later as Chairman of the Hospital Planning Committee. Dr. Ainsworth was ahead of his time in many of his ideas in the field of hospital and medical organization and administration; and, fortunately, was certainly ahead of most of our clinical staff.

His interest in administrative medicine led him to resign from the medical staff of The Bryn Mawr Hospital and, in September of 1970, at the age of 50, he joined the American Hospital Association as Associate Director. In this position, he organized

the Committee on Physicians and served as the first Chairman of this Board of Trustees Committee. He also established the *Hospital Medical Staff Journal* and served as its first editor. He represented the American Hospital Association in the formation of the Coordinating Council on Medical Education and the Liaison Committee on Graduate Medical Education, and served as staff representative of the American Hospital Association to those two councils. In addition, he maintained a constant liaison with most national medical organizations, including the American Medical Association, the American College of Physicians, the American College of Surgeons, and the Joint Commission on Accreditation of Hospitals. He served as a consultant on the Steering Committee for the Experimental Medical Care Review Organization (EMCRO) projects for the National Center for Health Service Research and Development for a two-year period. He also maintained an active liaison with governmental agencies within the Department of Health, Education and Welfare and served on the American Medical Association's Task Force on Rules and Regulations for Professional Standards Review Organization (PSRO). He was the American Hospital Association's representative to the Administrative Board of the Council of Teaching Hospitals of the Association of American Medical Colleges.

In 1974, he resigned as Associate Director of the American Hospital Association and became Medical Director and Director of Medical Education Programs at Illinois Masonic Medical Center in Chicago. He continued as a consultant at the American Hospital Association and was the author of the Quality Assurance Program (QAP) for Medical Care that is currently used by most hospitals in the United States.

In 1977, he decided to enter the consulting field on a fulltime basis and was eventually responsible for the development of some 13 Experimental Quality Assurance projects funded by the Kellogg Foundation and numerous others scattered throughout the United States. During this period, he also founded a corporation for the promotion of "Wellness" known as Hale Inc., based in Philadelphia and later in California. He had finally become convinced that the prevention of disease and improved fitness were more rewarding than the treatment of diseases. Unfortunately, in 1979, Dr. Ainsworth, who was an addicted smoker for 40 years, developed a chronic cough, later diagnosed as Carcinoma of the Lung, which required a pneumonectomy in February 1980. After much soul-searching and numerous consultations, he eventually was admitted to a Canadian protocol for the immunotherapy of lung cancer, using the Hollingshead antigen in April, May and June of 1980. Despite being impaired by pulmonary insufficiency, Dr. Ainsworth continued his interest in the company he founded for the promotion of "wellness" and "fitness." During the next three years, he wrote a provocative and somewhat controversial book entitled *Love or Die,* which was published by MacMillan Publishing Company in 1983. The book offers an insider's view of the history, politics and socioeconomics of the medical establishment. In the book, he attempts to show where one serious problem lies and why rapidly escalating costs, restricted access to adequate family care, overspecialization and technological overkill could bring the world's best health care system to its knees. In fact, the book covers most of his experiences and activities of the last 20 years of his life in administrative medicine, and the insights he gained from them.

Dr. Ainsworth was the author of numerous papers and speeches, primarily

on subjects dealing with Quality Assurance Programs, Hospital Medical Staff Relationships, Health Care Services in Hospitals, Financing Health Services, the Rising Cost of Medical and Hospital Care, Health Maintenance Organizations and National Health Insurance. He was a member of numerous professional societies including the American Medical Association, the Philadelphia Academy of Surgery, The American College of Surgeons, the College of Physicians of Philadelphia and the American Hospital Association.

Dr. Ainsworth died March 25, 1989, at his home in Carmel Valley, California, after a long illness. He is survived by his wife, Sandy, a son, Thomas III, of Charlotte, North Carolina, a daughter, Ann McFarland, of Los Gatos, California, a sister, Margaret Conway, of Newtown Square, Pennsylvania, and a granddaughter.

—WILLIAM C. STAINBECK, M.D.

