TRANSACTIONS

OF THE

PHILADELPHIA

ACADEMY OF SURGERY

VOLUME XXVIII

1943-1950

PHILADELPHIA
PRINTED FOR THE ACADEMY
1952

NOTICE

The twenty-eighth volume of the TRANSACTIONS OF THE PHILADELPHIA ACADEMY OF SURGERY covers the eight years from 1943 to 1950, inclusive. Some of the Secretary's records for the years 1948, 1949 and 1950 were destroyed by a fire, but it has been possible to reproduce most of this material from other sources.

A large part of the cost of printing these Transactions has been borne by the J. B. Lippincott Company, publishers of the ANNALS OF SURGERY.

JONATHAN E. RHOADS,
Recorder

PRINTED IN THE UNITED STATES OF AMERICA

CONTENTS

CONSTITUTION AND BY-LAWS

(As Amended to 1950)

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, Army and Navy, and Honorary Fellows.

Section 2. The Active Membership shall be limited to seventy (70) 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 and their dues shall be five dollars ($5.00) per year.

Section 4. Upon request, any Fellow in good standing, who may remove from the City of Philadelphia, to reside at a distance exceeding thirty (30) miles from the City, may be made a Non-resident 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.

Section 5. A new class of membership shall be created consisting of such officers of the Government Services temporarily stationed in Philadelphia from time to time who shall be elected by the Academy, 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, and shall be exempt from the payment of dues.

ARTICLE IV

The Officers of the Academy shall consist of a President, two Vice-Presidents, a Secretary, a Treasurer, a Recorder, a Council, a Business Committee, and Trustees of the S. D. Gross Prize Fund and Library.

ARTICLE V

The Officers, with the exception of the Trustees of the S. D. Gross Prize Fund and Library, who shall be appointed by the President every fifth year, shall be elected by ballot each year, and shall be eligible for re-election. The term of office of the President shall not exceed two (2) years.

ARTICLE VI

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

ARTICLE VII

The candidate for admission as an Active Fellow must be a graduate of at least ten (10) years' standing of a reputable Medical School practicing his profession in the City of Philadelphia or within thirty (30) miles of the City, and must have earned some reputation in surgery as a practitioner of Surgery, a Teacher, an Author, or an original Investigator.

On and after January 1, 1940, candidates applying for admission to membership in the Philadelphia Academy of Surgery must be certified by their respective American Boards.

ARTICLE VIII

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

Any violation of the regulations of the Academy, and of the Code of Medical Ethics adopted by it, shall be punished by reprimand, suspension, or expulsion.

ARTICLE IX

The Academy shall be governed by the Code of Ethics adopted by the American Medical Association.

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.

SECTION II

SPECIAL MEETINGS

A special meeting may be called at any time by the President, and it shall be his duty to do so upon the requisition, in writing, of any three (3) Fellows.

SECTION III

QUORUM

For the transaction of ordinary business any number of Fellows shall, at any meeting, constitute a quorum. For elections, for changes in the Constitution and By-Laws, for ordering assessments, or for the appropriation or expenditure of any sum of money exceeding twenty-five dollars ($25.00), or for any other business affecting the interests of the Academy, or of its individual Fellows, ten (10) Fellows 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, approve bills ordered to be paid by the Academy, appoint committees, not otherwise provided for, announce the results of elections, and perform all other duties pertaining to his office. The Vice-Presidents shall assist the President in the discharge of his functions, and in his absence preside in the order of seniority.

SECTION V

SECRETARY

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

SECTION VI

TREASURER

It shall be the duty of the Treasurer to receive all moneys and funds belonging to the Academy, unless otherwise provided for; he shall pay all

bills when properly ordered at the instance of the Academy, collect all dues and assessments as promptly as possible, and present an annual account for audit.

SECTION VII

RECORDER

The Recorder shall receive all papers read before the Academy, and, as a member of the Business Committee, take charge of their publication. He shall submit proof copies of all papers and discussions to authors, or to those taking part in discussions, before their publication, for examination and revision.

SECTION VIII

COUNCIL

The Council shall consist of six (6) Fellows, including the President, First Vice-President, Secretary and Treasurer. It shall be its duty to report on all nominations for Fellowship; it shall act as a Board of Censors, and shall consider any business referred to it by the Academy. It shall hold meetings for the transaction of routine business upon notice from the Secretary and special meetings shall be held on the call of the President or on the call of any two (2) of its own number.

SECTION IX

TRUSTEES OF THE S. D. GROSS PRIZE FUND AND LIBRARY

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.

SECTION X

BUSINESS COMMITTEE

The Business Committee shall consist of three (3) Fellows, including the Recorder. 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 non-resident, 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 XI

ADDRESS IN SURGERY—APPOINTEE

There shall be appointed by the President at the stated meeting in February in each year, a Fellow whose duty it shall be to deliver at a stated meeting of the year following an address in Surgery.

SECTION XII

ELECTION OF OFFICERS

The Officers of the Academy shall be nominated at the December meeting of each year, and elected at the January meeting. The election shall be by ballot, and a majority of all those present shall be necessary to a choice.

SECTION XIII

PROPOSALS FOR FELLOWSHIP

Proposals for Fellowship shall be in writing signed by three (3) Fellows with a letter from each vouching for the character of the candidate. The nominations shall be referred to the Council, who shall report on the same at the second stated meeting after that at which the nominations were made.

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 considered by the Council shall be published with the notices of the meeting immediately preceding such consideration. The sponsor of any candidate is required to be present at the meeting at which the candidate is to be voted upon and to speak for him.

SECTION XIV

ELECTION OF FELLOWS

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 an election.

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

SECTION XV

SIGNING THE CONSTITUTION

Every person elected to be a Fellow shall pay the initiation fee and shall sign the Constitution and By-Laws. No person shall acquire the rights of Fellowship unless he makes payment of the initiation fee and signs the Constitution and By-Laws within three (3) months of his election.

SECTION XVI

INITIATION FEE

Every Fellow shall, on admission, pay an initiation fee of ten dollars ($10.00).

SECTION XVII

ANNUAL DUES

There shall be an annual assessment of seven dollars ($7.00), to be paid within three (3) months after the meeting in January. Fellows elected in November or December shall not be subject to the annual assessment for that year. The annual assessment for Non-resident Fellows shall be two dollars ($2.00).

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

SECTION XVIII

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

SECTION XIX

INVITED GUESTS

Any Fellow may invite any medical man in good standing to a meeting of the Academy, and every such visitor shall be introduced to the President, and by the President to the Academy, and his name entered upon the minutes. The President may invite any such person to participate in the discussion; but all invited guests shall withdraw from the meeting when matters relating to the private calendar are under consideration.

SECTION XX

ESSAYS, REPORTS AND PAPERS

All papers read before the Academy shall be considered its property, and shall be delivered to the Recorder at the time of their presentation.

Every Fellow shall be entitled to one (1) copy of every publication of the Academy.

SECTION XXI

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 XXII

ORDER OF BUSINESS

I. Scientific Proceedings:
   1. Reading of the minutes of the proceedings of the last meeting.
   2. Admission of new Fellows, and introduction of invited guests.
   3. Reports of committees on scientific business.
   4. Reading of papers.
   5. Verbal communications.

II. Private Business:
   1. Reading of the minutes of the last meeting.
   2. Unfinished business.
   3. New business.
   4. Reports of committees on private business—Annual reports.
   5. Election of Officers.
   6. Election of Fellows.
   7. Adjournment.

SECTION XXIII

RULES OF ORDER

The proceedings of the Academy shall be conducted under the usual parliamentary rules of order.

SECTION XXIV

ALTERATIONS OF THE CONSTITUTION AND BY-LAWS

No part of the Constitution or By-Laws shall be amended, altered, or repealed, except at a stated meeting subsequent to the one at which a notice

to that effect, signed by two (2) Fellows, shall have been given, and then only by a vote of three-fourths of the Fellows present.

SECTION XXV

The President shall appoint at the November meeting each year a committee on nominations consisting of three (3) Fellows. It shall be the duty of said committee to report at the December meeting proposals for nominations for the offices of President, two Vice-Presidents, Secretary, Treasurer, Recorder, Council and Business Committee.

Report of this committee, however, shall not exclude any other proposals for nominations for above offices.

FOUNDERS

(Deceased)

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, 1951

President

DR. CALVIN M. SMYTH

Vice-Presidents

DR. I. S. RAVDIN
DR. L. KRAEER FERGUSON

Secretary

DR. J. MONTGOMERY DEAVER

Treasurer

DR. S. DANA WEEDER

Recorder

DR. JONATHAN E. RHOADS

Council

DR. FRANCIS C. GRANT
DR. THOMAS A. SHALLOW

With the President, First Vice-President, Secretary, and Treasurer

Business Committee

DR. JOHN H. GIBBON, JR. (Chr.)
DR. W. EMORY BURNETT
With the Recorder

Trustees of the Samuel D. Gross Prize

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

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	1922	JOHN H. JOPSON
1884	D. HAYES AGNEW	1924	EDWARD B. HODGE
1891	WILLIAM HUNT	1926	CHARLES F. MITCHELL
1895	THOMAS G. MORTON	1928	ASTLEY P. C. ASHHURST
1898	DEFOREST WILLARD	1930	GEORGE P. MULLER
1902	RICHARD H. HARTE	1932	JOHN SPEESE
1904	HENRY R. WHARTON	1934	WALTER ESTELL LEE
1906	JOHN B. ROBERTS	1936	DAMON B. PFEIFFER
1908	WILLIAM J. TAYLOR	1938	J. STEWART RODMAN
1910	ROBERT G. LECONTE	1940	ELDRIDGE L. ELIASON
1912	GWILYM G. DAVIS	1942	ROBERT H. IVY
1914	JOHN H. GIBBON	1944	HUBLEY R. OWEN
1916	CHARLES H. FRAZIER	1946	JOHN B. FLICK
1918	EDWARD MARTIN	1948	THOMAS A. SHALLOW
1920	GEORGE G. ROSS	1950	CALVIN M. SMYTH

VICE-PRESIDENTS

ELECTED		ELECTED	
1880	D. HAYES AGNEW	1889	JOHN H. PACKARD
1880	R. J. LEVIS	1891	WILLIAM W. KEEN
1884	SAMUEL W. GROSS	1891	J. EWING MEARS
1898	JOHN ASHHURST, JR.	1922	CHARLES F. MITCHELL
1900	RICHARD H. HARTE	1924	ASTLEY P. C. ASHHURST
1900	HENRY R. WHARTON	1926	ASTLEY P. C. ASHHURST
1902	JOHN B. DEAVER	1926	GEORGE P. MULLER
1904	JOHN B. ROBERTS	1928	JOHN SPEESE
1905	WILLIAM J. TAYLOR	1930	WALTER ESTELL LEE
1906	ROBERT G. LECONTE	1932	DAMON B. PFEIFFER
1908	G. G. DAVIS	1934	J. STEWART RODMAN
1910	JOHN H. GIBBON	1942	JOHN B. FLICK
1912	CHARLES H. FRAZIER	1943	THOMAS A. SHALLOW
1914	EDWARD MARTIN	1945	CALVIN M. SMYTH
1916	GEORGE G. ROSS	1948	L. KRAEER FERGUSON
1918	JOHN H. JOPSON	1936	E. J. KLOPP
1919	H. C. DEAVER	1938	ELDRIDGE L. ELIASON
1920	JOHN H. JOPSON	1938	ROBERT H. IVY
1920	EDWARD B. HODGE	1940	HUBLEY R. OWEN
		1950	I. S. RAVDIN

SECRETARY

ELECTED		ELECTED	
1880	J. Ewing Mears	1922	Hubley R. Owen
1885	J. Henry C. Simes	1930	DeForest P. Willard
1893	Thomas R. Neilson	1935	Henry P. Brown, Jr.
1896	William J. Taylor	1940	John B. Flick
1905	John H. Gibbon	1942	L. Kraeer Ferguson
1909	Charles F. Mitchell	1943	Calvin M. Smyth
1915	George P. Muller	1945	L. Kraeer Ferguson
1920	J. Stewart Rodman	1948	J. Montgomery Deaver

TREASURER

ELECTED		ELECTED	
1880	William Hunt	1920	Duncan L. Despard
1891	William G. Porter	1922	William B. Swartley
1904	James P. Hutchinson	1935	L. Kraeer Ferguson
1911	Edward B. Hodge	1938	Harry E. Knox
	1947	S. Dana Weeder	

RECORDER

ELECTED		ELECTED	
1880	John B. Roberts	1905	John H. Jopson
1881	DeForest Willard	1915	John Speese
1884	C. B. G. deNancrede	1920	Henry P. Brown, Jr.
1884	J. Ewing Mears	1922	J. William Bransfield
1891	Lewis W. Steinbach	1926	Calvin M. Smyth, Jr.
1902	John H. Gibbon	1937	Adolph A. Walkling
	1950	Jonathan E. Rhoads	

COUNCIL

ELECTED		ELECTED	
1880	John Ashhurst, Jr.	1927	Charles F. Mitchell
1880	John H. Brinton	1930	Astley P. C. Ashhurst
1894	William B. Hopkins	1930	Hubley R. Owen
1895	Henry R. Wharton	193_	George P. Muller
1898	Thomas R. Neilson	1935	DeForest P. Willard
1900	W. Joseph Hearn	1936	Walter Estell Lee
1902	Robert G. LeConte	1936	Robert H. Ivy
1906	Thomas R. Neilson	1940	J. Stewart Rodman
1910	J. Chalmers DaCosta	1940	Damon B. Pfeiffer
1920	Charles F. Mitchell	1941	Edward B. Hodge
1922	George G. Ross	1942	Thomas A. Shallow
1922	James H. Baldwin	1942	Eldridge L. Eliason
1923	William J. Taylor	1943	Robert H. Ivy
1924	John H. Jopson	1946	Hubley R. Owen
1924	John Speese	1947	Charles F. Mitchell
1925	Edward B. Hodge	1948	Francis C. Grant
1926	Damon B. Pfeiffer	1950	Thomas A. Shallow

With President, Vice-President, Secretary and Treasurer

BUSINESS COMMITTEE

ELECTED		ELECTED	
1895	William J. Taylor	1900	G. G. Davis
1895	DeForest Willard	1902	John H. Jopson
1896	Richard H. Harte	1905	George G. Ross
1897	Robert G. LeConte	1908	Francis T. Stewart

1914	John Speese	1930	John B. Flick
1916	Walter Estell Lee	1931	Henry P. Brown, Jr.
1916	Morris Booth Miller	1932	Edward T. Crossan
1917	Damon B. Pfeiffer	1935	B. Franklin Buzby
1917	Astley P. C. Ashhurst	1936	John B. Flick
1919	A. Bruce Gill	1938	L. Kraeer Ferguson
1919	J. Stewart Rodman	1940	J. Montgomery Deaver
1920	Arthur Billings	1942	Calvin M. Smyth
1922	Damon B. Pfeiffer	1943	Frederick A. Bothe
1924	DeForest P. Willard	1943	W. Emory Burnett
1928	Walter Estell Lee	1944	Adolph A. Walkling
1930	Edward T. Crossan	1946	J. Montgomery Deaver
	1950	John H. Gibbon, Jr.	

With the Recorder

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.

FELLOWS OF THE PHILADELPHIA ACADEMY OF SURGERY

1949 Allbritten, Frank F., Jr., M.D., F.A.C.S., 1025 Walnut Street. Assistant Professor of Surgery, Jefferson Medical College; Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Jefferson Medical College Hospital; Surgeon, Pennsylvania Hospital; Surgical Director, Barton Memorial Division of Jefferson Hospital.

1928 *Bates, William, B.S., M.D., F.A.C.S., 2029 Pine Street. Professor of Surgery and Chairman of Department of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Graduate Hospital, Presbyterian Hospital and Wills Eye Hospital.

1915 *Billings, Arthur E., M.D., 2020 Spruce Street. Formerly Clinical Professor of Surgery, Jefferson Medical College; Formerly Surgeon-in-Chief, Bryn Mawr Hospital; Consulting Surgeon Bryn Mawr Hospital.

1934 †Birdsall, Joseph C., A.M., M.D., F.A.C.S., 1900 Spruce Street. Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Urologist, Graduate Hospital of the University of Pennsylvania; Consulting Urologist, Presbyterian Hospital.

1929 *Bothe, Albert E., A.B., M.D., M.S.C., D.S.C., 255 South 17th Street. Associate Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Urologist, Jeanes Hospital, Misericordia Hospital and Fitzgerald-Mercy Hospital.

1928 *Bothe, Frederick A., B.S., M.D., M.S., F.A.C.S., 255 South 17th Street. Assistant Professor of Surgery, School of Medicine, University of Pennsylvania; Surgical Chief, Presbyterian Hospital; Chief of Department of Surgery, Jeanes Hospital.

1932 *Bower, John O., M.D., F.A.C.S., 2008 Walnut Street. Surgeon, Philadelphia General Hospital, St. Luke's and Children's Medical Center.

1921 *Boykin, Irvine M., M.D., Boykin, South Carolina. Formerly, Assistant Professor of Surgery, School of Medicine, University of Pennsylvania; and Formerly, Director of Surgery, Episcopal Hospital.

* Denotes Senior Fellow.

† Deceased since 1951.

1921 *Bransfield, John William, M.D., F.A.C.S., 2101 Spruce Street. Professor of Oral Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, American Oncologic Hospital, Doctor's Hospital, Clinic for Cleft Palate and Allied Conditions at Children's Hospital; Consulting Surgeon, St. Vincent's Hospital for Women and Children.

1944 *Brooke, John A., A.M., M.D., Sc.D., F.A.C.S., F.A.A.O.S., 1431 Spruce Street. Emeritus Professor of Orthopedic Surgery, Hahnemann Hospital; Emeritus Senior Attending Surgeon and Courtesy Staff, Orthopedic Department, Hahnemann Hospital.

1938 Burnett, W. Emory, M.D., F.A.C.S., 3401 North Broad Street. Professor of Surgery and Head of the Department of Surgery, Temple University School of Medicine; Chief of Surgery, Temple University Hospital; Visiting Surgeon, Philadelphia General Hospital; Consulting Surgeon, Shriner's Hospital for Crippled Children; Thoracic Consultant, Philadelphia General Hospital.

1949 Chodoff, Richard Joseph, A.B., M.D., F.A.C.S., 255 South 17th Street. Instructor of Surgery, Jefferson Medical College; Associate Surgeon, Mount Sinai Hospital; Assistant Surgeon, Jefferson Medical College Hospital; Chief Surgeon, Kensington Hospital.

1943 Colonna, Paul C., A.B., M.D., F.A.C.S., 3400 Spruce Street. Professor of Orthopedic Surgery, School of Medicine, University of Pennsylvania; Professor of Orthopedic Surgery, Graduate School of Medicine, University of Pennsylvania; Chief, Orthopedic Surgery, Hospital of the University of Pennsylvania; Orthopaedic Surgeon-in-Chief, Children's Seashore House, Atlantic City, New Jersey; Consulting Orthopaedic Surgeon, Children's Hospital, Rush Hospital, Valley Forge Army Hospital, and the Alfred I. DuPont Institute, Wilmington, Delaware.

1919 *Crossan, Edward T., M.D., R.F.D. 1, Conshohocken, Pennsylvania. Resident Surgeon Lumberman's Mutual Casualty Company; Assistant Professor of Surgery, School of Medicine, University of Pennsylvania.

1932 Curtis, Lawrence, A.B., D.D.S., M.D., F.A.C.S., 255 South 17th Street. Professor of Maxillofacial Surgery, School of Dentistry, University of Pennsylvania; Professor of Plastic Surgery, Graduate School of Medicine, University of Pennsylvania; Chief, Plastic Surgery Service, Presbyterian Hospital and Graduate Hospital of the University of Pennsylvania; Consultant in Plastic Surgery, Phoenixville Hospital, Phoenixville, Pennsylvania.