SAMUEL LUKENS CRESSON, M.D.
1916-1986

GEORGE PLATT PILLING, IV, M.D.
1918-1986

Samuel Lukens Cresson and George Platt Pilling, IV, both long-time members of this Academy, died within months of each other last year. They began their practices together in July of 1951 as the first pediatric surgeons at St. Christopher's Hospital for Children. Larry Somers and I were their first pediatric surgical residents: Larry in 1962, and I in 1963, initiating their newly approved pediatric surgery residency. Our own friendship started with Sam and George, we miss them, and we would like to give their memoirs together.

Sam Cresson was born on August 6th, 1916 in Philadelphia. He grew up in Swarthmore where he went to high school and Swarthmore College. He received his medical degree from Jefferson Medical College in 1943, during World War II, and interned at Pennsylvania Hospital. He then joined the Navy, took part in landings at Normandy and southern France, and after discharge from the Navy in 1946, took a three-year surgical residency at Pennsylvania Hospital. He followed this with a 2½-year residency in pediatric surgery under Robert E. Gross at the Boston Children's Hospital, and returned to Philadelphia in July, 1951, as Chief of Surgery at St. Christopher's Hospital for Children, a position he held for nearly 30 years. In 1953, he was asked to join the Lankenau staff as a pediatric surgeon, and he was Professor of Pediatric Surgery at Temple University School of Medicine.

Sam married Betty Keay in 1943, and they had three children, Betsy, Sandy, and Louisa, all of whom live in the Philadelphia area. Betsy, a nurse, is married to Jim Nutt, an orthopedic surgeon in Norristown, and they have three boys. Sandy teaches at Agnes-Irwin, and Lousa, a paralegal, is currently in law school. Sam loved Betty and was proud of his family.

Sam had many friends, and he had myriad interests, many of which involved nature and the outdoors. He always looked forward to "Doctors' Golf." He was a member of Trout Unlimited and Ducks Unlimited, and you knew when he was going to the monthly meetings of these organizations by the ties he would wear, small fish or small ducks, to identify the organization. On days when he was going to the Union League, he would wear the button in his lapel. He was a member of various clubs and organizations, and his favorite hat was studded with the pins and feathers, emblematic of these organizations. He loved to make trout flies for himself and for his many friends. He was a bird-watcher. He collected hundreds of birdskins which he stuffed with cotton and kept for use in identifying birds. He would not kill birds; he collected birds that died from natural causes. I remember how excited he was one morning when his daughter, Louisa, called him about an owl she saw lying on the roadside; she had persuaded her busdriver to stop so that she could retrieve it for her father to stuff. He also carved birds out of wood and painted them.

Sam was an active man. Vacations with Betty were busy—he wanted to get all he could out of everything, visiting museums, historic sites, never slowing down.

He walked fast, and unless an elevator opened right away, he would take the stairs. On surgical rounds, we had to walk fast to keep up with him, and he was always smiling, talking to people. He was a sociable person with many friends.

He did not like controversy, but if there were differences, he did not hesitate to tell you what he thought. He said what was on his mind without ambiguity, with a smile, and an open, straightforward way, an attitude he attributed to his Quaker upbringing. He always cautioned about putting strong differences in writing; however, because, after all, your aim was peace.

George Pilling, IV, was born in 1918 in Montreal, Canada. He attended high school at the Hill School in Pennsylvania, moved on to college at Yale University, and graduated from Cornell Medical School in 1943. After an internship at Pennsylvania Hospital, he served as a captain of the medical corps in the U.S. Air Force. Returning for surgical residency at Lankenau and Boston Children's Hospital, he joined Sam Cresson in the practice of pediatric surgery at St. Christopher's Hospital in 1951, where he served as Associate Chief of Pediatric Surgery until the time of his retirement from surgery. He was an Associate Professor of Pediatric Surgery at Tmple University Medical School, a member of the College of Physicians of Philadelphia, the American Pediatric Surgical Association, and the British Pediatric Surgical Association. He was a member of the Board of the Philadelphia Chapter of the American Cancer Society.

George married Barbara Bosworth, and had five children, one of whom was named George P. Pilling, V. He was an avid outdoorsman, a former Director of the Anglers Club, a member of the Sierra Club, the Wilderness Club, Ducks and Trout Unlimited, and the Philadelphia Conservationists. He was a former chairman of the Philadelphia Cricket Club's pheasant hunting program.

The legendary interactions between these two men as surgeons were colorful, and they are remembered by medical and surgical colleagues, house staff, and hospital personnel for these interactions. We would be remiss if we did not give you an example. I'll play Sam, and Dave will play George on a typical morning's surgical rounds.

Dialogue

GPP: "This two-day old baby was admitted last night with free abdominal gas."
SLC: "Chip, why didn't you operate?"
GPP: "Sam, you don't read much, do you? Didn't you see that case report in *Pediatrics* last month about spontaneous closure of a perforation without surgery?"
SLC: "You're being unrealistic, Chip." (Turning to the residents) "Sometimes I don't believe him."
GPP: "I certainly don't intend to operate."
SLC: "Are you trying to give me another heart attack?"
GPP: "Sam, now don't be absurd. Read the literature for God's sake."
SLC: "You don't believe I had a heart attack, do you?"
GPP: "Sam, we all know you had a heart attack."
SLC: (Reaching for his wallet) "Do you want to see my EKG?"
GPP: "Sam, I've seen that EKG 100 times, most recently yesterday morning on rounds."
SLC: "Chip, I'm going to show you my EKG." (Passing the EKG tracing around to the residents)
GPP: "This is absurd."
SLC: "This baby is my patient, Chip; it was referred to me last night."
GPP: "Sam, you haven't taken night call in 10 years."
SLC: "It was referred to me. The family wants me to operate."
GPP: "Sam, I came in last night. The baby's on my service. You haven't even met the family."
SLC: "Chip, you don't believe that I had a heart attack, do you?"
GPP: (Leaving rounds) "That's absurd."

Their partnership, in actuality, was one of mutual understanding and comaraderie. After our monthly partnership dinner meetings at the Chestnut Hill Cricket Club, we would go next door to St. Martin's in the Field Church where George was a member, and together have a moment of silence and contemplation.

I remember Sam as a superb surgeon, who rarely made a mistake, and as an energetic, life-loving man. My wife remembers him as a caring doctor, when he put his arm around her shoulder after our son's herniorrhaply, and said, "Mother, your boy's doing just fine." His patients and friends loved him.

And I remember George as the residents' friend, a sensitive, caring man who generally cared about the loves in his life, nature and outdoors, family, friends, and work. Both men will be remembered by colleagues for their legacy in pediatric surgery at St. Christopher's Hospital. We remember them with affection, and we miss them.

—DAVID K. WAGNER, M.D. and
LAURENCE A. SOMERS, M.D.

DONALD C. GEIST, M.D.
1901-1988

Donald C. Geist was born March 3rd, 1901 in Hazelton, Pennsylvania. He received his primary and secondary education in Hazelton and graduated from the University of Pennsylvania with a Bachelor or Arts degree in 1923. In 1926 he received an M.D. from the University of Pennsylvania School of Medicine. He served an internship at Misericordia Hospital in Philadelphia from July, 1926 to June, 1927, and then served as a rsident and Chief Resident at Misericordia Hospital from 1927 to 1931.

He was appointed an Assistant in Surgery at Misericordia Hospital in 1930, and became Chief of a Surgical Service in 1940. He served as an Instructor in Surgery at the Graduate School of Medicine of the University of Pennsylvania from 1935 to 1942. He was inducted as a Fellow of the American College of Surgeons in 1935 and was certified by the American Board of Surgery in 1940. He entered on active duty in the U.S. Navy in 1942 and served as a surgeon in the Pacific on the hospital ship USS Florence Nightingale until 1946 when he completed his tour of duty with the rank of Commander.