1939 *Davis, David M., B.S., M.D., 255 South 17th Street. Professor Emeritus of Urology, Jefferson Medical College; Visiting Urologist, Jefferson Medical College Hospital.

1934 Deaver, J. Montgomery, B.S., M.D., F.A.C.S., 1830 Delancey Place. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Professor of Surgery, Jefferson Medical College; Chief, Surgical Service "A," Lankenau Hospital, and Children's Hospital of the Mary J. Drexel Home.

1930 *Deibert, Irvin E., B.A., M.D., F.A.C.S., 538 Cooper Street, Camden, New Jersey. Chief, Surgical Service and Director of Department of Surgery, Cooper Hospital, Camden, New Jersey, Chief, Surgical Service, Zurbrugg Memorial Hospital, Riverside, New Jersey; Lecturer, Graduate School of Medicine, University of Pennsylvania, and Consulting Surgeon, Camden County Hospital for Treatment of Tuberculosis.

1949 DePalma, Anthony F., M.D., 255 South 17th Street. James Edwards Professor of Orthopedic Surgery, Jefferson Medical College; Attending Orthopedic Surgeon and Head of Department of Orthopedics, Jefferson Medical College Hospital; Attending Orthopedic Surgeon, Methodist-Episcopal and Philadelphia General Hospitals.

1928 *Downs, T. McKean, M.D., 921 Mount Pleasant Road, Bryn Mawr, Pennsylvania. Formerly, Assistant Surgeon, Pennsylvania Hospital, Bryn Mawr Hospital and Germantown Hospital.

1944 Eger, Sherman A., B.A., M.D., F.A.C.S., 2029 Delancey Street. Associate Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital; Chief Surgeon, Delaware County Hospital.

1909 *Elmer, Walter G., B.S., M.D., F.A.C.S., 1801 Pine Street. Emeritus Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Emeritus Professor of Orthopedic Surgery, Woman's Medical College of Pennsylvania; Active Consultant in Orthopedic Surgery, Philadelphia General Hospital; Consulting Orthopedic Surgeon, Graduate Hospital of the University of Pennsylvania, Woman's Medical College Hospital, Jewish Hospital, and Municipal Hospital for Contagious Diseases.

1934 Engel, Gilson Colby, A.B., M.D., F.A.C.S., 255 South 17th Street. Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Professor of Surgery, Jefferson Medical College; Chief, Surgical Service "B," Lankenau Hospital and Children's Hospital of the Mary J. Drexel Home.

1941 Erb, William H., A.B., M.D., F.A.C.S., 4 Morton Avenue, Ridley Park, Pennsylvania. Professor of Clinical Surgery, School of Medicine, University of Pennsylvania; Surgeon, Philadelphia General Hospital and the Hospital of the University of Pennsylvania; Chief of Surgery, Taylor Hospital, Ridley Park, Pennsylvania.

1931 FERGUSON, L. KRAEER, A.B., M.D., F.A.C.S., 133 South 36th Street. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Professor of Surgery, Woman's Medical College of Pennsylvania; Surgeon, Graduate Hospital of the University of Pennsylvania, Woman's Medical College Hospital, Philadelphia General Hospital, and Doctor's Hospital; Consulting Surgeon, U. S. Naval Hospital, Philadelphia.

1926 *FLICK, JOHN B., M.D., F.A.C.S., 330 South 9th Street. Professor of Clinical Surgery, School of Medicine, University of Pennsylvania; Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania; Director, Division of Surgery, Pennsylvania Hospital; Surgeon-in-Chief, Bryn Mawr Hospital; Consultant in Surgery, Valley Forge Army Hospital.

1941 FRY, KENNETH EVANS, B.S., M.D., F.A.C.S., 255 South 17th Street. Associate Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital; Civilian Consultant, Valley Forge Army Hospital; Consulting Surgeon, Girard College.

1942 GAMON, ROBERT S., A.B., M.D., F.A.C.S., 514 Cooper Street, Camden 2, New Jersey. Surgeon, Cooper Hospital, Camden, N. J.; Consulting Surgeon, Zurbrugg Memorial Hospital, Riverside, N. J.

1939 GEIST, DONALD C., A.B., M.D., F.A.C.S., 1930 Chestnut Street. Clinical Instructor in Surgery, Woman's Medical College of Pennsylvania; Chief of a Surgical Service, Misericordia Hospital; Associate in Surgery, Fitzgerald-Mercy Hospital and Jeanes Hospital; Courtesy Surgical Staff, Woman's Hospital.

1899 *GIBBON, JOHN H., M.D., D.Sc., Lynnfield Farm, Media, Pennsylvania. Emeritus Professor of Surgery, Jefferson Medical College; Consulting Surgeon, Pennsylvania Hospital, Bryn Mawr Hospital and Jefferson Medical College Hospital.

1933 GIBBON, JOHN H., JR., A.B., M.D., F.A.C.S., 1025 Walnut Street. Professor of Surgery and Director of Surgical Research, Jefferson Medical College; Attending Surgeon, Jefferson Medical College Hospital; Consulting Surgeon, Pennsylvania Hospital.

1914 *GILL, A. BRUCE, A.B., M.D., D.Sc., 1930 Chestnut Street. Emeritus Professor of Orthopedic Surgery, School of Medicine, University of Pennsylvania; Orthopedic Consultant, Presbyterian Hospital; Consulting Orthopedic Surgeon, Alfred I. Du Pont Institute for Crippled Children, Wilmington, Delaware.

1928 *GILMOUR, WILLIAM R., M.A., M.D., F.A.C.S., 1524 Chestnut Street. Visiting Surgeon, Methodist Episcopal Hospital; Surgeon, Northeastern Hospital.

1932 GOLDSMITH, N. RALPH, M.D., F.A.C.S., 1351 Tabor Road. Senior Attending Surgeon and Chief of Fracture Service, Albert Einstein Medical Center.

1925 *GRANT, FRANCIS CLARK, A.B., M.D., F.A.C.S., 3400 Spruce Street. Professor of Neurosurgery, School of Medicine, University of Pennsylvania; Professor of Neurosurgery, Graduate School of Medicine, University of Pennsylvania; Chief, Neurosurgical Service, Hospital of the University of Pennsylvania and Graduate Hospital of the University of Pennsylvania.

1934 GREENE, LLOYD B., M.D., 801 Medical Arts Building, 16th and Walnut Streets. Associate Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Urologist, Burlington County Hospital and Bryn Mawr Hospital; Associate Urologist, Pennsylvania Hospital.

1939 GROFF, ROBERT ARMAND, A.B., M.D., F.A.C.S., 1930 Chestnut Street. Assistant Professor of Neurosurgery, Graduate School of Medicine, University of Pennsylvania; Chief, Neurosurgical Service, Graduate Hospital of the University of Pennsylvania; Chief Neurosurgical Consultant, Philadelphia General Hospital, Pennsylvania Hospital, Presbyterian Hospital, Bryn Mawr Hospital, Abington Memorial Hospital, and Valley Forge Army Hospital.

1945 HAWTHORNE, HERBERT R., M.D., F.A.C.S., 250 South 18th Street. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Chief of a Surgical Service, Graduate Hospital of the University of Pennsylvania.

1913 *HEARN, WILLIAM P., B.S., M.D., F.AC.S., 227 South 42nd Street. Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital.

1922 *HERMAN, LEON, B.S., M.D., D.Sc., 740 Beacom Lane, Merion, Pennsylvania. Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Chief, Urology Service, Pennsylvania Hospital and Bryn Mawr Hospital.

1925 *HINTON, DRURY, M.D., F.A.C.S., F.I.C.S., 4501 Cedar Lane, Drexel Hill, Pennsylvania. Director of Surgery, Delaware County Hospital; Associate Surgeon, Fitzgerald-Mercy Hospital; Consultant Surgeon, Woman's Hospital.

1934 HOWELL, JOHN CARNETT, M.D., F.A.C.S., 326 South 19th Street. Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Graduate Hospital of the University of Pennsylvania and Presbyterian Hospital; Visiting Chief in Surgery, Radiological Division, Philadelphia General Hospital.

1948 IVERSON, PRESTON C., A.B., M.D., 1930 Chestnut Street. Associate Professor of Plastic Surgery, Graduate School of Medicine, University of Pennsylvania; Instructor of Plastic Surgery, Cornell University Medical Center, New York City; Chief, Plastic Surgery, Radiology Division, Philadelphia General Hospital; Associate Surgeon in Plastic Surgery, Pennsylvania Hospital; Assistant Attend-

ing Surgeon and Chief of Plastic Surgery, Bryn Mawr Hospital; Assistant Plastic Surgeon, Graduate Hospital of the University of Pennsylvania; Chief, Maxillofacial Surgery, Delaware County Hospital; Consulting Plastic Surgeon, Valley Forge Army Hospital and Walter Reed General Hospital, Washington, D. C.; Plastic Surgeon, Head and Neck Service, Memorial Hospital, New York City.

1915 *Ivy, Robert Henry, M.D., D.D.S., F.A.C.S., 1930 Chestnut Street. Emeritus Professor of Plastic Surgery, School of Medicine, University of Pennsylvania; Emeritus Professor of Plastic Surgery, Graduate School of Medicine, University of Pennsylvania; Emeritus Professor of Maxillofacial Surgery, School of Dentistry, University of Pennsylvania; Consultant in Plastic Surgery, Graduate Hospital of the University of Pennsylvania, Presbyterian and Children's Hospitals; Consultant in Plastic Surgery, U. S. Naval Hospital, Philadelphia.

1946 Jaeger, J. Rudolph, A.B., M.D., F.A.C.S., 1025 Walnut Street. Clinical Professor of Neurosurgery, Jefferson Medical College; Chief of Department of Neurosurgery, Jefferson Medical College Hospital and Wills Eye Hospital; Neurosurgical Consultant, Coatesville Veterans Administration Hospital.

1922 *John, Rutherford L., B.S., M.D., 256 South 21st Street. Associate in Orthopedic Surgery, School of Medicine, University of Pennsylvania; Consulting Orthopedic Surgeon, Episcopal Hospital, St. Christopher's Hospital and Fitzgerald-Mercy Hospital.

1942 Johnson, Julian, M.D., Sc.D. (Med.), F.A.C.S., 3400 Spruce Street. Professor of Surgery, School of Medicine, University of Pennsylvania; Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Chief, Surgical Division I, Hospital of the University of Pennsylvania; Senior Surgeon, Children's Hospital; Associate Surgeon, Philadelphia General Hospital; Consulting Thoracic Surgeon, Valley Forge Army Hospital.

1947 Kaplan, Louis, A.B., M.D., F.A.C.S., 2040 Pine Street. Clinical Professor of Surgery, Hahnemann Medical College; Chief, Surgical Service II, Mount Sinai Hospital.

1938 King, Orville Carried, A.B., M.D., F.A.C.S., 330 South 9th Street. Associate in Surgery, School of Medicine, University of Pennsylvania; Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Pennsylvania Hospital, Presbyterian Hospital, and Benjamin Franklin Clinic of Pennsylvania Hospital.

1930 *Knox, Harry E., M.D., F.A.C.S., 719 Sixty-sixth Avenue. Chief Surgeon, Germantown Hospital; Consulting Surgeon, St. Christopher's Hospital.

1914 *Laws, George M., B.S., M.D., 1907 Spruce Street. Consulting Gynecologist, Presbyterian Hospital.

1916 †Lee, Walter Estell, M.D., F.A.C.S., 1833 Pine Street. Emeritus Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon, Pennsylvania Hospital, Graduate Hospital of the University of Pennsylvania, Bryn Mawr Hospital, Germantown Hospital and Burlington County Hospital.

1938 Lehman, James A., M.D., F.A.C.S., 255 South 17th Street. Chief Surgeon, Chestnut Hill Hospital, Roxborough Memorial Hospital, St. Joseph's Hospital, Misericordia Hospital and Fitzgerald-Mercy Hospital.

1932 Lemmon, William T., B.S., M.D., F.A.C.S., 133 South 36th Street. Clinical Professor of Surgery and Assistant Professor of Applied and Topographic Anatomy, Jefferson Medical College; Attending Surgeon, Philadelphia General Hospital and Doctors' Hospital; Assistant Surgeon, Jefferson Medical College Hospital.

1934 Levering, J. Walter, M.D., F.A.C.S., Rydal, Pennsylvania. Visiting Surgeon, Abington Memorial Hospital.

1926 *Lipshutz, Benjamin, A.B., M.D., F.A.C.S., 2031 Locust Street. Assistant Professor of Neuroanatomy, Jefferson Medical College; Surgeon, Mount Sinai Hospital.

1946 Manges, Lewis C., Jr., A.B., M.D., F.A.C.S., 2001 Delancey Place. Assistant Clinical Professor of Surgery, Woman's Medical College of Pennsylvania; Assistant Demonstrator in Surgery, Jefferson Medical College; Attending Surgeon, Philadelphia General Hospital and Woman's Medical College Hospital; Assistant Surgeon, Doctors' Hospital, Jefferson Medical College Hospital and Philadelphia Hospital for Contagious Diseases; Chairman, Surgical Section, Mercy-Douglas Hospital.

1940 May, Hans, M.D., F.A.C.S., 255 South 17th Street. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital, Children's Hospital of the Mary J. Drexel Home and Chestnut Hill Hospital; Chief, Division of Plastic and Reconstructive Surgery, Germantown Hospital; Consulting Plastic Surgeon, Abington Memorial Hospital, Memorial Hospital (Roxborough), Misericordia Hospital, Nazareth Hospital, and St. Christopher's Hospital for Children.

1929 *McCarthy, Patrick A., M.D., F.A.C.S., 1737 Chestnut Street. Assistant in Surgery and Instructor in Anatomy, Jefferson Medical College; Surgeon, Philadelphia General and St. Mary's Hospitals.

1933 †McCloskey, John F., M.D., F.A.C.S., 8720 Germantown Avenue, Chestnut Hill. Consulting Surgeon, Chestnut Hill Hospital; Emeritus Professor of Surgery, Woman's Medical College Hospital.

1943 McLaughlin, Edward Francis, A.B., M.D., F.A.C.S., 4116 North Broad Street. Associate in Surgery, Graduate School of Medicine, University of Pennsylvania; Clinical Associate in Surgery, Wom-

an's Medical College of Pennsylvania; Chief of a Surgical Service, Germantown Hospital, and Nazareth Hospital; Senior Associate in Surgery, Chestnut Hill Hospital; Clinical Associate in Surgery, Woman's Medical College Hospital.

1931 *Mecray, Paul M., M.D., F.A.C.S., 405 Cooper Street, Camden 2, New Jersey. Vice-President, Board of Managers, and Honorary Chief of Staff, Cooper Hospital, Camden, New Jersey; Vice-President, Board of Managers, New Jersey State Hospital, Trenton, New Jersey.

1904 *Mitchell, Charles Franklin, M.D., 2003 Pine Street. Consulting Surgeon, Pennsylvania Hospital, Bryn Mawr Hospital, Germantown Hospital and Chestnut Hill Hospital.

1934 Mogavero, Francesco, M.D., F.A.C.S., 1930 Chestnut Street. Clinical Associate, Woman's Medical College of Pennsylvania; Chief Surgeon, Misericordia Hospital.

1938 Moore, John R., A.B., M.D., F.A.C.S., 3401 North Broad Street. Professor of Orthopedic Surgery, Temple University School of Medicine; Visiting Professor of Orthopedic Surgery, Graduate School of Medicine, University of Pennsylvania; Chief Surgeon, Temple University Hospital and Shriner's Hospital for Crippled Children; Orthopedic Surgeon, Philadelphia General Hospital.

1921 *Murphy, Eugene C., M.D., F.A.C.S., 1841 South Broad Street. Surgeon, Doctors Hospital; Attending Surgeon, United States Public Health Service.

1938 Nicholson, Jesse T., B.S., M.D., F.A.C.S., 330 South 9th Street. Chairman of Department of Orthopedics and Professor of Orthopedic Surgery, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon, Graduate Hospital of the University of Pennsylvania, Pennsylvania Hospital, Philadelphia General Hospital, Children's Hospital, Atlantic City Children's Seashore House for Invalid Children; Consultant Orthopedist to Children's Department of Mary J. Drexel Home, Lankenau Hospital and U. S. Naval Hospital, Philadelphia.

1938 *Orr, Theodore E., B.S., M.D., 1930 Chestnut Street. Assistant Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Chief of Orthopedic Surgery, Presbyterian Hospital; Chief of Orthopedics, Chester County Hospital and Delaware County Hospital; Orthopedic Surgeon, Graduate Hospital of the University of Pennsylvania; Orthopedic Consultant, U. S. Naval Hospital, Philadelphia.

1915 *Owen, Hubley R., M.D. LL.D., F.A.C.S., 1505 Walnut Street. Emeritus Professor of Clinical Surgery, Woman's Medical College of Pennsylvania; Instructor in Surgery, Jefferson Medical College; Consultant Surgeon, Philadelphia General Hospital.

1939 Parker, Alan P., B.A., M.D., F.A.C.S., Bryn Mawr Medical Building, Bryn Mawr, Pennsylvania. Instructor in Surgery, Jefferson Medical College; Surgeon, Pennsylvania Hospital; Attending Surgeon, Bryn Mawr Hospital.

1947 Parker, William Sturges, B.A., M.D., Bryn Mawr Medical Building, Bryn Mawr, Pennsylvania. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, School of Medicine, University of Pennsylvania; Assistant Surgeon, Bryn Mawr Hospital; Surgical Consultant, Valley Forge Army Hospital.

1912 *Pfeiffer, Damon B., B.A., M.D., F.A.C.S., Abington Memorial Hospital, Abington, Pennsylvania. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon, Lankenau Hospital and Woman's Hospital; Director of Surgical Service and Director of the Pfeiffer Surgical Clinic, Abington Memorial Hospital.

1916 †Randall, Alexander, B.A., M.A., M.D., F.A.C.S., Laughlin Lane, Chestnut Hill, Philadelphia. Formerly, Professor of Urology, School of Medicine, University of Pennsylvania; Urologist, Abington Memorial Hospital, Hospital of the University of Pennsylvania and Chestnut Hill Hospital.

1938 Rankin, Lynn McGaughey, A.B., M.D., D.Sc., F.A.C.S., 200 Long Lane, Upper Darby, Pennsylvania. Instructor in Surgery, School of Medicine, University of Pennsylvania; Assistant Surgeon, Jefferson Medical College Hospital; Associate Chief Surgeon, Presbyterian Hospital; Co-Director of Surgery, Delaware County Hospital.

1924 *Ravdin, I. S., B.S., M.D., F.A.C.S., 3400 Spruce Street. John Rhea Barton Professor of Surgery, School of Medicine, University of Pennsylvania; Director, Harrison Department of Surgical Research, University of Pennsylvania; Surgeon-in-Chief, Hospital of the University of Pennsylvania.

1949 Reese, John D., B.S., M.D., 2037 Locust Street. Clinical Professor of Plastic Surgery, Jefferson Medical College; Assistant Surgeon, Division of Plastic Surgery, Jefferson Hospital; Chief of Department of Plastic Surgery, Philadelphia General Hospital; Consulting Plastic Surgeon, Montgomery County Hospital and Quakertown General Hospital, Quakertown, Pennsylvania.