He was a member of the faculty of the Medical College of Pennsylvania and was an Assistant Professor of Clinical Surgery at Jefferson Medical College. He was a member of the Philadelphia County Medical Society, the Pennsylvania State Medical Society, and the American Medical Assocation, and a Fellow of the Philadelphia Academy of Surgery and the College of Physicians of Philadelphia. He served on the Board of Directors of Philadelphia County Medical Society, was a member of the Committee on Trauma of the American College of Surgeons, and served as a consultant surgeon to Jeanes Hospital and to Fitzgerald Mercy Hospital. A member of the Association of Military Surgeons and the American Medical Writers Association, he published many papers in the surgical literature and served as Editor of *Philadelphia Medicine* from 1966 to 1975, when he retired.

In the words of William Weiss, M.D., the current Editor of *Philadelphia Medicine,* "He was a gentle, kind, and sympathetic human being not only to his patients but to his colleagues and friends." Donald Geist continued his interest in surgery even after his retirement from active practice, and regularly attended meetings of this Academy and regional surgical meetings and conferences until his death August 2nd, 1988 at the age of 87.

—JOHN J. McKEOWN, JR., M.D.

ELMER C. GRIMES, M.D.
1914-1990

Dr. Elmer Grimes was born in suburban Boston on August 25, 1914. He was born of Irish immigrant parents. Elmer was educated in public schools in Winthrop and then at Harvard College. He graduated from Harvard in 1936 and then entered Tufts Medical School in Boston, from which he graduated in 1940, having worked his way through both college and medical school. The basic medical education at that time was designed to prepare the well-rounded general practitioner. After graduation, Elmer met Dr. Tom Kline, then medical chief at Philadelphia General Hospital (PGH), who advised him to consider a rotating internship at PGH. Elmer took his advice. At that time there were 500 applicants for 33 openings and the pay was $50 for two years, with $35 for uniform allowance and $15 for the dues for the Medical Society key. We were then supposed to be entitled to free medical care for the rest of our lives.

Elmer rotated through medical services, pediatrics and general surgical services, including one with Dr. L. K. Ferguson. When he completed his internship he was able to get a residency in Pathology, and spent one year as a pathologist at PGH where he did approximately 700 autopsies. He was then called into the Navy and served in the South Pacific. He developed an osteochondroma on his fourth finger and was to return to the States for treatment. While waiting at Pearl Harbor, the war ended. Elmer was sent back to the States for discharge.

At that time no opening was available in surgery at PGH, so he went into private practice with a friend in Illinois. He subsequently came back to PGH where he started his surgical residency. He got a good, general surgical training and spent some time as a resident in Dr. Ferguson's office. That is where he met his lovely wife, Julie.

Prior to his discharge from the armed services, Elmer and I visited the Sloan Kettering Institute and arranged for Dr. Allen O. Whipple to exchange residents between Memorial and PGH so that Elmer and I were to spend six months at Memorial and Memorial was to send one of their residents to PGH. As it turned out, Elmer spent the entire year at Memorial and I preferred to stay at PGH. Elmer then went to work with Dr. Bill Erb in Ridley Park and an opportunity arose for him to join the staff at Our Lady of Lourdes Hospital in Camden. Elmer went there and subsequently became Chief of Surgery.

Elmer was a hard-working, dedicated surgeon, very thorough, very capable and well-versed in many aspects of surgery. At that time, being in general surgery and pioneering many areas meant that general surgeons did all kinds of surgery. Elmer and I performed one of the first microcommissurotomies with Dr. Charles Bailey at Philadelphia General Hospital in 1947. Elmer loved to teach and to learn. He arranged to visit various clinics around the United States and in foreign countries. He became very active with the Camden Medical Society and was especially interested in the malpractice field, which was getting out of hand. He subsequently worked at Cooper Hospital and was also on the staff of the University of Pennsylvania and Jefferson Medical School. He was a dedicated teacher and spent many hours taking care of the sick and needy, giving no thought to the cost; whether or not a patient

could pay, he gave the same kind of service. At PGH there were no fees for service and the stipends as a resident were very low. He continued to work at PGH until it closed and also did much free work at Our Lady of Lourdes. He spent many hours running back and forth among the several hospitals he operated in.