1943 Rhoads, Jonathan Evans, B.A., M.D., Sc.D. (Med.), F.A.C.S., 3400 Spruce Street. Professor of Surgery and Surgical Research, School of Medicine, University of Pennsylvania; Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Assistant Director, Harrison Department of Surgical Research, University of Pennsylvania; Chief, Surgical Division II, General

Surgical Service, Hospital of the University of Pennsylvania; Senior Surgeon, Children's Hospital.

1941 RISTINE, EDWIN RUSSELL, A.B., M.D., F.A.C.S., 300 Broadway, Camden 3, New Jersey. Attending Surgeon, Cooper Hospital, Camden, New Jersey; Surgeon, Lakeland Hospital, Blackwood, New Jersey.

1928 *ROBBINS, FREDERICK ROSS, B.S., M.D., F.A.C.S., 330 South 9th Street. Associate in Surgery, School of Medicine, University of Pennsylvania; Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Bryn Mawr Hospital and Pennsylvania Hospital; Civilian Consultant in Surgery, Valley Forge Army Hospital; Medical Officer in Command, U.S.N.R., Medical Division 4-8, Medical Unit 4-8.

1913 *RODMAN, JOHN STEWART, M.D., F.A.C.S., 225 South 15th Street. Emeritus Professor of Surgery, Woman's Medical College of Pennsylvania; Consulting Surgeon, Woman's Medical College Hospital, Bryn Mawr Hospital and Presbyterian Hospital; Secretary-Treasurer, American Board of Surgery; Medical Secretary, National Board of Medical Examiners.

1945 ROSEMOND, GEORGE P., M.D., M.S., F.A.C.S., 3401 North Broad Street. Professor of Clinical Surgery, Temple University School of Medicine; Surgeon, Temple University Hospital; Visiting Surgeon, Philadelphia General Hospital; Associate Attending Surgeon, St. Christopher's Hospital for Children; Chief, Surgical Service "B", Episcopal Hospital.

1928 *ROTHSCHILD, NORMAN STANLEY, M.D., 245 South 16th Street. Surgeon, Albert Einstein Medical Center.

1930 RYAN, THOMAS J., M.D., F.A.C.S., Presidential Apartments, Philadelphia, Pennsylvania. Chief of Surgery, Misericordia and Fitzgerald-Mercy Hospitals.

1922 *SHALLOW, THOMAS A., M.D., F.A.C.S., 1611 Spruce Street. Samuel D. Gross, Professor of Surgery, Jefferson Medical College; Attending Surgeon, Jefferson Hospital; Consulting Surgeon, Philadelphia General Hospital, Montgomery County Hospital, Sacred Heart Hospital, Norristown, Pennsylvania, and Grand View Hospital, Sellersville, Pennsylvania.

1947 SHEARBURN, EDWIN W., A.B., M.D., M.S., F.A.C.S., 1830 Delancey Place. Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania; Instructor in Surgery, Jefferson Medical College; Associate Surgeon, Lankenau Hospital and Children's Hospital of the Mary J. Drexel Home; Consultant in Surgery, Valley Forge Army Hospital.

1924 *SMYTH, CALVIN MASON, B.S., M.D., F.A.C.S., Pfeiffer Clinic, Abington Memorial Hospital, Abington, Pennsylvania. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Senior Surgeon, Abington Memorial Hospital; Director of Surgery and Surgeon-in-Chief, Woman's Hospital; Consulting Surgeon, United States Naval Hospital, Philadelphia.

1945 STEINER, CHARLES A., M.D., F.A.C.S., Upper Darby National Bank Building, Upper Darby, Pennsylvania. Clinical Assistant Professor of Surgery, Woman's Medical College of Pennsylvania; Chief of Surgical Service and Chief of Vascular Surgery, Delaware County Hospital; Assistant Attending Surgeon, Bryn Mawr Hospital.

1948 STEVENS, LLOYD W., A.B., M.D., F.A.C.S., 215 Gypsy Lane, Wynnewood, Pennsylvania. Assistant Professor of Clinical Surgery, School of Medicine, University of Pennsylvania; Surgical Chief, Philadelphia General Hospital; Associate in Surgery, Presbyterian Hospital; Surgical Staff Member, Hospital of the University of Pennsylvania.

1935 *SUMMEY, THOMAS J., M.D., F.A.C.S., 800 Golf View Road, Moorestown, New Jersey. Associate in Surgery, School of Medicine, University of Pennsylvania; Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Burlington County Hospital, Mount Holly, New Jersey, and Pennsylvania Hospital.

1919 *SWARTLEY, WILLIAM BLAINE, M.D., F.A.C.S., 6002 Greene Street. Demonstrator in Anatomy, Jefferson Medical College; Consulting Surgeon, Germantown Dispensary and Hospital, Chestnut Hill Hospital, Philadelphia Hospital for Contagious Diseases and Jefferson Medical College Hospital.

1915 *THOMAS, W. HERSEY, A.B., M.D., F.A.C.S., 145 East Gorgas Lane, Mount Airy, Philadelphia. Emeritus Professor of Urology, Temple University School of Medicine; Visiting Staff, Temple University Hospital and Philadelphia General Hospital.

1928 *WAGONER, GEORGE, M.D., The Medical Building, Bryn Mawr, Pennsylvania. Professor of Orthopedic Pathology, Graduate School of Medicine, University of Pennsylvania; Consulting Orthopedic Surgeon, Pottstown Hospital, Woman's Hospital, and Bryn Mawr College; Orthopedic Surgeon, Bryn Mawr Hospital and Graduate Hospital of the University of Pennsylvania; Chief of Staff, Bryn Mawr Hospital; Commander, Medical Corps, United States Naval Reserve.

1928 *WALKLING, ADOLPH A., M.D., F.A.C.S., 136 South 16th Street. Associate Professor of Surgery, Jefferson Medical College; Surgeon, Pennsylvania Hospital and the Benjamin Franklin Clinic of the Pennsylvania Hospital; Assistant Surgeon, Jefferson Medical College Hospital; Consulting Surgeon, Frankford Hospital and Pottstown Hospital.

1928 *WEEDER, S. DANA, M.D., 250 West Tulpehocken Street. Clinical Professor of Surgery, Jefferson Medical College; Director of Sur-

gery, Germantown Hospital; Chief Surgeon, Chestnut Hill Hospital.

1919 *Willard, DeForest P., B.S., M.D., Cottage 85, Sea Island, Georgia. Emeritus Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Formerly, Consultant, Graduate Hospital, Pennsylvania Hospital, Abington Memorial Hospital, Bryn Mawr Hospital, Babies Hospital and Home of the Merciful Saviour.

1939 Willauer, George, B.S., M.D., F.A.C.S., 1930 Chestnut Street. Associate Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital; Surgeon-in-Chief, Methodist Hospital; Surgeon, Eagleville Sanatorium; Visiting Surgeon, Barton Memorial Division of the Jefferson Hospital.

1927 *Williamson, Ernest G., M.D., F.A.C.S., 6353 Woodbine Avenue. Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Instructor in Surgery, School of Medicine, University of Pennsylvania; Surgeon, Presbyterian Hospital, and Graduate Hospital of the University of Pennsylvania.

1898 *Wood, Alfred C., M.D., Ph.G., F.A.C.S., 2035 Walnut Street. Formerly Consulting Surgeon, Norristown Hospital and State Hospital, Norristown, Pennsylvania; Honorary Consulting Surgeon, Philadelphia General Hospital.

1949 Zintel, Harold A., B.S., M.D., Sc.D. (Med.), F.A.C.S., 3400 Spruce Street. Professor of Clinical Surgery, School of Medicine, University of Pennsylvania; Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon, Hospital of the University of Pennsylvania.

NON-RESIDENT FELLOWS

1919 Brown, Henry P., Jr., B.S., M.D., F.A.C.S., "Brinkwood", West Willow Grove Avenue and Cherokee Street. Consultant Surgeon, Pennsylvania Hospital and Presbyterian Hospital; Consultant in Surgery, Children's Hospital.

1931 Fleming, Bruce L., M.D., F.A.C.S., 1145 West Main Street, Stroudsburg, Pennsylvania. Formerly, Surgeon, Memorial Hospital, Roxborough; Clinical Assistant in Surgery, Jefferson Medical College Hospital.

1938 Freeman, Norman E., B.A., M.D., F.A.C.S., 1435 Fourth Avenue, San Francisco, California. Associate Clinical Professor of Surgery, University of California School of Medicine; Director, Vascular Research Laboratory, Franklin Hospital; Consultant in Vascular Surgery, Letterman General Hospital and Veterans Administration.

1941 Harney, Charles H., B.S., M.D., Veterans Administration Center, Mountain Home, Tennessee. Chief of Surgical Services, Veterans Administration Hospital, Mountain Home, Tennessee; Consulting Surgeon, Memorial Hospital, Johnson City, Tennessee.

1925 Keating, Peter M., M.D., 222 King William Street, San Antonio 4, Texas. Active Staff Orthopedic Service, Santa Rosa Hospital. Baptist Memorial Hospital, and Nix Hospital.

1933 Mason, James Bryant, A.B., M.D., M.S., F.A.C.S., 4679 South 34th Street, Arlington, Virginia. Special Assistant to the Surgeon General, United States Army for Reserve Forces.

1931 Meade, Richard Hardaway, B.S., M.D., 750 San José Drive S.E., Grand Rapids, Michigan. Consultant in Thoracic Surgery, Blodgett Memorial Hospital and St. Mary's Hospital; Senior Thoracic Surgeon, Butterworth Hospital; Consulting Thoracic Surgeon, Sunshine Sanatorium.

1934 North, John Paul, A.B., M.D., F.A.C.S., Veterans Hospital, McKinney, Texas. Clinical Professor of Surgery, Southwestern Medical School, University of Texas; Chief of Surgery, Veterans Hospital, McKinney, Texas.

1942 Schell, James F., B.S., M.D., F.A.C.S., Veterans Hospital, Wilmington, Delaware. Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania; Chief of Surgery, Veterans Hospital, Wilmington, Delaware.

1908 Sweet, Joshua E., A.M., M.D., Sc.D., F.A.C.S., Unadilla, New York. Emeritus Professor of Surgical Research, Cornell University Medical College; Honorary Chairman of the Board, The Hospital, Sidney, New York.

1934 Weber, Edgar H., B.S., M.D., F.A.C.S., 123 Southeast Second Street, Evansville, Indiana. Chief, Surgical Section, St. Mary's Hospital, Evansville, Indiana.

1923 Wells, J. Ralston, B.A., M.D., F.A.C.S., Veterans Administration Hospital, Grand Island, Nebraska. Manager, Veterans Administration Hospital, Grand Island, Nebraska.

ARMY AND NAVY MEMBERS

1948 Bell, Luther George, B.S., M.D., F.A.C.S., Captain, Medical Corps, U. S. Navy. Instructor of Surgery, Residency Training Program, and Chief of Surgery, U. S. Naval Hospital, Philadelphia, Pennsylvania.

1948 Brown, Robert B., B.S., M.D., D.Sc. (Med.), F.A.C.S., Captain, Medical Corps, U. S. Navy, U. S. Naval Hospital, National Naval Medical Center, Bethesda, Maryland.

1948 Conrad, Harold Alvin, A.B., M.D., F.A.C.S., F.I.C.S., Colonel, Medical Corps, U. S. Army. Valley Forge Army Hospital, Phoenixville, Pennsylvania. Chief of Surgical Service, Valley Forge Army Hospital.

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

ELECTED		DIED
1906	WILLIAM J. MAYO, Rochester, Minn.	July 28, 1939
1906	ROBERT ABBE, New York, N. Y.	March 7, 1928
1906	C. B. G. DENANCREDE, Ann Arbor, Mich.	May 6, 1921
1907	JOHN C. MUNRO, Boston, Mass.	December 6, 1910
1908	J. EWING MEARS, Philadelphia, Pa.	May 28, 1919
1909	LEWIS STEPHEN PILCHER, Brooklyn, N. Y.	December 24, 1934
1916	W. W. KEEN, Philadelphia, Pa.	June 7, 1932
1920	HENRY R. WHARTON, Philadelphia, Pa.	December 3, 1925
1927	JOHN CHALMERS DACOSTA, Philadelphia, Pa.	May 16, 1933
1929	D'ARCY POWER, London, England	May 18, 1941
1929	ALBIN LAMBOTTE, Esneux, Belgium	
1929	HENRI HARTMANN, Paris, France	
1929	TH. TUFFIER, Paris, France	October 27, 1929
1929	JOSEPH GUYOT, Bordeaux, France	
1929	GEORGES JEANNENEY, Bordeaux, France	
1929	F. DEQUERVAIN, Berne, Switzerland	January 23, 1940
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.	
1929	ELLISWORTH ELIOT, JR., New York, N. Y.	
1929	RUDOLPH MATAS, New Orleans, La.	
1929	DEAN D. LEWIS, Baltimore, Md.	1941
1929	EUGENE H. POOL, New York, N. Y.	1940
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.	

FELLOWS DECEASED SINCE LAST PUBLICATION

1943	JOHN F. X. JONES
1944	B. FRANKLIN BUZBY
1944	JAMES A. KELLY
1945	ROBERT S. ALSTON
1945	EDWARD B. HODGE
1945	WILLIAM JOHN RYAN
1947	WARREN B. DAVIS
1947	GEORGE P. MULLER
1948	J. BERNHARD MENCKE
1948	T. TURNER THOMAS
1949	BASIL R. BELTRAN
1949	GEORGE M. DORRANCE
1950	ELDRIDGE LYON ELIASON
1950	JOHN S. LOCKWOOD
1950	LOUIS H. MUTSCHLER

BENJAMIN FRANKLIN BUZBY

Benjamin Franklin Buzby was born July 31, 1891, at Swedesboro, New Jersey, the son of Doctor Benjamin F. and Mrs. Emma Holbrook Buzby. Thus, something more than fifty-three years comprised the span of his life, a life devoted to the healing art and particularly to his own branch, the specialty of orthopedic surgery. During this time, he accomplished much, indeed more than many men who have attained the proverbial age of three score years and ten. He was a profound student, enthusiastic worker, excellent teacher, wise counselor and a friend to many.

His early education was acquired at the local country schools. He received his Bachelor of Arts degree from the University of Pennsylvania in 1911 and the degree of Doctor of Medicine in 1914, at the same institution. His internship was served at the Episcopal Hospital, Philadelphia, Pennsylvania, in whose unit he served overseas during World War I, with the rank of Captain. During this time he was intimately associated with the late Doctors A. P. C. Ashhurst and Charles H. Frazier.

Shortly after World War I, Doctor Buzby was made chief of the Orthopedic Service at the Cooper Hospital, Camden, New Jersey. This service he made the ideal of many orthopedic surgeons, and from this source contributed much to the art and to the medical literature. He also made many original presentations before this Society. He was consulting orthopedic surgeon to the Burlington County Hospital, Burlington, New Jersey, the Underwood Hospital, Woodbury, New Jersey, the Germantown Hospital, Philadelphia, Pennsylvania, and the Zurbrugg Memorial Hospital, Riverside, New Jersey. He served these institutions well and faithfully. For many years, Doctor Buzby served as the outstanding member of the Crippled Children's Commission to the State of New Jersey and accomplished much toward the comfort of the State's small but important charges.

Doctor Buzby was past President of the Camden County Medical Society, past President of the Philadelphia Orthopedic Club, a Fellow of the American College of Surgeons, a Fellow of the American Academy of Orthopedic Surgery, a Diplomate of the American Board of Surgery, a Member of the American Orthopedic Association, the College of Physicians of Philadelphia and our own organization, the Philadelphia Academy of Surgery. He was extremely fond and proud of this group and cherished his membership.

Being an ardent golfer, Doctor Buzby was an outstanding member of the Philadelphia Doctor's Golf Association, and also served that organization as President. He was a member of the Philadelphia Country Club, Pine Valley Golf Club, Tavistock Country Club, the Racquet Club of this city, and the City Club of Camden, New Jersey.

During the early part of the year 1944, Doctor Buzby was stricken ill and it was necessary for him to cease all work. The diagnosis of an atypical leukemia was established and he died at the Cooper Hospital in Camden on Sunday, October 22, 1944.

It can be said that his sterling honesty, his high standard of attainment of the best in his own field, his dissatisfaction with the "good enough," his air of inspiring confidence in his patients, his diagnostic acumen and his careful preparation of every case intrusted to him are qualifications which we who knew him best so well appreciated. His memory will ever live with us and the many whom he helped to a more abundant life.

Irvin E. Deibert.

JAMES A. KELLY

In the annals of Philadelphia medicine, the name KELLY has occupied an honored position for several generations.

The first of the name, the grandfather of the author and the father of James A. Kelly, was a distinguished physician of this city for many years. Shortly after the termination of the Civil War, in which he served with conspicuous gallantry, he matriculated at Jefferson Medical College with the class of 1868. After graduation he began the practice of medicine in that quaint section of Philadelphia known as Manayunk. He acquired a large clientele, and his reputation was respected by everyone. He was austere and pious. One who knew him well states that he was noted for his brevity and correctness in speaking and writing. He had good sense and was utterly honest and sincere; with a love for all that was right and good and a hatred for all that was evil.

In 1866 he married Emma Ferguson and their third son, born on February 20, 1878, was named James Alphonsus. His early education was acquired in the parochial school. He was given his A.B. at La Salle College in 1897 and his M.D. at the University of Pennsylvania in 1901. Like his father, he was associated, during all his professional life, with St. Mary's Hospital, first as an interne and later as surgeon. He did special work in surgery at Boston City Hospital from 1902 to 1904, returning to Philadelphia to establish a practice in surgery. In 1905 he was appointed surgeon to St. Mary's Hospital and the following year received a similar appointment to St. Timothy's Hospital. He was associated for a time with the late John B. Roberts at the old Polyclinic Hospital, with whom he was co-author of an excellent textbook on fractures. Appointments as Surgeon to St. Joseph's Hospital in 1910 and to Misericordia Hospital in 1917 followed. He held the title of Associate Professor of Surgery in the Graduate School of the University of Pennsylvania. His last appointment was as surgeon to the Fitzgerald-Mercy Hospital in Darby.

During his lifetime he had an enormous surgical experience and few of his contemporaries exhibited equal skill. He was an extremely rapid operator, but never sacrificed care and meticulous technic for speedy completion of an operation. He excelled in fracture work and was an early enthusiast for the open reduction of fractures. His interest in this type of work never waned. Although he was not a prolific writer, he always spoke with logic and knowledge at surgical meetings. Owing to this ability, his wide experience and his enormous practice, he earned an enviable reputation as an operator and his opinion was sought by many. In the field of clinical surgery he was surpassed by few.

He was given honorary degrees by La Salle College, Villanova College and St. Joseph's College. He was a Fellow of the American College of Surgeons and a member of the Philadelphia Academy of Surgery as well as a member of the Founders Group of the American Board of Surgery. In 1905 he married Rose McDonald, who, with a daughter, Mrs. James A. Moore, survives him.

Doctor Kelly fought hard for things in which he believed, but if shown a better way, he submitted and supported. He was a man of strong convictions. His interest was never lukewarm, it was always hot. There was no department of the Hospital in which he was not interested. He criticized sharply sometimes, but always constructively. He was not always angelic, but always emphatic. Everyone knew where he stood. He had independence and courage. His promise was a fulfillment. His enthusiasm for his work was unbounded. His influence on the personnel about him was wholesome, stimulating and well worth emulating. He loved friends and had hosts of them, but his close friends he cherished as something sacred. He was a great companion. He loved to play golf and was one of the Founder members of the Doctor's Golf Association.

His greatest interest, however, was in his chosen profession. He stood at the very top in his specialty. He sought the intellectual light in his work. He was a good diagnostician and a skilled technician. He had a gentle human touch.