Subsequently, Elmer retired and shortly thereafter developed a malignant brain tumor which took his life. He had lived through one of the great eras of medical advancement, with the advent of antibiotics, tremendous improvements in anesthesia, replacement of body parts, particularly with use of the laser, and many newer developments which continue to change and modify the practice of surgery.

To me, Elmer was a surgeon with the eye of an eagle, the heart of a lion, the courage of a warrior, the gentleness of a maiden, the empathy of an angel, along with honesty, loyalty, dependability, and dedication to duty. Philadelphia and Camden have lost a great surgeon. We will all miss him; he has contributed much to those of us who knew and worked with him.

—MANRICO TRONCELLITI, M.D.

JOHN ROYAL MOORE, M.D.
1899-1986

John Royal Moore, M.D., died at the age of 87 in Stone Harbor, New Jersey. He was born in Elko, Nevada on December 25, 1899. His father was the sheriff of that town and his mother ran a frontier stagecoach hotel and also taught school. The family shortly moved to Sagerton, Texas, and when John Moore was ten his mother moved the family to greener pastures in Salem, Oregon.

John graduated from Willamette University and received his medical degree at the University of California in 1925. His residency in orthopaedics was in the University of California program and this included a two-year period as Resident Surgeon in the Shriner's Hospital for Crippled Children in San Francisco.

After residency, he decided that he would like to write a history of the major orthopaedic centers in the United States and to implement this goal he signed on as an oiler on the S.S. Mongolia, sailing from San Francisco and arriving in New York City in the fall of 1927. His first stop along the historical route was Atlanta, Georgia where he met Drs. Hoke, Thornton, and Kite. When he successfully solved the problem of a chronic draining hip which had its origin in an appendiceal abscess, he was asked by Dr. Hoke to stay on as his Fellow at a time when Fellowships were unheard of. In 1928, the position of Chief Surgeon at the Shriners Hospital in Philadelphia became available, and on the recommendation of Michael Hoke, he was appointed Chief Surgeon there, assuming his duties in June, 1928, at the age of 28. Two years later he was appointed the first Professor of Orthopaedic Surgery at Temple University Medical Center, holding both positions until retirement in 1965.

His practice was extremely demanding, not only because of the number of patients, but the logistics and the distance between Shriners Hospital and Temple University Medical Center. It was mainly because of the travel time involved between his home at Shriners Hospital and the busy accident dispensary at Temple that the decision was made to reduce all the fractures on Tuesday, which was the birth of delayed reduction. He proved the practicality of the precept that fractures don't have to be done immediately unless compromised by circulatory or neurologic problems or the open fracture — a precept that is true today and was embraced by the Armed Forces during World War II and subsequently.

His publications, though not numerous, were well-conceived. He utilized the delayed bone graft in salvaging many children's legs with pseudoarthrosis of the tibia, and his cartilaginous cup arthroplasty was a very early use of autologous cartilage grafts to maintain the integrity of hip function with nonunions of fracture of the femoral neck. He was a sports medicine physician before the specialty had been conceived, taking care of the injuries of all the professional teams in Philadelphia for a period of 30 years.

He was Vice-President of the American Academy of Orthopaedic Surgery and President of the American Orthopaedic Association. His honors were many, but the one that he seemed to covet most was the prestigious Stritmatter Award given by the City of Philadelphia, on rare occasions, to the Outstanding Physician in the region.

His interests included bird watching and flying and he excelled in both. He commuted on weekends to his home and family in Stone Harbor, flying down when

weather permitted and driving by motorcycle when it didn't. He made a solo flight across the United States in 1940, and was an enthusiastic supporter of the use of the private plane for transportation.

He was an excellent surgical technician, insisting on in-depth and thorough preparation by all of his assistants and was a stern disciplinarian if the results of that preparation did not meet with his approval. His teaching was by example and demonstration.