> 'Tis the human touch in this world that counts,
> The touch of your hand and mine,
> Which means far more to the fainting heart
> Than shelter and bread and wine,
> For shelter is gone when the night is o'er
> And bread lasts only a day;
> But the touch of the hand and the sound of the voice
> Sing on in the soul alway.

His death on March 7, 1944, was due to mesenteric thrombosis, a complication of a long-standing condition which had interrupted his career on several occasions. He died, as he wished, active to the end. He worked until the day before he was operated upon. He never recovered fully from the operation. It could truly be said of him "He hath done all things well."

JAMES A. LEHMAN.

EDWARD B. HODGE

Doctor Edward B. Hodge, the son of the Reverend Edward B. Hodge and Alice C. Van Rensselaer Hodge, was born in Burlington, New Jersey, on August 21, 1875. He graduated from Van Rensselaer Seminary in 1892, received his degree of Bachelor of Arts from Princeton University in 1896, and his degree of Doctor of Medicine from the University of Pennsylvania in 1899. He then served as interne in the Presbyterian Hospital in Philadelphia.

The prominence which he attained in his profession is attested by his many hospital appointments:

Children's Hospital
Assistant Surgeon.....1902-1907
Surgeon 1907-1925
Presbyterian Hospital
Assistant Surgeon.....1903-1910
Surgeon 1910-1941
Consulting Surgeon.....1941-1945 (June 19th)
Pennsylvania Hospital
Assistant Surgeon.....1903-1920
Associate Surgeon.....1920-1945
Germantown Hospital
Surgeon 1925-1941
Consulting Surgeon.....1941-1945
Chester County Hospital
Chief Surgeon 1927-1944
Consulting Surgeon.....1944-1945

In addition, he was an assistant to Doctor G. G. Davis at the Presbyterian Hospital and was Assistant Surgeon at the Widener Memorial School for Crippled Children from 1905 to 1942.

He was a member of the American Medical Association, the Philadelphia Academy of Surgery (President 1924-1925), the College of Physicians of Philadelphia (Vice President 1943 to June 19th, 1945), the American Surgical Association, and was a Diplomate of the American Board of Surgery (Founders Group).

He held many positions of trust and honor: Trustee of Princeton University and of Princeton Theological Seminary, Trustee of Lincoln University, member of the Board of Managers of the Children's Hospital and of the Children's Seashore House, member of the Board of Directors of the Southeastern Pennsylvania Chapter of the American Red Cross and Technical Supervisor of the Blood Donor Center. He was also a member of the Board

of Christian Education of the Presbyterian Church in the United States, a Director of the Presbyterian Publishing Company and a Director of the Northern Trust Company.

He was a Captain in the Army Medical Reserve Corps in Base Hospital Number 10 and was in active service overseas from May 19, 1917, to April 27, 1919. On September 20, 1918, he became commanding officer of Mobile Hospital Number 8. He was commissioned a lieutenant colonel on February 17, 1919.

This constitutes a long and unusual record of service to the medical profession, to his community and to his country. It testifies to his intellectual ability, his high character, his devotion to duty, his surgical skill, his fundamental good judgment, his perfect uprightness and honesty, and his keen obligation of service to his fellow man and to his country.

He sprang from a long line of eminent and God-fearing ancestors. He was deeply religious, yet he never thrust his religion upon others. He lived it. He was an aristocrat by birth and by training. It showed in his face, in his conduct and in his character. He never betrayed nor in the least degree forfeited his place in gentility.

He was generous to his younger assistants and associates and gave them opportunities to advance in their profession. Many of us are indebted to him for training in surgical skill and judgment and in medical ethics. He was not a voluminous writer, he was not a teacher in a medical school, he was not an inventor of new surgical methods and procedures, yet he exerted a lasting influence in the development of many young surgeons who were associated with him during his forty-five years of active surgery.

Doctor Hodge will stand out in our memories as a striking example of the upright, honest, kindly and unselfish gentleman, of the careful, skillful surgeon, of the conscientious citizen, who responded to all demands upon his time and energies.

A. Bruce Gill.

WARREN B. DAVIS

Doctor Warren B. Davis, who died at his home in Overbrook, Philadelphia, on July 7, 1947, was born on the Cave Spring Stock Farm in Jessamine County, Kentucky, on September 6, 1881. He was the son of Luther A. and Mary Donnohue Davis.

His background, his boyhood days, and his departure from the old homestead could be of immense interest to us, but suffice it to say that he was a direct descendant of Daniel Boone and that he was the great, great nephew of James Wilson, one of the signers of the Declaration of Independence and a Justice of the Supreme Court of the United States.

The early education of Doctor Davis was obtained in the County District Schools, supplemented by private instruction from his father, a deacon in the Baptist Church, and from his mother, who played a great part in molding his life and future.

It is from his background that those of us who were his intimates understood his love for his work, his scrupulous attention to his patients and, at times, his severity with those who were under his influence. Throughout his professional life, he began his day at seven o'clock in the morning, working until he was finished, which was often late at night. He visited his hospital patients twice a day and always on Sunday. His charitable contributions to ward patients were well known to us, and it was no uncommon occurrence for him to pay the entire hospital expense, even in private rooms, for patients who could not afford hospital care. At his own expense, he air-conditioned an operating room and a fluoroscopic room at the Jefferson Hospital.

Following his preliminary education, Doctor Davis took a special scientific course at the University of Kentucky, at the completion of which he entered Jefferson Medical College in 1906. Here he took an active part in The Spitzka Anatomic League, The Dercum Society, The Keen Surgical Society and the Hare Medical Society. He was an excellent student, graduating with honors in the Class of 1910. He received an appointment to internship in Jefferson Hospital.

As an interne, Doctor Davis came into close contact with the late Doctor William Roe, who was then pioneering in operations to correct hare lip and cleft palate. At the time of Doctor Roe's death, Doctor Davis was well on the path which eventually led him to recognition and fame for his work in plastic and reconstructive surgery. The goal, however, was not reached without tedious work, long hours and constant application.

Following his internship, Doctor Davis was awarded the Corinna Borden Keen Research Fellowship by the Faculty of the Jefferson Medical College, a fellowship which was made possible by the generosity of that master surgeon, the late Doctor W. W. Keen. Doctor Davis spent more than a year in

Germany, working on the development of the accessory sinuses and deformities and abnormalities of the skull. The specimens he prepared are still on exhibition in the Daniel Baugh Institute of Anatomy of Jefferson Medical College. The American Medical Association awarded the silver medal for these and other original work done by Doctor Davis in conjunction with Doctor J. Parsons Schaeffer. As a result of this research, Doctor Davis wrote a monograph entitled *Development and Anatomy of the Nasal Accessory Sinuses in Man.*

On his return from Germany, Doctor Davis was appointed to the Surgical Service of the late Doctor J. Chalmers DaCosta. He continued his research under Doctor J. Parsons Schaeffer in the Baugh Institute, and with the death of Doctor Roe, he became the logical successor in the field of surgery for the correction of cleft palate and hare lip, a specialty which he helped to develop and on which he became a national and international authority.

Not content to restrict his activities to this limited field, he enlarged his scope of surgery to include maxillofacial surgery, finally expanding into plastic and reconstructive surgery, so that at the time of his death he was the clinical professor of this division of surgery in the Jefferson Medical College.

Doctor Davis served his country in both World Wars. In World War I, he was a Captain in charge of maxillofacial surgery at Fort Oglethorp, Georgia. In World War II, he served on the National Faculty of Plastic and Reconstructive Surgeons.

He was the author of many papers on plastic and reconstructive surgery, and he took an active part in most of the discussions on this subject throughout the United States and Canada. For this he received an honorary degree from the University of Kentucky.

As a man, his qualities were his loyalty to his family and his friends and his devotion to his profession. He took no active part in sports, his only diversion being an occasional fishing trip on the Delaware Bay.

He is survived by his wife, Ada Springer Davis, and four sons: Warren S. Davis, J. Wallace Davis, M.D., J. Leslie Davis, and Richard C. Davis.

The death of Doctor Davis was a loss which will long be suffered by his family, his friends and the devoted patients who benefitted by his skill. With his passing, the medical profession has lost an outstanding member.

Thomas A. Shallow.

GEORGE PAUL MULLER

GEORGE PAUL MULLER, son of Philip R. and Frances Hughes Muller, was born in Chestnut Hill, Philadelphia, on June 29, 1877. He attended public schools in Philadelphia and graduated from Central High School in 1895. In 1899, he received his M.D. degree from the University of Pennsylvania. Internship followed in the Lankenau Hospital from 1899 to 1902, and he was assistant pathologist at Lankenau Hospital until 1904. He then began his teaching career in the University of Pennsylvania School of Medicine as Assistant Instructor in Surgery. He continued his connection with the University as Associate and finally as Professor of Clinical Surgery. He also served as Professor of Surgery at the Graduate School of Medicine of the University of Pennsylvania from 1920 to 1933. From 1932 to 1942, he served as Chief of Surgical Service "B" in the Lankenau Hospital and Surgeon in the Misericordia Hospital from its conception until 1945. From 1937 to 1946 he held the Grace Revere Osler Professorship of Surgery at the Jefferson Medical College. He was consulting surgeon at the Rush and the White Haven Sanitoria.

Doctor Muller served as President of the Philadelphia County Medical Society, President of the Philadelphia Academy of Surgery, President of the College of Physicians of Philadelphia, President of the American Association for Thoracic Surgery and, in 1938, as President of the American College of Surgeons, of which he was also a member of the Board of Regents. He was a member of the American Surgical Association, the Society of Clinical Surgery and the Interurban Surgical Society. He served on the Editorial Board of the *Annals of Surgery*. During World War I he was on the advisory board of the Medical Corps and during this period gave one day a week to teaching at the University of Pennsylvania.

In 1910 he received an honorary degree of Master of Science from Villanova College and in 1937 an honorary degree of Bachelor of Science from Muhlenberg College.

On September 20, 1905, Doctor Muller married Miss Helen Ramsay. Their four children, George R., Helen R., Philip and John, all survive.

From the above skeleton list of his activities and honors, it can be concluded that George Muller was no ordinary man. He had a quick, keen mind which he filled with a great variety of orderly and precise knowledge. He was greatly aided by the possession of a prodigious memory and this, in turn, by the rapidity of his perceptions and the unusual ability to fix a page of reading matter with his eyes and read without the necessity of following the type line by line. With these qualifications and with his industry, it was inevitable that he would become an outstandingly learned man who would earn recognition.

His door of entry into surgery was through surgical pathology, which aided in a sound grasp of surgical principles. He soon moved into clinical surgery and acquired a mastery of surgical technic and judgment. He wrote well and talked well. As a teacher he had few equals because of his orderly presentation and fluency. Doctor Muller contributed continuously and profusely throughout his life papers on a great variety of general surgical subjects. He also revised Davis' *Applied Anatomy*.

He was plagued through his life, particularly in his earlier years, by frequent attacks of migraine, during which he seemed irritable, curt and inconsiderate. Despite this, he had many friends and could be a charming companion. His hobbies were in earlier years fishing, later came golf and photography and always reading. He had a full life, and the world is the better for his having lived in it. He remained young in appearance and activities until almost the end of his career.

Looking backward, it would seem that certain subtle changes were occurring in his physical and mental stamina, although he looked in the best of health. Then came a severe pulmonary infection, never fully understood, which brought him practically to extremis for several days. Though he fought through, he was fatally damaged and, after progressive physical and mental deterioration, he died in coma in Margate, New Jersey, on February 18, 1947.

DAMON B. PFEIFFER.

ELDRIDGE LYON ELIASON

On March 6, 1950, at the age of seventy-one, Philadelphia lost one of its outstanding surgeons by the death of Doctor Eldridge Lyon Eliason.

Doctor Eliason, a graduate of Hotchkiss School and Yale University, came to Philadelphia and the University of Pennsylvania Medical School in 1901 where he graduated in 1905. After an internship at the University Hospital, Doctor Eliason began his preceptorship in Surgery and continued throughout the remaining part of his life in the practice of surgery in Philadelphia at the Hospital of the University of Pennsylvania, the Philadelphia General Hospital, the Presbyterian Hospital and various other hospitals.

Many honors were given to him during his lifetime. He was a member of Phi Beta Kappa, Sigma Xi and Alpha Omega Alpha honorary societies. He was a Fellow of the American College of Surgeons, American Surgical Association and several other surgical societies. He was made a fellow member of the Philadelphia Academy of Surgery in 1921 and served as its President from 1940 to 1942. Doctor Eliason served his country in World War I as a member of the Surgical Service of Base Hospital Number Twenty. He will probably be best remembered as a teacher of Surgery. He served his apprenticeship and eventually became the John Rhea Barton Professor of Surgery at the University of Pennsylvania, which position he filled from 1936 to 1945. He was a Professor of Surgery at the Graduate School of Medicine of the University of Pennsylvania from 1938 to 1946.

Doctor Eliason had many characteristics that endeared him to those who knew him and he was respected by all those who had any dealings with him. His most outstanding characteristic was his integrity. He expected the same characteristics in others and hated any form of trickery and chicanery. Another outstanding characteristic was his meticulousness. He was most meticulous about everything he did, about his person, about his surgery, about his patients and in his dealings with his associates. He instilled the spirit of carefulness and conscientiousness in all those with whom he had intimate contact.

His third characteristic was his safety. In his surgery he constantly made sure that what he did was a safe procedure. He was slow to accept new ideas and new technics unless they were tried and proven. This characteristic was recognized and instilled great confidence in his patients.

His fourth characteristic was his love of teaching. He loved to teach, not from any spirit of showmanship but because he really enjoyed and took pride in teaching younger men the art of surgery. He will be remembered by every student who had the opportunity to hear him talk. He had the ability to simplify the teaching of surgery so that the student understood and remembered what he was taught. He was noted for his use of homely ex-

pressions in his teaching, and such bywords "sure as gun's iron" and "standing out like a dirty deuce in a new deck" are two that will be well remembered by generations of medical students.

A final characteristic which will be recalled by all was his virility. From his early days he felt that a self-discipline sufficient to keep physically fit was a requirement for an active life. Despite a very busy, active professional career he always had time, even during his later years, to keep himself in excellent physical shape.

With Doctor Eliason's death, Philadelphia has lost one of its most respected surgeons, one whose memory will live long in the hearts of the surgical profession of Philadelphia. His host of patients and friends join with us in mourning our loss.

L. KRAEER FERGUSON.

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, California.

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.

* This essay has never been published by the author as required under the terms of the award.

LIST OF FELLOWS WHO HAVE DELIVERED THE ANNUAL ADDRESS

1881	S. D. Gross	1916	Edward B. Hodge
1882	D. Hayes Agnew	1917	J. Edwin Sweet
1883	William Hunt	1918	None
1884	John H. Brinton	1919	None
1885	John H. Packard	1920	John G. Clark
1886	R. J. Levis	1921	J. Torrance Rugh
1887	J. Ewing Mears	1922	George P. Muller
1888	C. B. G. deNancrede	1923	Walter Estell Lee
1889	John B. Roberts	1924	Robert H. Ivy
1890	DeForest P. Willard	1925	John Speese
1891	William G. Porter	1926	Damon B. Pfeiffer
1892	T. G. Morton	1927	Emory G. Alexander
1893	C. W. Dulles	1928	Edward J. Klopp
1894	W. B. Hopkins	1929	Edward T. Crossan
1895	John B. Deaver	1930	J. Stewart Rodman
1896	James M. Barton	1931	Hubley R. Owen
1897	Thomas R. Neilson	1932	Eldridge L. Eliason
1898	O. H. Allis	1933	George M. Dorrance
1899	William J. Taylor	1934	DeForest P. Willard
1900	None	1935	A. Bruce Gill
1901	H. R. Wharton	1936	Alexander Randall
1902	J. M. Spellissy	1937	Henry P. Brown, Jr.
1903	R. G. LeConte	1938	Isidor S. Ravdin
1904	G. G. Davis	1939	John B. Flick
1905	J. Chalmers DaCosta	1940	Francis C. Grant
1906	Richard H. Harte	1941	William Bates
1907	Edward Martin	1942	S. Dana Weeder
1908	Charles H. Frazier	1943	Frederick A. Bothe
1909	John H. Gibbon	1944	Calvin M. Smyth
1910	Astley P. C. Ashhurst	1945	Adolph A. Walkling
1911	John H. Jopson	1946	John H. Gibbon, Jr.
1912	George G. Ross	1947	L. Kraeer Ferguson
1913	William L. Rodman	1948	Jonathan E. Rhoads
1914	Alfred C. Wood	1949	Francis C. Grant
1915	Francis T. Stewart	1950	W. Emory Burnett

ANNUAL ADDRESS FOR 1943

SURGERY IN METABOLIC DISEASES*

FREDERICK A. BOTHE, M.D.

PHILADELPHIA

Fifty years ago our knowledge of diseases of the metabolism was very limited and vague. As in other fields of medicine, increased clinical experience and extensive investigative work have enabled us to acquire a more intelligent understanding of this group of diseases. It was only twenty years ago that we began to master the fear of operating upon certain glands of internal secretion, but since then many notable advances have been made in the surgery of metabolic diseases. Obstacles have been overcome which heretofore had made surgical treatment of these syndromes either extremely hazardous or impossible. It would be too lengthy to consider adequately all the metabolic diseases which have been either cured or controlled to some extent by surgical treatment. In this review, surgery of the thyroid, the surgical diabetes, hyperinsulinism, hyperparathyroidism, and tumors of the adrenal glands with metabolic changes will be considered. Lesions of the ovaries and pituitary gland which commonly produce metabolic changes will only be touched upon as is necessary for a complete discussion of the chosen topics. Collectively, the subjects under consideration are exemplary as to the progress made and to be made in the field of surgery.

THYROID DISEASE

Diseases pertaining to the thyroid gland are the most frequent metabolic disturbances which require surgical treatment. The patients were very fearful of operations performed upon the thyroid gland in the early days, and rightfully so, because of the high operative mortality. However, in the last few decades, numerous contributions have been made to the preoperative, operative, and postoperative management of these cases. Consequently the operative results have improved, and the mortality of thyroidectomy has been greatly reduced. In some large goiter centers, the mortality for thyroidectomy is 1 per cent or less. The preoperative management of the patient suffering from a toxic goiter has been more or less standardized, and today the patient is so prepared that thyroidectomy is performed at the most favorable time for each individual case.

Iodine was known to be of beneficial value in hyperthyroidism for many centuries; however, the work of Plummer[67] in 1923 instituting its preoperative use on a rational basis is perhaps the most important advance in the

*The material presented in this review formed the basis for the Annual Address before the Academy of Surgery, Philadelphia, Pennsylvania, November 1, 1943. Published in Surgery, Gynecology and Obstetrics **78**:461-476, 1944.

preoperative preparation of this group of cases. We now know the dangers of prolonged and indiscriminate use of this drug, and have learned to recognize the so-called iodine-fast, as well as the "burned out" thyroid patient. Today most medical centers have developed a routine for the management of both of these types of patients. That the thyroid gland plays a principal role in the metabolism of iodine in the body is not surprising when we realize that a gland which weighs only 20 Gm. contains one-fifth of all the iodine in the body. Salter[71] found that not more than 10 per cent of the iodine in the thyroid gland was inorganic in nature; 90 per cent or more was in organic combination and approximately two-thirds of this was in the form of di-iodotyrosine and one-third, tyrosine. In 1927 Harrington and Barger[41] showed that thyroxine was a derivative of tyrosine. Hinton[45] believes that failure to supply an adequate amount of tyrosine to the thyroid gland because of a disturbance in the protein metabolism may be a potent factor in the production of thyroid disease. The first comprehensive work published on the determination and significance of blood iodine was that by Kernan and Lahey.[66] Since then it has been repeatedly demonstrated that the blood iodine is elevated in hyperthyroidism, and returns to normal following thyroidectomy. The preoperative estimation of blood iodine has assisted in establishing the diagnosis and evaluating the severity of this disease. In 1939 Perkin and Cattell[65] reported preoperative and postoperative blood iodine levels on 256 cases of exophthalmic goiter, and analyzed the relation of these levels to the recurrence of hyperthyroidism. One hundred of these patients were followed up for one year. It was noted that the number of patients who showed a normal blood iodine level after subtotal thyroidectomy decreased with the duration of symptoms before treatment. The majority of patients with symptoms for one year had a normal blood iodine level, and following subtotal thyroidectomy, the level was increased. The incidence of recurrence in these patients was 19.7 per cent. If symptoms of hyperthyroidism were present from four to five years, the blood iodine level was generally normal and usually uninfluenced by subtotal thyroidectomy. It was believed that these findings could be used as an index of the amount of thyroid gland to be removed in patients with thyrotoxicosis.

In 1942, Hinton[45] reported a study on blood iodine in 235 patients who had, or were suspected of having, thyroid disease. In 34 patients the hyperthyroidism was of more than one year's duration, but in these cases, he found the blood iodine was elevated, which was in contrast to the findings of Perkin and Cattell.[65] In 16 patients with persistent or recurring hyperthyroidism, the incidence of an elevated blood iodine was no greater than that observed in nonthyroid patients. This strongly suggests an error in the diagnosis of hyperthyroidism, and that the decreased iodine content of the blood is not indicative of exhaustion of the thyroid gland. Many clinicians believe that blood-iodine levels will supplant the basal-metabolism estimations in the future, when a simpler method for their estimation is devised.

Another contribution of significance is the use of oxygen in thyroid crises.

The clinical appearance and well-being of the patient responds promptly to oxygen therapy. We have learned through the use of oxygen, that anoxia is a significant factor in the tachycardia of thyrotoxicosis. This is best illustrated by the patient who leaves the operating table with a pulse rate of approximately 100, but because of the diminished oxygen obtained from the atmosphere as compared with that obtained from the anesthetic mixture, his pulse promptly rises to 130 or more. For the past ten years I have had oxygen administered to all patients whose pulse rate was above 120 upon their return from the operating room. In some cases the tachycardia responds in a few hours; however, since the adoption of this routine, the postoperative course of the very toxic cases has been much smoother.

In 1930 Lahey[52] brought to our attention a type of hyperthyroidism which he called apathetic hyperthyroidism. He stated that this condition remained undiagnosed for long periods, and when diagnosed the degree of toxicity was underestimated. In an analysis of his cases, he noted that his mortality was largely related to the apathetic type of hyperthyroidism. This type is seen in the later decades of life, and the clinical and physical findings would not impress one as being those of severe hyperthyroidism. They present a picture simulating a mild form of the disease with a slight loss of weight over a period of several years: the skin is dry and wrinkled, and instead of the characteristic stare and marked nervousness of severe hyperthyroidism, they present an apathetic face, and the basal metabolic rate is only slightly elevated. At the operating table, the patients apparently withstand the operation very well; in most instances the pulse rate remains well below 100 throughout the procedure. The postoperative course is very satisfactory for the first twenty-four to thirty-six hours; then drowsiness develops, which becomes progressively worse, and finally the patient goes into coma and dies very peacefully. This is quite a different picture from that customarily seen when a toxic thyroid patient succumbs in a thyroid crisis following thyroidectomy. Lahey pointed out that these patients are more seriously ill than we formerly realized. He recommended that after careful preoperative preparation, thyroidectomy should be performed as a two-stage procedure with approximately six weeks between stages. By this method of treatment, the mortality in this group was practically eradicated. This contribution has sharpened our diagnostic acumen, and we now know that this type of thyrotoxicosis is much more prevalent than we formerly believed. Lahey not only brought to our attention the fact that this type of hyperthyroidism is frequently missed entirely by the clinician, but also pointed out that many patients die from this condition following an operation for a pathological lesion elsewhere in the body. This further substantiates the dictum which we learned from experience, of the proper time to treat the foci of infection in a patient suffering from this disease; namely, that in a patient suffering from hyperthyroidism, surgery other than that directed at the thyroid, though it may be minor in character, may precipitate a severe thyroid crisis, or be the cause of a fatality.

Considerable interest has been stimulated recently in the alterations in the

calcium and phosphorus metabolism in hyperthyroidism. In 1891 von Recklinghausen[69] first described the demineralization of the skeleton in a case of hyperthyroidism of many years' duration. Subsequently Koeppen[51] and Askanazy[6] reported similar cases. In these early cases, the pronounced demineralization was apparent at autopsy when the bones were being studied. It was many years later before the picture of general osteoporosis was demonstrated clinically in a case of hyperthyroidism by the roentgenologists. In 1929 Plummer[68] and Dunlap and Moore[31] reported 5 cases of hyperthyroidism in which there was pronounced demineralization of the skeleton. In 2 cases the bone changes were so extensive that metastatic malignancy was considered. It is generally believed that the demineralization involves practically the entire skeleton. The small and flat bones usually show the change first, and later it is evident in the spine and long bones. Shrinking of the general stature, kyphosis, and flaring of the lower ribs have all been reported. A few cases of spontaneous fracture due to the demineralization are now appearing in the literature. In 1942, Bothe, Simpson and Rowntree[13] reported a case in which the patient, suffering from long-standing hyperthyroidism, had both spontaneous and traumatic fractures of the spine. Aub and his coworkers[7] reported an increase in the excretion of calcium and phosphorus in the feces and urine in clinical hyperthyroidism; therefore, just as in cases of hyperparathyroidism, we may have patients with hyperthyroidism in a negative calcium balance. This has been confirmed by other investigators.

In the last few years, many extensive studies have been made of the degree of liver damage and diminished liver function in patients suffering from hyperthyroidism. Jaundice has frequently been observed in severe hyperthyroidism. Weller[81] and Beaver and Pemberton[9] found variable types of hepatic pathology in patients who succumbed to thyrotoxicosis. They observed acute changes consisting largely of degenerative fatty infiltration and a chronic lesion described by Weller as a patchy, chronic, parenchymatous interlobar hepatitis. Many investigators believe that further evidence of liver damage is the fact that the liver of a patient suffering from hyperthyroidism is unable to store glycogen in normal amounts. Numerous efforts have been made to determine whether these evidences of liver damage were of any significance or relation to the clinical picture of thyrotoxicosis, particularly the thyroid crises. Youmans and Warfield[84] were able to show impairment of liver function in 50 per cent of 44 patients with hyperthyroidism. Mills,[62] using the galactose test, carefully studied a small group of patients and demonstrated a diminished liver function in the presence of hyperthyroidism, which disappeared following relief of the disease by thyroidectomy.

Others believe that the hippuric-acid test is more valuable than the galactose test, in that it gives us a reliable index of one clinically important hepatic function, namely, the function of detoxification. Chesky et al.[21] made a very careful study of the liver functions determined by the hippuric-acid test in more than 300 goiter patients admitted for surgery. Under the routine treatment which we all use for thyroid crises or severe hyperthyroidism, most

patients showed improvement in liver function; in some the improvement was very slight; in others progressive hepatic insufficiency was observed. In view of these findings, the patients with the most severe liver damage received, in addition to the routine treatment, intravenous glucose with or without insulin, insulin by hypodermic injection, bile salts, liver concentrate, and glycine. In every case so treated, the liver function was either greatly improved or restored to normal. They also demonstrated decreased liver function after thyroidectomy. The decrease was most pronounced in the first forty-eight hours after operation; improvement followed thereafter. Chesky and his associates[21] did not contend that the hippuric-acid test should supplant clinical judgment as to operative risk, but suggested serial hippuric-acid tests in severe cases of hyperthyroidism as an aid in evaluating the response of the liver to this treatment. Some investigators believe that damaged liver function plays such an important part in thyroid crises that they should be called liver crises. This contention is gaining weight in that more cases have been reported in which the treatment of the liver damage has been very helpful in the severe crises.

The interrelationship between the thyroid and the anterior portion of the pituitary gland is far from being clearly understood. It has been shown that the thyroid-stimulating thyrotropic hormone elaborated by the anterior pituitary is an entity separate and distinct from the other trophic principals which can be demonstrated in the extracts of fresh anterior-lobe substance such as the gonadotropic, adrenotropic, diabeticogenic, parathyrotropic, and mammotropic hormones. In 1929 Loeb and Bassett[55] reported the production of hyperplasia of the thyroid gland in the guinea pig by the injection of extract of the anterior pituitary lobe. Later Collip[25] and Selye[73] showed experimentally that the pituitary and thyroid glands can be definitely enlarged in the female rat by the prolonged administration of hormone of the anterior pituitary lobe, and that the enlargement of both these organs ran approximately parallel to the weight increase in the ovaries. These findings suggest that there may be cases of hyperthyroidism that are pituitary in origin. Thompson and his associates[78] studied the effect of the administration of pituitary extracts containing the thyrotropic principle to 71 patients. In 39, or 56 per cent, there was an increase in the basal metabolic rate. In patients requiring more than one series of injections, it was of interest to note that the increases were greater in the first series of injections, and that the second course of treatment usually, but not invariably, failed to produce any increase in metabolism. Thompson concluded that the increased metabolic activity was temporary, in spite of prolonged treatment, and that the metabolism eventually returned to its level before treatment or even to a lower level. No increase was noted in 4 patients with marked myxedema, but in patients with mild or moderated hypothyroidism, the rate could be raised to normal; in patients with nontoxic goiter a thyrotoxic state could be produced temporarily; and in patients with exophthalmic goiter, the disease could be made temporarily more severe. It appeared that the effect could be

produced only in patients with thyroid tissue capable of responding. If these observations were confirmed by others it would tend to discredit the thought that the pituitary gland may be the etiological factor in some cases of thyrotoxicosis.

Recently we have been able to differentiate more satisfactorily the types of chronic thyroiditis. In 1896 Riedel[70] described the type of thyroiditis which bears his name. In 1912 Hashimoto[43] described a second type in which there was a lymphocytic infiltration throughout the gland. In 1922 Ewing[33] stated that they were one and the same disease, and that the histological picture found in Hashimoto's disease was the early stage of Riedel's struma. This was very widely accepted until the work of Graham[39] in 1931 showed rather conclusively that they were two distinct pathological processes. An analysis of the reported cases showed that Riedel's struma, which was supposed to be the later stage of the disease, occurs earlier in life than does Hashimoto's disease. Riedel's struma occurs in both sexes and is unilateral in from 40 to 60 per cent of the reported cases, whereas Hashimoto's disease occurs only in the female, and is always bilateral. Myxedema is more common in Hashimoto's disease than in Riedel's struma. If the gross pathological appearance and the microscopical findings in a frozen section lead to the diagnosis of Hashimoto's disease, only half of the gland should be removed, in the hope that it will postpone or possibly prevent the development of this complication. In Hashimoto's disease the pathology is confined to the thyroid gland, whereas in Riedel's struma it frequently extends through the capsule into extrathyroid tissue. This explains the frequent occurrence of grave pressure effects in Riedel's struma, and the relative infrequency of severe pressure symptoms in Hashimoto's disease. The gland is firm in Hashimoto's disease but does not become stony hard as it usually does in Riedel's struma. Even though a clinical diagnosis of either type of thyroiditis seems apparent, the firm consistency of the gland makes it necessary to explore all such cases to exclude malignancy.

In the last few decades, there have been no outstanding changes in the operative technique of thyroidectomy which have aided in the reduced mortality. However, both the mortality and postoperative complications have been greatly decreased by the modern method of administering anesthetics by a well trained physician anesthetist.

There are several steps which, many surgeons believe, have improved the operative results; namely, the division of the thyroid gland at the isthmus, and the elevation of the lateral lobes before the resection is begun. Lahey[53] suggested the routine dissection and demonstration of the recurrent laryngeal nerve. He feels rather strongly that the strip of tissue which is left over the recurrent nerve, in the technique usually employed, results in leaving too much thyroid tissue, and may be the basis for the persistence or recurrence of the hyperthyroidism. In the hands of a master surgeon such as Lahey, this procedure offered no difficulty and has been free from complications; however, I believe it is safe to say that many surgeons performing thyroidectomies would rather not deliberately expose the nerve, lest they may injure it.

SURGICAL DIABETES

Since the discovery of insulin by Banting and Best in 1921, surgery performed upon the diabetic has become more and more specialized, and the fear of both the patient and the surgeon has been conquered in a large measure by the excellent operative results obtained. The management of the diabetes has become so intricate that fortunately very few surgeons attempt to treat this feature of the case. Unquestionably, the most important factor in obtaining the excellent results has been the combined management of the surgical diabetic by the metabolist and the surgeon throughout the preoperative, operative, and postoperative periods. There are very few communities which do not have this combined service available. This field is all the more important when we realize that the number of diabetics in the United States has increased tremendously in the past fifteen years. It is now estimated that from 1½ to 2 per cent of the population of the United States are either diabetics or potential diabetics. The increase in the number of diabetics is not distributed uniformly throughout life or in either sex, but has been shown to occur to a greater extent in children and in women between forty and sixty years of age. In both of these age groups operations are frequently required, not only for conditions commonly associated with diabetes, but for lesions entirely foreign to the disease. Many predict that the stress and strain of the present emergency will further increase the number of diabetics.

The diabetes adds little or nothing to the operative risk in operations of election. This is true only if the diabetes and the general metabolic state is adequately controlled preoperatively by the metabolist. Hence, under proper preparation the diabetic of today is as good an operative risk as the condition of his cardiovascular system indicates. In emergency cases, particularly those complicated by infection, the operative risk is greatly increased, and the closest co-operation between the medical and surgical departments in the management of these cases cannot be overemphasized.

Many diabetics are controlled more easily with protamine zinc insulin. Unfortunately, it has proved unsatisfactory to use insulin in this form in the surgical diabetic, as its action may become cumulative at any time. For this reason, we have discontinued the use of protamine zinc insulin during the immediate preoperative and postoperative periods and instead use either regular or crystalline insulin. The insulin requirements of the surgical diabetic may diminish very rapidly as the operation frequently relieves the condition which created the need for a greater insulin requirement. This is seen most frequently after adequate drainage of an infection or in the preoperative treatment of severe hyperthyroidism associated with diabetes. If this possibility is not anticipated, both the preoperative and postoperative periods may be complicated by severe insulin shock.

Frequently the abdominal pain in the diabetic is difficult to evaluate. The earlier symptoms and physical findings of acidosis, which may closely simulate those found in acute abdominal conditions, have been clearly demonstrated. Even leucocytosis and fever, so characteristic of the acute abdominal condition, are associated with acidosis in a high percentage of cases. We no longer wait until the patient is drowsy or about to go into coma before acidosis is considered. These clinical findings are neurological manifestations of the terminal stages of acidosis.

When this clinical picture occurs, the surgeon is often called upon to differentiate between acidosis and a surgical condition of the abdomen; frequently it is only the urine examination and the blood-sugar and carbon-dioxide determinations which enable him to arrive at the proper diagnosis. If acidosis is present it should be controlled before surgery is performed. As a rule, this takes only a few hours and there are very few emergencies in which the risk of operation is increased as much by this delay as by performance of the operation in the presence of acidosis. If the emergency will permit, operation should be postponed until the carbon dioxide is 50 volumes per cent; in extreme cases an operation may be done with a carbon dioxide of 40 volumes per cent. Regardless of the findings upon examination of the abdomen when acidosis is found, as soon as this complication is controlled, another careful abdominal examination should be made to rule out a co-existing lesion which may require surgical treatment.

In conjunction with Beardwood,[12] a study was made to evaluate the abdominal symptoms of the diabetic in comparison with those of the non-diabetic. It was found that the most severe abdominal pain occurs in a diabetic acidosis; in the absence of acidosis, other things being equal, the abdominal pain due to an underlying pathological process is less severe in the diabetic than in the nondiabetic. The last portion of the findings has proved most valuable since there was a tendency to underestimate the presence of a surgical lesion in the abdomen of a diabetic, as the symptoms and physical findings did not appear to be severe enough. In a series of more than 50 diabetics who had lesions in the abdomen associated with pus, acidosis did not occur in a single case. This is contrary to our experience with diabetic patients who had small collections of pus elsewhere in the body.

Even though acidosis is the most hazardous complication in the surgical diabetic, infection is the most common and a serious one. The diabetic has a lowered local and general resistance to infection on account of the cardio-vascular disturbance (primarily arteriosclerosis) and the abnormal carbohydrate metabolism. If localization of the infection is not apparent and lymphatic involvement occurs, the surgical risk is great. Sepsis is borne very poorly by the diabetic, and treatment should be directed toward the infection rather than the diabetes. A vigorous attempt to make the patient's urine sugar-free is to be avoided. It is better to drain or remove the infectious process, as then the carbohydrate metabolism will improve. The blood-sugar level need not influence the decision to operate, provided that

the carbon dioxide is in the region of safety. In some cases of gangrene it has been necessary to incise and drain a collection of pus to control the diabetes, even though the carbon dioxide was a little lower than desired.

In the past we applied antiseptics locally in a very weak form in the diabetic, but this handicap has been overcome with the discovery of the sulfonamides. The 10 per cent sulfathiazole ointment has been used with excellent results in patients with diabetic gangrene involving only the digits. The number of patients successfully treated by conservative surgery has been increased by the use of this therapeutic agent. In more extensive processes the local application of microcrystals of sulfathiazole in conjunction with the oral administration of this substance has universally proved to be the most effectual treatment. In spite of these favorable reports, experience shows that we should not procrastinate in the employment of radical surgery when it is indicated by the local findings and not contraindicated by the patient's general condition. Zinc peroxide when applied as suggested by Meleney and Johnson[61] has proved very beneficial in chronic infectious processes complicated by sinus formation.

Gangrene is perhaps the most common as well as one of the most serious infectious processes which we encounter in the diabetic. There is a wide variation in the mortality rate in the various series of cases of diabetic gangrene as reported by well organized services for diseases of the metabolism. An analysis of the figures shows that the mortality is in direct proportion to the percentage of cases complicated by extensive and severe infection, and that the highest mortality occurred among the diabetics admitted to the hospital in a very toxic state, who usually have a gangrenous process which will not localize. In this last group refrigeration of the involved part, as advocated by Allen,[4] bids fair to become a therapeutic measure which will enable us to treat the condition with much greater success and greatly reduce the mortality. In his early reports Allen emphasized the fact that metabolic activity is lowered when the temperature of the tissues has been reduced, and he suggested that we lower the temperature of the infected parts in patients having a peripheral vascular lesion from which very toxic substances are absorbed. In his experimental work on animals, he applied a tourniquet at the site elected for amputation and kept the extremity distal to the tourniquet at 5°C. After the leg was kept at this temperature for several hours, he demonstrated that amputation could be done at the level at which the tourniquet was applied, or the upper limit of refrigeration of the tissues, without anesthesia. Thus, he has not only demonstrated a means of diminishing the toxic state of the patient, which permits surgical intervention with a much greater degree of safety, but he has also shown that amputation can be performed through the refrigerated tissue without the use of an anesthetic. Recently Allen[4] reported 45 cases of gangrene in which the local application of refrigeration had been used; the results were most gratifying.

There are three distinct divisions (Table 1) in the use of refrigeration in surgery[3]; (1) its use preceding operation with a tourniquet; (2) its post-

operative application to the flaps; and (3) its prolonged use without a tourniquet, for preoperative preparation in certain cases, or when an uncertainty exists as to whether operation is necessary.