Stories about "Dinty" Moore are legion, many apocryphal, but, if not all true, they received credence because of his lifestyle and personality.

His wife, Isabelle, and he were married in 1938 in Westminster Abbey; she died in 1985. He is survived by four daughters, ten grandchildren, and four great-grandchildren. He will be sorely missed by the many patients and physicians who were touched by his brilliance.

—HOWARD STEEL, M.D.

N. HENRY MOSS, M.D.
1925-1990

Dr. Moss was born in Philadelphia on April 6, 1925. He was educated at Princeton University and the School of Medicine of the University of Pennsylvania. He served his internship at the Hospital of the University of Pennsylvania and stayed on as a surgical resident under the late Dr. I. S. Ravdin, finishing as senior resident in 1954-55.

Dr. Moss was an outstanding student at Princeton, at the School of Medicine at the University of Pennsylvania and proved to be an excellent intern and resident. Not only did he take care of patients well and operate well but he was a great student of the surgicl literature and prepared a number of important papers that were published in such journals as *Surgery, Gynecology* and *Obstetrics.* He had a special interest in the pancreas, possibly accentuated by the illness of his father who succumbed from a pancreatic neoplasm. In 1960 he published with Drs. John Howard and Jonathan Rhoads a review of the reported cases of pancreatic islet cell carcinoma which included between 300 and 400 such patients. This collected series was analyzed very thoroughly for the percentage which were malignant, the sex ratio, the number of tumors that were multiple, the number that were extra pancreatic and their locations, the results of therapy in the various categories and the result of so-called blind resection of the body and tail of the pancreas when no tumor was identified at operation on patients having hypoglycemic attacks. Some years later, Dr. Howard joined with Dr. George Jordan in preparing a full length monograph of diseases of the pancreas and requested an updated account of the cases of islet cell tumors reported in the literature. At this time, Dr. Moss was able to find more than 700 such cases and these were similarly analyzed in great detail.

Henry Moss's curiosity extended to a wide range of sciences; he was actively involved with the New York Academy of Sciences, becoming successively a member of one of its important committees, of its Board and finally served as its President in 1977. He had an awareness of what was going on in many areas of science and a knowledge of many of the participants. Furthermore, he was extremely good at recruiting key talent among speakers and accumulating a notably complete and interesting symposium. Some years after completing his work as President at the New York Academy, he conceived the idea of forming an Academy of Sciences at Philadelphia along somewhat similar lines. This, he did with remarkable success and the Academy joined with some other interested persons who had won the Nobel Prize in Physiology and Medicine over the last several years. This symposium was held at St. Joseph's University and was a great success because of the quality of the papers delivered and the excellent attendance which these notable speakers attracted. It was then particularly sad that his endeavors for Philadelphia science were interrupted by attacks of angina which led ultimately to his having a coronary bypass operation not long before his demise. Actually, he was walking in the hall with his wife, Helen, just before an early discharge from the hospital when he suddenly collapsed and lost cardiac function. He died on November 30, 1990. From a professional point of view he had an exceptional career; his academic connections were with Temple University and he did much of his operative work at the Einstein

Hospital where he was an avid teacher of the house staff.

In perusing Henry Moss's curriculum vitae, I found there were many facets of his life of which I was not aware or had forgotten about. He served in the Air Force from March 1951 to December 1951. He was Chief of Surgery at Fairbanks, Alaska and was promoted to the rank of Captain. He was an active member of the Wainwright Tumor Clinic Association of Pennsylvania, serving on their Board of Directors and then as President-elect and President, 1967-68. He was elected a member of the Philadelphia Academy of Surgery in 1964. He was also active in the American Medical Writers' to Aid the Handicapped in 1987. At the national level, he rose through the ranks to be President of the American Medical Writers' Association in 1971. He was a member of the End Results Group of the National Cancer Institute for 12 years, 1958-70, and was elected Vice Chairman during five of those years—and there are many other entries. He was listed in *Who's Who in the East* and also *Who's Who in America* and was the author of some 60 scientific papers or chapters in books.