Table 1. Three Divisions in the Use of Refrigeration

1. Preceding operation with tourniquet (for anesthesia)
A. For thigh amputation 2½ hrs.
B. Disarticulation at knee 2 hrs.
C. Amputation at middle of leg 2 hrs.
2. Postoperative application (applied to flaps)
A. Lowers metabolism, less blood supply necessary to nourish tissue and less sloughing
B. Inhibits pain, edema, and bacterial activity
3. Prolonged use without tourniquet
A. Arteriosclerotic legs in ice
B. In extensive gangrene to check infection and toxic absorption.

When tissue is refrigerated in preparation for operation, it is important to realize that the tissues are not frozen, but are kept between 5° and 10°C., and as near 5°C. as possible. Necrosis or frostbite do not occur. This procedure is based on the fact that tissues have been kept alive for long periods in an ice box. When properly applied, refrigeration will produce surgical anesthesia. The fundamental principle of refrigeration for surgical anesthesia is that cold nerves cannot transmit impulses and cold tissues cannot respond with shock or any other harmful reaction. Superficial chilling of the skin may produce enough anesthesia for a superficial operation; i.e., skin grafting. For a deeper operation a tourniquet is indispensable. The length of time required for preparatory refrigeration is still not definite. Allen found that with thin, weak patients with arteriosclerosis, the most plausible time is as follows: for a thigh amputation, two and one-half hours; for disarticulation at the knee or through the middle of the leg, two hours; for the lower half of the leg or the foot, one and one-half hours; and for the metatarsus or the toes, one hour. If the tourniquet is properly applied and securely fastened and the chilling is continuous to all parts at the correct temperature, there is complete local anesthesia so that the patient is not aware when the nerve is cut or the bone is sawed. It is still not certain whether the tourniquet should be placed at the site of amputation and the refrigeration carried on several inches or a number of inches above the level of amputation.

Ice is applied to the flaps after the operation, and the lower temperature produced reduces the local metabolism so that a limited blood supply may be adequate for the need, and this diminishes the tendency to sloughing. It also aids by inhibiting pain, edema, and bacterial activity. The principle of refrigeration need not be confined to peripheral vascular lesions; it has been used in crushing wounds of various types. Arteriosclerotic legs have been packed in ice for periods up to one month without a tourniquet until the patient gained sufficient strength for an ultimate successful amputation. Prolonged refrigeration without a tourniquet has been used in extensive gangrene, with the result that advancing gangrene, infection, and systemic intoxication have been checked. It has been shown that gangrene of both arteriosclerosis and embolism can be delayed by reduced temperature.

Quite extensive studies have been made of the vascular tree of the legs which have been amputated for the diabetic gangrene. This was done by Henderson and Beardwood[44] by means of a mercurial injection of the arterial tree at what was the patient's mean pressure. As a control, the blood vessels of normal amputated legs, both of children and adults, were included in the study. The specimens were then x-rayed to obtain a picture of the vascular tree. Among other things it was clearly demonstrated that if a patient has had diabetes a long time before gangrene occurs, a rich collateral circulation develops, whereas if a patient has only had diabetes for a short period of time before the gangrene occurs, there is very little evidence of a collateral circulation. This finding has enabled us to be more conservative in the management of a gangrenous process of the digits in a known diabetic of many years' standing. On the other hand, the lack of a developed collateral circulation is in keeping with the poor response to local measures of a gangrenous process of the digits in a patient who has had diabetes only for a short time. In the last ten years, there has been an increasing number of cases in which successful conservative surgical procedures have been performed when the diabetes had been present for a number of years, and it is believed that the explanation can be found in these studies.

The newer physiological concepts of diabetes have pointed to the fact that many endocrine glands other than the pancreas may play an important role in its syndrome. While surgical attempts to help or cure this disease to date have proved unsatisfactory, when the physiological pathology of diabetes is completely understood it is not beyond the realm of hope that the surgeon may be able to relieve this metabolic disorder.

HYPERINSULINISM

In 1924 Harris[42] described an entity which he believed was due to an excessive secretion of insulin, and was characterized by periods of hypoglycemia. He called it hyperinsulinism, and reported his several cases in minute detail. Since his excellent contribution, considerable investigative work has been done upon the hypoglycemic state which occurs in hyperinsulinism. In 1927, Wilder and coworkers[83] reported a case of hyperinsulinism which was operated upon by W. J. Mayo. At operation he found a carcinoma of the islands of Langerhans with metastasis to the liver. Extensive studies were carried out upon this case, including the evaluation of the amount of insulin in the tumor. In metastatic nodules from the liver, they were able to isolate as much as 40 units of insulin from 100 Gm. of the tumor. In 1929, Howland, Campbell, Maltby and Robinson[48] reported a case in which Roscoe Graham successfully removed a benign islet adenoma, and obtained a cure of the hyperinsulinism. After a careful study of these 2 cases, the

problem of hyperinsulinism seemed to be solved. Unfortunately it is complicated by our knowledge of the effect of the liver, adrenals, anterior lobe of the pituitary, thyroid, sympathetic nervous system, and possibly other factors on sugar metabolism, particularly their effect upon the development of hypoglycemia. Some believe that other glands of internal secretion exert some regulating mechanism on the activity of islet tissues by means of their secretions. Recently we have learned that hyperthyroidism may be associated with some cases of hyperinsulinism, and a fatal thyroid crisis may develop as a complication to the removal of the tumor. Cases showing islet tumors and signs of hyperthyroidism were reported by Aiken[1] and Gilmore and Walton.[37] Whipple[82] saw 1 patient die from a postoperative thyroid storm, and 3 other postoperative crises have been reported. In the light of this experience it has been recommended that the basal metabolism rate should be determined in all islet tumor cases, and if it is at all elevated, the operation should be postponed until the patient has been given a course of Lugol's solution, such as we administer preoperatively to a patient suffering from toxic goiter. Hypoglycemia is quite common, but other glands of internal secretion may produce this condition because of some disturbance in the hormonal balance; these should be excluded as much as possible before the opinion is formulated that the hyperinsulinism originated in the pancreas alone.

Three types of anatomical changes in the islets have been reported in hypoglycemia. The first is generalized hypertrophy and hyperplasia; this type is commonly found in infancy, often in the offspring of a diabetic mother, and it responds to suitable dietary measures. The second type is an adenoma of the islands of Langerhans, composed of beta cells; and the third type is carcinoma of the islet cells. Our consideration will be limited to the last two types, as the accepted treatment of these lesions is surgical. Clinically we recognize two types of hyperinsulinism; the type due to an actual islet tumor, and the functional type in which there is little or no alteration of the structure of the islands of Langerhans.

The symptoms most commonly occurring in an attack of hyperinsulinism are: sweating, pallor, tremor, tachycardia, elevation of the blood pressure, fear, mild dyspnea, and severe abdominal pain. Neurological findings are frequently associated with these seizures, the most common being diplopia, dizziness, convulsions, and coma. As the disease progresses, it is not uncommon to find psychiatric manifestations such as emotional instability, disorientation, amnesia, and mania. Attacks of hypoglycemia, as a rule, occur four or more hours after the ingestion of food. They may occur sooner if the patient has undertaken strenuous exercise, thereby increasing metabolic activity and depleting the supply of sugar. Whipple,[82] who is one of the most ardent contributors in this field, has given us a characteristic triad of symptoms which he thinks are essential in the diagnosis of an islet adenoma. This triad of symptoms are: (1) attacks of hypoglycemia, however manifested, coming on during the fasting or overfatigued state; (2) associated blood-

sugar readings of 50 mgm. per 100 cc. or less during the attacks; and (3) relief of the shock promptly following the ingestion of glucose. Beardwood[8] believes that the glucose tolerance curve is characteristic and of considerable diagnostic value. He points out that it is important for the patient to be on a diet sufficiently high in carbohdyrates to avoid any influence that undernutrition might have on the curve, and he also emphasizes that the test should be conducted for six hours. Whipple,[82] however, believes that the fasting blood sugar after a twelve- to fifteen-hour fast is a far more reliable test of islet-cell function than is the glucose-tolerance curve. He believes the glucose-tolerance test to be a liver-function test, rather than a test of pancreatic function.

There has been a report[47] of 5 cases of extra-pancreatic islet adenoma producing symptoms of hyperinsulinism. We must realize this, when operation is undertaken, as a possibility in cases in which no tumor is found in the pancreas. In 1940 Faust and Mudgett[34] recorded the location of such aberrant tissue in 370 published cases. In 95 instances the pancreatic tissue was found in the stomach, in 105 in the duodenum, in 65 in the jejunum, in 18 in the ileum, and in 49 in diverticuli of the stomach, duodenum, or the small intestine. There are many other localities in which a very small number have been found. An extra-pancreatic islet adenoma of the duodenum has been reported with no symptoms of hyperinsulinism. As in other islet tumors, the treatment is surgical. Many contributors have repeatedly emphasized the necessity of complete exposure and palpation of the entire pancreas, including exposure of the head by mobilization of the duodenum. If such an exploration does not reveal a tumor, search should then be made for an adenoma in extrapancreatic tissue, particularly in the sites just mentioned where this tissue is most likely to occur. If no tumor is found, the case should then be regarded as one of the functional type, and a subtotal pancreatectomy performed. In the early functional cases, partial pancreatectomy was performed with rather discouraging results; however, in 1928 Finney and Finney[35] reported an excellent result following subtotal pancreatectomy, and in 1934 Graham and Hartman[38] reported an excellent result following a subtotal pancreatectomy performed upon an infant. Since these pioneers led the way to subtotal pancreatectomy, many others have reported similar cases, and the results from the more extensive operations have been much more satisfactory and lasting.[28]

In hyperinsulinism, the glycogen reserve is frequently used up, and it is well known that any surgical procedure is dangerous if the liver is deficient in glycogen. Guerry and McCutcheon[40] reported a fatality during operation in which there occurred what they termed an operative insulin crisis, even though they safeguarded themselves by having blood-sugar determinations made at the time of the induction of the anesthesia, again when the pancreas was exposed, and also after the resection was completed. In the light of this knowledge and experience, glucose should be administered freely for twenty-four hours before operation, and throughout the surgical procedure. In 1941

Whipple[82] compiled and analyzed 160 hypoglycemic cases that had either been reported in the literature or were reported to him by personal communication. A tumor was found in 136 of these 160 cases; in 107 patients the tumor was found at operation and in 29 it was found at necropsy. The pathological lesion was benign adenoma in 103, questionably malignant adenoma in 22, carcinoma with metastasis in 8, and hamartoma in 1; 2 cases were not differentiated. There were 16 postoperative deaths (15 per cent) among the 107 cases in which a tumor was found at operation. Eight additional deaths occurred after the patients left the hospital; 6 of these patients had carcinoma with metastasis. Bell and Best[10] warned against too extensive a pancreatectomy; they noted that if too much of the pancreas was removed, degenerative changes occurred in the remaining beta cells, and severe diabetes could result. This change is thought to be due to overactivity. This conception is supported by the fact that these degenerative changes do not occur if insulin is administered to the patient.

HYPERPARATHYROIDISM

The parathyroid glands were discovered by Sandstrom in 1880,[72] and in 1900 DeSanti[29] was the first to recognize a tumor of parathyroid origin. The association of parathyroid tumors with the clinical syndrome of von Recklinghausen's disease, generalized osteitis fibrosa, had long been recognized. In some respects this has been a most unfortunate association, as many individuals have generalized osteitis fibrosa cystica or localized cystic changes in bone but do not have hyperparathyroidism. It also focused the attention of most clinicians on the late stages of the disease.

Recently there have been some cases reported in which the patients, which were being treated for stones in the genitourinary tract, and after a careful study of the calcium and phosphorus metabolism were found to have hyperparathyroidism. This does not infer that we should suspect hyperparathyroidism in all cases of kidney stone; on the contrary, it is present in only a very small percentage of these cases. It is evidence, however, that we have become interested in the disease in its earlier stages. The spark which ignited this interest was the report of a cure of the disease following the surgical removal of an enlarged parathyroid gland by Mandel in 1926.[58] Prior to Mandel's case the literature contained scattered reports of parathyroid tumors found at autopsy.

Castleman and Mallory[19] and Albright and his co-workers[2] have made the most exhaustive pathological and clinical studies of the disease. They classified hyperparathyroidism into two types, primary and secondary. The primary types are divided into adenomas, the usual clinical type, and the diffuse type. They pointed out that in the last all parathyroid glands are enlarged. There is a cellular hypertrophy rather than hyperplasia and all the cells are of the Wasserhelle type. Albright[2] reported 6 cases of this type, upon which Churchill had performed a partial parathyroidectomy on all 4 parathyroid glands. This diffuse type of hyperparathyroidism was compared to the diffuse toxic goiter and hyperthyroidism. After these cases were observed for two years, it was apparent that adequate parathyroid tissue was removed, as the increased function of the parathyroid glands was relieved. Other findings, including the amount of tissue to be left, have been included in this study.

In 1905 MacCallum[57] called attention to the fact that renal disease may overshadow the skeletal changes in hyperparathyroidism, when he reported a tumor of the parathyroid gland associated with chronic renal disease. Since that time considerable evidence has been accumulated, indicating that long-standing renal insufficiency may give rise to parathyroid hyperplasia and hyperparathyroidism.[5] Experimentally, the effect of renal damage on the parathyroids was shown repeatedly. When the renal function is reduced the parathyroids increase in size, and correlated with this, bony lesions comparable to osteitis fibrosa cystica develop. Donahue, Spingarn, and Pappenheimer[30] have demonstrated experimentally that the parathyroid hyperfunction so induced may also in turn have its effect on the kidney. They found that the calcium content of the residual renal tissue was increased in proportion to the parathyroid enlargement. Early removal of the parathyroids prevented this increase in renal calcium. Virchow[79] in 1855 was the first to note metastatic calcification in cases of nephritis, and since that time the association of renal lesions and the deposition of calcium has been reported many times. There are numerous studies demonstrating the frequency with which parathyroid enlargement accompanies long-standing renal disease. To a lesser extent it has been found in rickets, osteomalacia, and pregnancy. Fowweather and Pyreh[36] studied the calcium and phosphorus in 102 cases of renal disease, and found the blood calcium to be over 11 mgm. per 100 cc. in about 50 per cent of the cases. This again points to the danger of attaching too much significance to a single elevation of the blood calcium. It has also been observed that the parathyroids are involved in renal dwarfism.

Langmead and Orr[54] reported a case of renal dwarfism in which the patient died at the age of twenty and in which the skeletal changes were consistent with hyperparathyroidism; four greatly enlarged parathyroids were found at necropsy. The evidence that hyperparathyroidism is an essential part of renal dwarfism appears overwhelming. Parke and Elliot[63] believe that instead of the term "renal rickets" in these cases, the disease should be called "renal hyperparathyroidism with osteitis fibrosa cystica." Elsom, Wood, and Ravdin[32] noted that the renal disease associated with primary hyperparathyroidism has certain clinical features which distinguish it from the usual types of chronic nephritis. Hypertension, cardiac enlargement, edema, and retinal exudates are usually absent or very inconspicuous. Albright and his associates[2] classify the renal lesions associated with hyperparathyroidism as (1) stone formation, (2) parenchymal calcium deposits and (3) parathyroid poisoning in which there are calcium deposits in the kidney, and, in addition, in other organs, such as the lung, stomach, and heart.

Briefly, the clinical findings are divided into three parts: (1) skeletal

changes, (2) findings in the genital tract and (3) a group of general symptoms commonly associated with chronic disease. In addition to these clinical findings we have a disturbance in the calcium and phosphorus metabolism. Typically the blood calcium is elevated, and the blood phosphorus is decreased, and both of these chemical elements are excreted in the urine in greater amounts than they are ingested. Before it is concluded that a patient has hyperparathyroidism we should not forget that many individuals may have at times an elevation in calcium or a fall in phosphorus which has no relation to hyperparathyroidism. A three-day calcium-balance study should be made before any deductions are drawn. By this procedure, we learn that the patient is in a negative calcium balance, and that the direction of flow of calcium in hyperparathyroidism is from the bones to the urine. The calcium-balance study is of paramount importance in early stages, as the clinical findings and metabolic disturbances just enumerated are not as apparent.

Since Mandel's successful cases in 1936, an increasing number of successful cases of parathyroidectomy have been reported in the literature. Unfortunately, parathyroidectomy has been used for lesions other than hyperparathyroidism, such as Paget's disease and hypertrophic arthritis. In these diseases normal parathyroid glands were removed without benefit, and I wish to emphasize again what has been stated before, that it is a very serious mistake to remove normal parathyroid glands, as we have no complete replacement and clinical experience shows that the procedure does not benefit the patient. In any given case of hyperparathyroidism it is important that a careful search be made until the tumor is found. Our surgical experience to date has shown that the parathyroid tumors need not necessarily be found close to the thyroid gland, but may be found in the anterior mediastinum, and as far back as behind the esophagus. It is not difficult to understand the various locations in which the tumors have been found if one studies the anlage of the parathyroid glands.[23] Churchill,[22] in his report of 30 cases of proved adenoma, mentioned 5 instances in which he found a tumor that had been overlooked at a previous operation. In 1 instance several previous operations had been performed.

Along with the complications which occur in any operation performed upon the neck, tetany is prone to occur after parathyroidectomy. This is in keeping with the insufficiencies observed in operations for hyperfunction of some of the other glands of internal secretion. Of the 30 cases reported by Churchill, 22 developed tetany postoperatively. The tetany was severe in 4, moderate in 3, and slight in the 15 others. When tetany does occur, large doses of calcium, parathormone, and A. T. 10 are the routine therapeutic procedure. The amount and method of administration of these therapeutic agents depend upon the degree of tetany present.

If advanced osteoporosis and cystic disease of the bones are present, many months may elapse before there is much redeposition of calcium in the affected areas. This is easily understood when we realize the small daily intake of calcium in the food. Calcium and vitamin D should be prescribed, and the patient should be encouraged to drink milk and eat cheese or other foods rich in calcium.

TUMORS OF THE ADRENAL GLAND

Tumors of the adrenal gland are difficult to diagnose accurately, and oftentimes are not suspected until discovered at the autopsy table. Although we have known of adrenal tumors since 1765, the first case successfully operated upon was reported by Collett[24] in 1924. There are many types of adrenal tumors, but our primary interest in this discussion will be focused upon the cortical and medullary tumors which produce metabolic changes. In 1927[76] extracts of the adrenal cortex were prepared which would prolong the life of adrenalectomized animals. This was of immeasurable value as it gave the surgeon a therapeutic agent which would combat the frequent and highly fatal postoperative complication of acute adrenal insufficiency. Since then, many observations and contributions have been made concerning the chemistry, physiology, and clinical diagnosis associated with these tumors.

TUMORS OF THE ADRENAL CORTEX

The symptoms of cortical tumors of the adrenal gland are in no way constant. They are dependent upon the age of the patient, the sex of the patient at the onset of the illness, the degree of endocrine activity of the tumor, and the duration of the disease. A study of the reported cases shows that the cortical tumors occur most frequently in young women, although they have occurred in young children. When the tumor develops in a child, precocious puberty usually occurs. In boys the precocious puberty is masculine in type, in girls it is heterologous. The most common findings indicating precocious puberty in girls are hypertrophy of the clitoris, deepening of the voice, masculine distribution of the hair, and occasionally precocious menstruation. One case has been reported[80] in which a child of two had two or three menstrual periods. When a cortical tumor occurs in an adult, virilism develops in the female, and feminism in the male. The most frequent evidences of virilism in the female are growth of hair in the masculine distribution, the growth of a beard and mustache, atrophy of the breasts, masculine habitus, cessation of the menses, atrophy of the reproductive organs, and hypertrophy of the clitoris. In the male the most common evidences of feminism are atrophy of the penis and testes, and enlargement of the breasts.