So we will recall Henry Moss as an energetic and extremely intelligent and empathetic physician with a special talent for organization and for working through groups. To my knowledge he is the only Philadelphian ever to become President of the New York Academy of Sciences and we must credit him, also, with organizing the Academy of Sciences at Philadelphia.

For 26 years he was married to Helen Kardon Moss (the mother of Brian Kardon and Bruce Kardon by a previous marriage). Henry and Helen had a daughter, Kathy Moss, who is a reporter and morning news anchor on radio station WWBD. Both of his stepsons are active in the Kardon Business. Helen is an artist and singer. She graduated from the Curtis Institute of Music and subsequently studied at the Julliard School in New York. She was active on Broadway and in operas and in 1990 she had her debut at Carnegie Hall giving a concert there in the spring that was sold out. Thus, Henry was not only a very talented person himself but surrounded himself with talented people.

Mr. President, may we have a moment of silence in memory of Dr. Henry Moss.

—JONATHAN E. RHOADS, M.D.

PASCHAL M. SPAGNA, M.D.
1935-1992

On July 30, 1992, a long-time member of the Academy, Paschal M. Spagna, M.D., died of natural causes in his summer residence in Wildwood, New Jersey. Pat was on the Council of the Philadelphia Academy of Surgery and an active member for more than two decades. Dr. Spagna was a dedicated University of Pennsylvania alumnus having completed his undergraduate, medical school and residencies in general and thoracic surgery at that institution. After completion of his surgical training with Drs. Rhoads, Ravdin and Johnson. Dr. Spagna was a Lieutenant Commander in the United States Navy and stationed at St. Alban's Naval Hospital from 1967 to 1969. From 1969 to 1980 Dr. Spagna was Chief of Cardiovascular Surgery at Episcopal Hospital and Associate Professor at Temple University Medical School. In 1980 he became the Chief of Cardiac Surgery at the Graduate Hospital and Associate Professor of Surgery at the University of Pennsylvania. From 1987 until his death, Pat was Professor of Surgery at the Medical College of Pennsylvania and Chief of Cardiothoracic Surgery.

He was the President of the Pennsylvania Association of Thoracic Surgery from 1990 to 1991, the Secretary/Treasurer of the Philadelphia Academy of Cardiology since 1976, and the President of the University of Pennsylvania Alumni Association from 1979 to 1985. He was a member of the American Medical Association, American Heart Association, American College of Surgeons, Society of Thoracic Surgeons, International Cardiovascular Society, College of Physicians and Surgeons of Philadelphia, the John Morgan Society, the Benjamin Franklin Society, the Union League, Knights of Columbus, and Sons of Italy. He was the co-author of numerous scientific papers. His early works included the parenteral hyperalimentation research in 1962 and 1963 with Dr. Jonathan Rhoads.

His superb skill and judgment as a surgeon was recognized early in his career. I can remember walking through the hospital at the University of Pennsylvania on our way to attending a conference. Pat had not been there for ten years. A voice out of nowhere called out, "Golden hands, how are you?" It was one of the old hospital employees referring to him by the nickname given as a resident. Pat was a brilliant and gifted surgeon with a photographic memory. He could quote passage, chapter, and verse of works he had seen twenty years before. He had an inquisitive mind that had to know how things worked. He often spent hours taking apart highly technical equipment and circuitry to see how it functioned.

More importantly, Pat was a gentle, compassionate, and loyal man who loved his home, his friends, and his alma mater, the University of Pennsylvania. He was a real "people" person who was always good company and made any situation more cheerful by his presence. Pat always acted as a peacemaker in every adversarial situation in which he was involved in the 23 years I knew him. I think these human qualities of loyalty, affection, and charity combined with his facile mind and skilled hands made him the beloved surgeon that so many patients, colleagues, nurses, administrators, friends and family knew. We will sorely miss him.

—GERALD LEMOLE, M.D.

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