Clinically, in addition to these changes in sex characteristics, the patients frequently develop the symptoms and clinical findings associated with Cushing's syndrome of basophilic adenoma; namely, obesity or a change in the distribution of the fat, hypertension, osteoporosis, purple striations of the skin, acne, polycythemia, frank diabetes or changes in the carbohydrate metabolism, and fatigue. If the tumor is large enough, local symptoms may develop which are characteristic of any other abdominal tumor of the same location. Any of these symptoms just enumerated may or may not be present

in a given case. Any or all have been absent, and almost every combination of them have been found in individual cases. Closely related both to a tumor of the adrenal cortex and to Cushing's disease is a condition which frequently has been referred to as adrenocortical "hyperplasia." This has been found at autopsy in an appreciable number of proved cases of Cushing's disease, and in another group of cases, in which the clinical picture is compatible with the diagnosis either of adrenocortical tumor or of basophilic adenoma of the pituitary body. Cushing's syndrome may be associated with tumors of the thymus, enlargement of the parathyroid glands, and arrhenoblastoma of the ovary, in addition to basophilic adenoma and adrenocortical tumor. It is commonly believed that the hyperfunctioning basophilic tumor of the pituitary gland may stimulate overactivity of the adrenal cortex. There is some experimental work to indicate that such might be the case. Evans[50] and Collip and his associates[26] have independently presented evidence indicating the existence of adrenotropic hormones. Smith[75] also demonstrated that removals of the pituitary gland greatly decreased the size of the adrenal glands and was associated with atrophy and vascular degeneration of the cortex, and that the administration of an extract of the anterior lobe of the pituitary gland produced marked hypertrophy of the cortex. Evans concluded that the adrenocortical tissue needs for its normal function some constituent of the anterior lobe of the pituitary gland. Cecil[20] reviewed the literature and noted that in 30 per cent of the reported cases of cortical tumor, the adrenal gland opposite to the one occupied by the tumor was absent or inadequate to support life. Ingle and Kendall[49] supported Cecil's findings by noting that the administration of large amounts of cortin produced atrophy of the adrenal cortex of the rat, but if the adrenotropic hormone was administered with cortin, the degree of atrophy was but slight.

Minor disturbances in the carbohydrate metabolism are found frequently in patients with a tumor of the adrenal cortex. However, in 1939 Shepardson and Shapiro,[74] in a careful search of the literature, could find only 17 proved cases of adrenocortical tumor associated with diabetes, and added 1 of their own. Lukens, Flippin, and Thigpen[56] reviewed the status of the carbohydrate metabolism in 44 cases of cortical tumors and 11 cases of bilateral adrenocortical hyperplasia reported from 1917 to 1935. There were no disturbances in the carbohydrate metabolism in 24 cases of tumor and in 4 cases of hyperplasia; but there were transitory glycosuria in 4 cases of tumor and 1 case of hyperplasia, and marked glycosuria in 13 cases of tumor and 6 cases of hyperplasia. On the basis of the clinical and experimental evidence, there is considerable doubt that the adrenal cortex is responsible for this carbohydrate imbalance.

Many believe that the greatest recent advance in the diagnosis of hyperfunctioning lesions of the adrenal cortex has been the clinical application of assays of the urine for hormonal-like substances. Cahill[16] classifies cortical tumors on the basis of the presence or absence of these hormonelike substances. He has observed that tumors with no recognizable hormonal changes

occur infrequently, usually in patients over thirty years of age, and in either sex. As a rule these tumors have been present for a long time without symptoms, and the prognosis is very poor as they are not diagnosed until late. The tumor is suspected and located very frequently because the patient complains of local symptoms which are due to the tumor, such as abdominal pain or pain in the flank of the affected side which is made worse by exercise.

Perhaps a contribution of greater value in the diagnosis of adrenocortical tumors is the quantitative determinations of neutral 17 ketosteroid in urinary assays. Extensive investigation in this field has been carried out by Callow[17] and Talbot[77] and their associates. Only a few months ago, Talbot concluded that the urinary 17-ketosteroid measurement has earned a place among those clinical laboratory procedures which provide diagnostic information. Determination of the output of the 17 ketosteroid is helpful in detecting both hyperfunction and hypofunction of the adrenal cortex, and hypofunction of the thyroid and pituitary glands. The output is markedly lowered in diminished function of the adrenal, thyroid, and pituitary glands; there is marked increase of excretion in hyperfunction of the adrenal cortex, but increased function of the thyroid or pituitary does not cause a significant elevation in excretion.

It has been shown that the 17 ketosteroid found in the urine is derived from the adrenal cortex and the gonads. This is not difficult to understand when we realize that both of these tissues have their anlage in the genital ridge. Further studies showed that the normal value of the 17 ketosteroid drops one-third in castrated subjects. From this we can deduce that two-thirds of the 17 ketosteroid comes from the adrenal cortex, and one-third from the gonads. The normal amount of the 17 ketosteroid found in the twenty-four-hour specimen of urine depends upon the age of the individual. The normal amount for a child up to ten years of age is 1 mgm. At puberty the amount varies with the sex of the individual: in the male from 10 to 20 mgm. is normal, whereas in the female the normal is from 9 to 15 mgm. If the determination of the 17 ketosteroid is 30 mgm. or over, it is almost certain to be due to an adrenocortical carcinoma. This was first observed by Callow and has been confirmed by Hoffman and many others. If the 17 ketosteroid is increased, although below 30, it is impossible to say whether it is due to simple diffuse hyperplasia or a nodular hyperplasia of the adrenal gland. If there is evidence of associated hyperpituitarism, the likelihood is that it is a hyperplasia. In the presence of a feminizing tumor of the male adrenal cortex there is no increase in the 17 ketosteroids, but estrogenic substances are increased. Based on the significance of the 17-ketosteroid determinations, the following functional classification (Table 2) was developed by Hoffman[46] while working in the laboratories of Custer.

As an aid to diagnosis, Carrelli[18] in 1901 demonstrated that carbon dioxide infiltrated into the perirenal fascia would be of value in the visualization of the kidney capsule and the adrenal gland by x-rays. Oftentimes, because of the rapid diffusion of the carbon dioxide, it was difficult to demonstrate the out-

TABLE 2. DYSCRASIAS OF THE ADRENAL CORTEX FUNCTIONAL CLASSIFICATIONS

	NEUTRAL 17 KETOSTEROIDS
1. Hypercorticalism	
Primary	
a. Adrenocortical carcinoma	++++
b. Diffuse hyperplasia	+++
c. Nodular	++
d. Feminizing tumor of the adrenal cortex (6 cases); estrogen increased	o
Secondary	
a. Hyperplasia secondary to hyperpituitarism, i.e., certain types of Cushing's syndrome	++
b. Secondary adrenocortical carcinoma associated with Cushing's syndrome	+++—++++
2. Aberrant corticalism (steroids of unusual types)	
3. Hypocorticalism	
a. Adenoma	(——) to (o)
b. Hypoplasia	—
4. Tumors of nonglandular epithelial origin	

line of the adrenal gland. In 1935 Cahill[15] reported the use of a measured amount of air injected directly into the perirenal space by hand pressure. He took roentgenograms shortly after completion of the injection, and at twelve, eighteen, twenty-four, and thirty-six hours after the injection, and found the organs and fascial planes to stand out very clearly, especially, as desired, around the adrenal gland. Accidents have occurred in the use of this diagnostic procedure, and this danger must be considered before an air injection is undertaken. Recently, Pendergrass[64] has reverted to Carrelli's original idea of the use of carbon dioxide in obtaining the roentgenogram of the adrenal glands. This gas is less dangerous, is absorbed rapidly should it be introduced into the blood stream, and, therefore, eliminates the dangers of air embolism and its sequelae. Intravenous urography should be included in the routine studies. If a tumor is present it demonstrates that the kidney is pushed down on the affected side.

All agree that the proper treatment of an adrenocortical tumor is surgical. It has been suggested that the adrenal gland should be explored in all cases of Cushing's syndrome lest we miss an opportunity to operate upon a cortical tumor early. Several surgical approaches have been advocated, the transthoracic, the posterior kidney incision, and an abdominal incision. The last two are used by the vast majority of surgeons. It seems to me that when we carefully consider all the factors, the abdominal approach is the most desirable. It has most of the advantages that the kidney incision affords the surgeon, and in addition both adrenal glands may be palpated through this incision before the tumor is removed. This, I think, is of great importance especially since Cecil found the opposite adrenal gland to be absent or inadequate to support life in 30 per cent of the reported cases. The condition of both adrenal glands should be noted before removal of the tumor, so that we may anticipate the development of postoperative adrenocortical insufficiency. Also, by the abdominal approach the pelvic organs may be explored in the female to determine whether or not an arrhenoblastoma of the ovary, or, more rarely, a granulosa tumor is present. The operative mortality in cortical tumors is quite high, practically 50 per cent in the reported cases. Lukens et al.[56] collected from the literature 40 cases of this type of tumor which had been operated upon, 19 of which terminated fatally. The most common complication following operation is adrenocortical insufficiency. This was responsible for the almost prohibitive mortality; however, recently a routine has been developed for the management of this complication which has proved very satisfactory, and has permitted us to carry the patient over the period of acute insufficiency until the remaining adrenal tissue could undergo sufficient hyperplasia to compensate for the loss of the other adrenal gland and produce adequate adrenal secretion to maintain life. The treatment of this complication is the same as is used when it occurs in Addison's disease; namely, the administration of adrenocortical extract, the sodium ion in the form of sodium chloride and sodium citrate, adequate doses of vitamin C to fix the salt, and a diet low in potassium. Walters[80] believes that we should anticipate postoperative adrenal insufficiency, and institute this treatment throughout the postoperative period. The patient should be watched carefully for any premonitory signs of acute adrenal failure. The more important symptoms of this complication are nausea, vomiting, weakness, a rapid weak pulse, and falling blood pressure. More recently, with the exception of the administration of the adrenocortical extract, the same regime has been used for a few days before operation. In reporting 8 cases operated upon for cortical tumor, Walters states that he had 7 consecutive successful results, the 1 fatality having occurred in 1924 before we knew how to control adrenal insufficiency adequately.

MEDULLARY ADRENAL-GLAND TUMORS

Medullary tumors of the adrenal gland have been observed for many years, but the surgical removal of these lesions is a comparatively recent adventure. In 1927 C. H. Mayo[59] reported the first successful removal of such a tumor. He called attention to the fact that the patient developed severe adrenal insufficiency in the course of the operation, and heroic measures, large doses of adrenalin, were necessary to keep the patient from dying on the operating table. This experience is a common one to surgeons operating upon patients with medullary tumors of the adrenal gland.

There are three pathological types of medullary tumors according to the type of cell from which they are derived: (1) sympathoblastomas from embryonic cells, (2) ganglioneuromas from mature ganglion cells and (3) pheochromocytomas, or paragangliomas, from mature chromaffin cells. It is

the last type in which we are interested as this is the only type which, it is thought, can produce epinephrine.

Clinically, the patient suffering from a pheochromocytoma is subject to paroxysms of hypertension at various intervals. The longer the patient's history, the briefer is the interval between paroxysms, and in some of the oldest cases the hypertension has become constant. The most common symptoms associated with these attacks of paroxysmal hypertension are pectoral pain, tachycardia, pallor and flushing, the sensation of cold followed by heat, profuse sweating, and nausea and vomiting. The majority, but not necessarily all of the symptoms occur in all cases. In the past few years, some cases have been reported in which patients suffering from pheochromocytomas have also presented the clinical picture of hyperthyroidism. McCullagh and Engle[60] gave a very complete report of two such cases, and in 1 of their cases a thyroidectomy was performed with no relief, and a subsequent removal of the medullary tumor resulted in complete abatement of the symptoms, including those of hyperthyroidism. The basal metabolic rate, which had been moderately elevated, was unaffected by the thyroidectomy and returned to normal following the removal of the medullary tumor. These cases again illustrated how confusing the symptomatology may be in the presence of increased function of the adrenal and thyroid glands. Cases have been reported in which massage over the tumor in the course of the physical examination produced a sharp rise in the blood pressure. Crane[27] reported a case in which this clinical finding was the most important factor in enabling him to diagnose the presence of a medullary tumor. While this may be very valuable in assisting one in arriving at the diagnosis, palpation of these tumors is not without danger, and sudden deaths have been reported following too vigorous palpation of a pheochromocytoma.

The diagnosis of medullary tumors is quite difficult. As a rule the patients have to be observed and studied in their paroxysms of hypertension before this lesion is considered. If the disease has progressed until the hypertension is constant, the problem becomes all the more difficult. As in the cortical tumor, the perirenal injection of air is of diagnostic value; however, it must be realized that at times these tumors are very small, and if the clinical picture leads one to suspect such a tumor, failure to find any enlargement of the adrenal gland as outlined by the injected air is no contraindication to surgical exploration.

Experience with these tumors is very limited; in 1940 Brunschwig and Humphreys[14] were able to find only 103 cases in the literature, most of which were found at autopsy. The treatment of medullary tumors is surgical. It is extremely hazardous to operate upon these patients, and death during the course of the operation is not infrequent from acute vasomotor collapse associated with hyperactivity of the tumor. Brunschwig and Humphreys pointed out the frequency with which this complication occurs in the clinical course of the patient and emphasized the importance of prompt diagnosis and treatment lest the patient die of a fatal attack. Not infrequently patients

with medullary tumors have succumbed to minor surgical procedures performed elsewhere in the body. Here again the abdominal approach is thought to be the best, as the vascular pedicle to the tumor may be clamped and tied before the gland is manipulated. This prevents the squeezing of excessive quantities of adrenalin into the circulation. In 1941 Biskand, Meyer, and Beadner[11] were able to find 29 reported cases which had been operated upon. They noted that the lesion was more common in females than in males, was more frequent on the right side than on the left, and was extrarenal in 4 instances. Eighteen patients developed acute adrenal insufficiency and 4 died immediately after the operation. Fifteen patients were followed up; 12 were symptom-free, 2 were alive with slight symptoms and blood-pressure variations, and 1 had died. Fortunately this lesion is rare, as the clinical outlook is very discouraging.

SUMMARY

With increased experience in the field of metabolic diseases, the multiplicity of glandular involvement in patients presenting metabolic problems has become more apparent as the complexity of these syndromes unfolds. Great progress has been made in establishing therapeutic measures aimed to prevent, as well as to treat successfully the insufficiencies which are so prone to develop in operations performed upon the glands of internal secretion. There are many phases of these diseases with which the surgeon could not hope to familiarize himself to the degree that the metabolist or the clinical pathologist does. This being the case, all patients who have surgical lesions with metabolic changes require the closest supervision and study by the internist and surgeon. One need only look at the progress made in surgery of the diabetic by this combined management to appreciate its full significance. In some fields, such as surgery in hyperinsulinism and hyperparathyroidism, we have a great deal to learn in diagnosis as well as in treatment. There are those who feel that there are unlimited possibilities in the future of surgery upon the adrenal gland. Many intricate diagnostic problems and surgical complications which occur in surgery in metabolic diseases are entirely foreign to other types of general surgery. Today many institutions have a department of metabolic disease in the medical division, and should have a similar department in the surgical division. By the close co-operation of these two departments, we would be able to diagnose and treat more satisfactorily, patients suffering from metabolic diseases which require surgical treatment, and thereby improve our operative results, and lower our operative mortality.

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ANNUAL ADDRESS FOR 1944

GRADUATE SURGICAL TRAINING IN AMERICA*

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For the selection of a philosophical rather than a clinical topic for this address, I make no apologies. In recent years on this occasion we have had so many excellent expositions of clinical subjects that it would be difficult to select one to warrant the imposition upon the time of those courteous enough to listen. The matter of graduate training or graduate education in surgery is one which has engaged the interest of the speaker for many years. In the light of present conditions it should properly engage the interest of all of us. I propose, therefore, to discuss this subject, particularly in relation to American surgery. It follows, that in order to do this intelligently one must first review the past, consider the present and venture a few suggestions regarding the future.

The beginning of the twentieth century saw surgery in America in the hands of "doctors who operated." These men were mostly physicians who, through interest and manual dexterity, were recognized by the public and their fellow practitioners as surgeons. For the most part, these men continued to engage in the general practice of medicine, and it is doubtful whether anyone really confined his work to surgery. It was the usual thing for the so-called surgeon to attend medical cases, deliver babies and to treat diseases of the eye, ear, nose, throat and skin. In general, the surgeon was merely a superior breed of doctor. He acquired proficiency either by long hours spent in the dissecting room or the dead house. In the former he learned anatomy, and in the latter became familiar with the gross appearance of diseased organs and tissues. This knowledge he applied to his practice. Many of the pioneers in American surgery followed this path to glory. In our own city the elder Gross and Agnew may be cited as examples. Later the period of "study abroad" appeared on the horizon, and of this probably the least said the better. Suffice it to say that the recent graduate, or the practitioner of better than average means took a year off and went to Vienna where he acquired or improved a taste for beer, sent postcards to the folks at home, and eventually returned to become a specialist, by announcement. In this country a number of short, or so-called polyclinic courses were given in various medical centers. To these came doctors for surcease from the rigors of practice and a little fun in the big cities. Neither "study abroad" nor the polyclinic course had any significant influence on the progress of surgery in this country.

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The American College of Surgeons, founded in 1913, represented the first attempt to set up a standard by which true specialization in surgery might be recognized. By this time there was a not inconsiderable number of surgeons who, through long apprenticeship and clinical experience, had attained the status of the specialist. In its earlier days the College attempted to formulate requirements for recognition of surgeons. Later it extended its activities to provide the facilities by means of which these requirements might be met. One need only consider the size of the country where surgery was being done in communities varying in population from the metropolitan city to the village to realize the enormity of the task undertaken by this organization. Those who have been inclined to criticize and belittle the work of the College should bear these facts in mind. That a uniform standard, applicable alike to the metropolis and the village, was impossible of achievement should not be allowed to negate the splendid contributions of the College to the cause of American surgery. It follows as inevitable that some unworthy ones were admitted to Fellowship.

Requirements for admission to Fellowship in the College have been gradually raised over a period of years. The present requirements are three years of hospital service, two of which must be spent in a surgical service acceptable to the College. In addition, it is stated that the candidate must restrict himself to "study, diagnosis and operative work in surgery," but in the same section the statement appears that "the minimum participation of specialization may vary according to the character and size of the community in which the applicant resides." Furthermore, it is stated that each application shall be judged upon its individual merits.

In 1915, the first formal Fellowships in general surgery were instituted at the Mayo Clinic, and to date approximately 825 men have completed this training. In 1916, the Graduate School of Medicine of the University of Pennsylvania was organized and offered the first full-time academic course in surgery, which included work in the basic sciences and led to a higher degree. The residency system, originated at the Johns Hopkins Hospital by Osler probably constitutes America's outstanding contribution to training in surgery. To date 41 men have completed the chief residency in surgery at this institution. Of the residency system, more later.

In 1916 the first Specialty Board was constituted in Ophthalmology. The organization of other similar constituted Boards for other specialties followed. The American Board of Surgery was constituted in 1937, and held its first examination the following year. To date 1279 have been certified by examination (1277 men and 2 women). To qualify for examination before this Board the candidate must present satisfactory evidence of at least five years of training following completion of a minimum internship of one year. This five-year period must be devoted exclusively to training and must include work in the basic sciences of Anatomy, Physiology, Pathology, Chemistry and Bacteriology. Three of the five years must be devoted to general surgery; not more than six months' credit is allowed for work in any one of the

previously mentioned basic sciences, or to such specialties as Gynecology, Urology, Orthopedics, *etc.* Recognizing the value of a sound basis of general diagnosis and internal medicine, the Board has accepted a full year of residency in medicine for credit toward the five years' training. In addition, evidence of adequate operative experience is required. The examination itself is comprehensive and rather difficult. From the foregoing it will be obvious that many men who can qualify for Fellowship in the College are unable to qualify for certification by the Board. The most frequent cause of rejection of applicants is inadequate training; failure to limit to surgery ranks second. A limited number of applicants are rejected on ethical grounds; for example, the practice of division of fees.

This brief summary of the past brings us to a consideration of the present status of graduate education or training in surgery.

Today the recent graduate who desires to become a surgeon has several paths open to him. Regardless of the plan the trainee elects, the objectives are the same. He must review the basic sciences with reference to their application to surgery. He will have forgotten much of his anatomy if he graduated from a school in which anatomy is still taught. If he comes from any one of the majority of the leading schools where anatomy in recent years has been compressed into periods of from three to six months, he will not have forgotten it for the simple reason that he has never learned it. With regard to physiology, likewise, the average graduate will recall that most of his undergraduate hours in this important subject were devoted to the making of nerve muscle preparations and recording the responses of the same on a smoked drum. As to the more important matters of the physiology of nutrition, of liver function, *etc.*, he will have at best a very hazy conception. His training in pathology will be somewhat better, but it will probably be some time since he has looked at a slide under the microscope. The failure to correlate these basic sciences with clinical medicine and surgery is largely due to the fact that these departments are for the most part headed by Ph.D.'s who regard the instruction of undergraduate medical students as a penance which must be fulfilled in order to enjoy the facilities for research in pure science. To put all this into the language of the street: "The tail wags the dog."

In addition to building a foundation upon the basic sciences, the candidate must have opportunity to adequately study surgical patients before operation in order to foresee what may happen to them after operation, and provide protection against such eventualities. Above all, he must have opportunity and time to read and to think, and to discuss his problems day by day as they arise with an older, more experienced and understanding preceptor.

The Apprentice System. This plan, under which most of the senior Fellows of this Academy were brought up, still has a definite place in the education and training of the surgeon, although in recent years it has become fashionable to refer to the apprentice system with something between reluctant tolerance and ill disguised contempt. For many an aspirant to surgical training, however, it offers the only opportunity. Under this system the tyro associates himself with an established surgeon, assists him at the operating table, takes charge of the pre- and postoperative care of patients, is gradually eased into greater responsibilities and, finally, is permitted to operate independently. During this apprenticeship, unless his chief pays him a salary, which is uncommon, the trainee is hard pressed to earn a living, and in spite of good intentions too often finds it necessary to engage in general practice "on the side." Unfortunately, the general practice often takes precedence over surgical training. This does not infer that by means of the surgical apprenticeship combined with general practice he may not become a competent operator, but he rarely is able to give sufficient time to surgery to become a well-rounded surgeon. Training of this type, in which the trainee conducts a general practice while acquiring surgical experience, is not acceptable for credit by the American Board of Surgery, and is responsible for the rejection of a considerable number of applicants for examination by that Board. Many of these men may qualify for Fellowship in the American College of Surgeons.

The Residency. Previous reference to this system has been made. As stated, it had its beginnings at Johns Hopkins Hospital. As originally set-up in that institution a young physician spent an indefinite number of years living in the hospital in order to complete his training. With the Hopkins' plan as a working model, the residency system has been extended to practically all teaching hospitals and to many not connected directly with teaching institutions. The properly set-up residency is probably the best system by which a young physician may receive adequate training in surgery. The widest differences, however, exist in these residencies. The council on Medical Education and Hospitals of the American Medical Association, and the American College of Surgeons, maintain services which inspect and approve institutions for such residencies. In their most recent publications the former has approved 275 and the latter 162. Each has set-up standards for approval. On paper these are excellent. In actuality, some of the approved residencies are nothing more than internships in which the trainee is shockingly exploited and spends his time in routine drudgery, failing completely to receive real instruction, and has neither opportunity nor time to think—both of which are essential. It is inevitable that the best residencies are to be found in teaching hospitals. It by no means follows that good residencies are limited to such institutions. It is not necessary that a residency be taken in a teaching hospital, but it is essential that the director of the residency be a teacher. One of the greatest difficulties in the nonteaching hospital is the meeting of the requirement pertaining to instruction in the basic sciences. Both of the approving bodies previously referred to require such facilities, as does also the American Board of Surgery. Provision for instruction in Anatomy, Pathology, Bacteriology and Surgical Physiology is essential. In a hospital connected with a medical school this is a simple matter. In the well conducted nonteaching hospital adequate instruction may usually be provided

in Pathology and Bacteriology. Anatomy and Physiology present difficulties, but not necessarily insurmountable ones. In New York City, for example, certain hospitals have negotiated an agreement with Columbia University whereby a resident desiring to meet the basic science requirements of a Specialty Board may obtain such instruction without cost, other than the payment of a nominal matriculation fee. This plan should be extended to other teaching centers throughout the country. It is already being applied, in part, in two other localities.

Formal full-time graduate instruction in a University School offers a solution to some of the problems which have proved difficult in the apprenticeship and the residency. The University of Pennsylvania was the first to offer such instruction. Organized in 1916, the first group of student physicians was received in 1919. Under this plan a full academic year is devoted to a comprehensive review of the basic sciences of Anatomy, Physiology, Pathology and Chemistry, together with systematic courses in operative surgery on the cadaver and on living animals. In the latter, the major effort is concentrated upon the mastery of fundamental surgical principles, such as prevention and management of shock, control of hemorrhage, prevention of infection, wound healing, *etc.*, rather than upon cutting and sewing—things which, after all, are the easiest that the surgeon is called upon to master. In conjunction with the foregoing, the student physician is afforded ample opportunity to observe the conduct of operations in the many clinics of Philadelphia, and in the University Hospitals, to maintain close contact with the pre- and postoperative care of patients.

From the beginning, the Graduate School has emphasized the fact that it did not attempt to make a surgeon in one academic year, but it has maintained that with such an academic year behind him, the student physician is placed in a position to become a surgeon through additional clinical training. Those of us who, for many years, have participated in this program of graduate education feel that it has a definite place in the making of surgeons and offers a solution to the conspicuous shortcomings of the purely apprentice type of training. That this feeling is shared by many is evidenced by a steadily increasing number of well qualified applicants each year. The Graduate School encourages the continuation of training by placing those who complete the basic year in acceptable residencies. After the satisfactory completion of two years in such a residency the trainee may, upon presentation of an approved thesis, be awarded the degree of Master of Science in Surgery, or upon the completion of an additional two years, that of Doctor of Science. The American Board of Surgery gives full credit for time spent in this program.

The Graduate School should take a more active part in securing clinical opportunities for those who complete the basic year than it has heretofore. Failure to do this constitutes one of the definite shortcomings which must be remedied.

These three plans—the apprenticeship, the residency and the graduate

school—are the paths by which the surgical neophyte may arrive. Most men undertaking training under any of these have as their objective certification by the American Board. In the light of experience acquired through participation in the examinations of this Board, and in the evaluation of candidates for examination, certain conclusions regarding the merits and shortcomings of the three may be drawn. For the apprenticeship plan, on the credit side one finds that it offers probably the maximum of actual operative experience and tends to the development of the ability to think and act quickly. It also offers greater opportunity for a young man to acquire a surgical practice and, hence, financial security at an earlier date. On the debit side, it is unquestionably true that the surgeon trained today under the apprenticeship plan is likely to know much more of the "how" than of the "why" of what he does. If apprenticed to a typical "cutter and sewer" he may see and not perceive, and may accumulate experience which consists, for the most part, of the repetition of mistakes. These men are prone to the early development of cerebral rigidity, characterized by resistance to progress or change, and an attitude of intolerance and criticism toward those whose horizons are not fixed.

The residency, if it be well organized, is without doubt the best form of training. The residency, unlike the apprenticeship is an educational discipline. The resident advances gradually along a well defined path. He is given instruction in the basic sciences as applied to surgery. He is encouraged, or rather compelled, to use his powers of observation in the study of his patients. He must stand up and be counted when it comes to errors in diagnosis, technic or judgment. In the final phase of his training he is given an opportunity to operate independently. He is encouraged to think his problems through and is given constant help and encouragement in their solution. Above all, if the residency is under the direction of a proper preceptor, the candidate cannot but profit through precept and example of the one who stands to him *in loco parentis chirurgicae.*

The residency is, however, not without its shortcomings. The question of the optimum length of time which should be so spent has not been answered to the satisfaction of everyone. The original residencies at the Johns Hopkins were of indeterminate duration, and this, in itself, is not altogether desirable. In time the resident who stays too long develops a cloister complex. He becomes so accustomed to having immediately at hand everything to work with, and someone to whom he may always appeal for help in extremity, that he inevitably loses some of the self-reliance which is part of the essential armamentarium of every surgeon who is confronted with surgical actualities. He may remain so long in his institution that his value to the surgically sick public may be permanently impaired. Again, these long term residents sometimes become sidetracked through concentration on some restricted phase of surgery. This is not by way of criticism of the magnificent work that has been done by many such individuals, but it is, nevertheless, a fact that a vast amount of effort, primarily intended to qualify a man to take care of surgical

patients has been wasted. For example, it is completely unnecessary to spend five years in a surgical residency in order to make contributions to the literature of the bacteriology of surgical infections. Such a man has not only wasted his own time but has also prevented another, possibly equally or even superiorly qualified, from becoming a surgeon. In the examination for certification of many men, the product of the protracted residency, the writer has been impressed with the shocking lack of knowledge of certain matters with which all clinical surgeons should be familiar.

Fellowship in the great clinics, of which there are a number in this country, offers certain things which the residency of indeterminate duration lacks. A more realistic attitude toward the practice of surgery is cultivated and the necessity of getting things done is impressed upon the trainee. He acquires a lively sense of responsibility with regard to results. He probably has a better opportunity to become familiar with the correlation of general surgery with the surgical specialties and internal medicine. He definitely does not have the opportunity to operate independently, nor in some instances even to act as first assistant at the operating table. This form of training is an excellent educational discipline, but before the trainee may take his place and receive the stamp of approval as one qualified to undertake unsupervised the complete responsibilities of the surgeon, he must supplement his Fellowship with some sort of apprenticeship or precepteeship. Here, as in the product of the protracted residency, there is a tendency toward intolerance of his less fortunate brethren of the common herd.

The student physician who completes the basic year in a Graduate School must supplement this year by further clinical training if he is to qualify before his examining board. There are, however, each year some who take the basic course in order to fill some hiatus in their training. Many of these have had considerable clinical experience, but feel that they lack knowledge of the basic sciences as applied to surgery. Only occasionally in recent years have those come who desired refresher courses and the policy of our own institution in normal times has been to discourage them. The Graduate School, in spite of all the criticism leveled at it, has served a useful purpose in the past and will continue to do so in the future. It offers to a significant group of qualified student physicians facilities for formal instruction which cannot be obtained elsewhere.

Having considered the past and the present in graduate training in surgery, we now come to the future.

Any discussion of the future must deal with two separate and distinct phases of the problem. First, the immediate post-war period and, finally, the long range aspect. The induction into the armed services of practically all able-bodied physicians of trainee age brought graduate education in the usual sense to an abrupt halt. Those who had partially completed their training were forced to interrupt it. Those who might, in the normal course of events, have begun training were forced to postpone it. Thus, the annual supply of trained young surgeons ceased. A whole medical generation may be

conceivably required to remedy the situation thus created. The so-called residency of nine months, which the War Manpower Commission has been graciously pleased to permit, is probably worse than nothing, since it will create a group of inadequately trained men who will consider themselves as superior to other less fortunate contemporaries who were placed on active duty at the end of a nine months' internship. Of these, it may be truly said that a little knowledge is a dangerous thing. The product of the nine months' residency will have no significant effect on the general problem of developing surgeons in this country. It is a stop-gap to permit the more efficient care of patients—nothing more or less.

In the period immediately following the cessation of hostilities our first consideration should be for those whose formal training was interrupted by entry into the services. To them should be given the opportunity of completing their training should they desire to do so. Replies to questionnaires sent to physicians in the armed forces indicate that a majority of medical officers desire some sort of graduate education upon discharge from the services. The "G. I. Bill of Rights" will make this available to all who desire it. We must, therefore, prepare ourselves to provide proper facilities for it. If in any way possible those whose training was interrupted should be allowed and encouraged to return to the clinics from which they came. The problem of evaluating their military service for credit before the Boards is a real one. In all fairness, it must be admitted that much of the experience incident to service will be of little permanent value. The surgery of war is, at best, rough and ready surgery and is largely confined to the surgery of trauma. Even in this restricted field the young surgeon has scant opportunity of following his patients or of knowing what eventually becomes of them. He, therefore, does not learn to know what to expect from what he does. In other words, there is but little chance to acquire surgical judgment. However worthy the service or patriotic the motive, military service inevitably will occupy a minor place in surgical education. The American Board of Surgery, after careful consideration of every aspect of this problem, has allowed one year of training credit for the first year of service with the armed forces, regardless of how that year is spent. This is a gesture recognizing the sacrifice that the young doctor has made and not an admission that a year in the Army or Navy is the equivalent of a year of training under normal conditions. Beyond this first year credit will depend upon the actual type of service engaged in. If a candidate is assigned to the surgical service of a regularly constituted military hospital, credit may be allowed upon the submission of an acceptable record of service. The Board has not lowered or modified in any way the other requirements for admission to examination, nor has there been any change in the character of the examination itself. To do either would be manifestly unfair to those who have qualified in the past and who will qualify in the future.

It is quite possible that the American College of Surgeons may change its method of admission to Fellowship. A special committee appointed to

make a study of the subject has already submitted a preliminary report. The following citations from this report are here given by permission of the chairman of the committee, Dr. Frederick Coller. The question has been raised: "Could not the American College of Surgeons conduct an examination in the basic principles of surgery, replacing Part I of the examination now given by many boards?" This would, in addition, serve as an initial test for those aspiring to Fellowship in the College. Following this examination, and after further training, those who passed the initial examination would later take the examinations given by the various Boards of Surgical Specialties. After having passed his particular Board, the candidate would become eligible for Fellowship in the College. This plan would take care of all men intending to limit their work, but it would not take care of a large group of men practicing in small towns and who, while doing good surgery, either are unable or unwilling to confine their practice to surgery. The report offers the suggestion that the character of Junior Fellowship might be changed. This might be reserved for those who have passed the initial examination, but subsequently failed to limit their practice. Since the title of Junior Fellow would be for a limited number of years, and be continued only during good behavior, this group would be encouraged to secure further training and to otherwise meet the requirements for certification by a Board and for Senior Fellowship in the American College of Surgeons. Such a plan would bridge the years between the present and the time when enough men with adequate training will be available to every community.

For those who had come within one year of completing their training, the problem is simple. For those who have had less, it will be difficult in direct proportion. For those who would have commenced their training but for the war, it will be most difficult of all. Many of this latter group will be, for one reason or another, permanently lost to surgery. Regardless of the subdivision into which they may fall, each and every one of these men deserves well of his profession, as he deserves well of his country, and it is the obligation of all of us who are concerned to see to it that he is not let down. This will be no simple obligation to fulfill. With each year of the war the number of men for whom training facilities must be provided after the war will increase. Even before the war there were not enough places where men qualified to undergo training in surgery could obtain it. Thus, the necessity for some long-range plan becomes obvious and naturally merges with the immediate post war problem.

Various estimates of the qualified surgeons required annually to replace those deceased, superannuated, or retired for other reasons, place the figure in round numbers from 200 upwards. These figures are not completely reliable since the methods of arriving at the annual requirements differ considerably. In order to supply 200 completely trained surgeons each year, approximately 1,000 would have to be in training if a steady supply is to be maintained. If more than 200 new surgeons are required annually, the number in training would have to be proportionately greater.

The need for increased training facilities can be met only by the creation of more residencies. There is in the country today a shocking waste of opportunity in this respect. As stated elsewhere, there are countless hospitals where the men and material are available for setting up excellent training programs if sufficient interest can be aroused. One often hears, when suggesting that such a plan be initiated, that it is too much trouble. Such people require education and should be made to see their responsibilities. No chief of a surgical service who has experienced the satisfaction which attends a well organized residency would ever voluntarily return to a system whereby he depends upon the services of an intern and a part-time apprentice. It is to be regretted that an occasional hospital superintendent questions in terms of dollars and cents the value of a resident to the hospital. These too must be shown the light.

I have attempted in this communication to discuss the past and the present in Graduate Surgical Training in America. Certain aspects of the future have been touched upon. Certain it is that the responsibility for keeping the torch of surgical progress burning will reside for many years to come in the hands of American surgeons. Let us dedicate our best efforts to the end that we may prove worthy of this responsibility.

ANNUAL ADDRESS FOR 1945
INGUINAL HERNIA WITH SPECIAL REFERENCE TO STRANGULATION

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The age of hernia is as long as that of man himself. One person in ten in the entire world is affected with hernia. About 90 per cent of all hernias are inguinal, and hernia was the third most frequent cause of rejection from military service.

The present opinion seems to be that, except for rare cases of true traumatic hernia, all indirect inguinal hernias are congenital in origin, regardless of the age of the subject when the hernia first appears. The processus vaginalis descends ahead of the testes, and the patent process, which occasionally remains, results in indirect hernia. The descent of the testes is an important factor in the cause of indirect inguinal hernia. Scrotal hernias have been produced experimentally in mice by the administration of estrogen and similar hormones.

Apparently, there is some divergence of opinion, as Keath[22] stated in 1923, "It is not the presence of a patent process of peritoneum which accounts for hernia frequency, but a weakness in the groin in man who has had to adapt this point of his anatomy to the upright posture." He points out that in nearly all animals, except man and gorilla, the process of peritoneum remains open, and yet inguinal hernia is rare in them at every stage of life.

Thiessin[1] records 31 cases of right inguinal hernia following lower abdominal incisions, most of which were of the McBurney type, and states that nonclosure or nonhealing of the transversalis fascia following low abdominal incisions, is the main reason for subsequent appearance of the inguinal hernia. The average interval between the incision in the right lower quadrant and the appearance of the hernia was about five years.

When the McBurney incision was palpated from within, a weakness could be felt in many cases. In all cases the transversalis fascia was found stretched abnormally around the internal inguinal ring. These observations tend to support Lytle's thesis[2] that the internal ring moves with a valvelike action, and it is possible that injury to the iliohypogastric nerve or failure to repair the transversalis fascia allows the internal ring to descend, thereby increasing its relative size and allowing the sac to form. This tends to refute the original idea that all indirect inguinal hernia have a preformed sac.

We believe that the indirect inguinal hernia results from an inherent weakness of the internal ring and its mechanism. The direct hernia results from an inherent anatomic weakness in the region of Hesselbach's triangle. The hernia increases in size mainly as the result of increased intra-abdominal pressure.

The complications of hernia are irreducibility, inflammation, obstruction and strangulation. The irreducible hernia may be the beginning of the other three and, if left untreated, may become inflamed and thereby favor the occurrence of obstruction and strangulation. All these complications exist in strangulation. The presence of omentum in the sac is the most frequent cause of strangulation, as it often becomes adherent to the sac and becomes edematous.

The inflamed and obstructed varieties of inguinal hernia almost always advance to strangulation, this being by far the most frequent and dangerous complication. The differentiation between inflammation, obstruction and strangulation is purely academic. Operation is not nearly so serious as procrastination. A strangulated hernia is one which contains abdominal viscera whose blood supply is partially or completely obstructed. Strangulation in hernia was known to Hippocrates.

It is often difficult to be certain whether the hernia is merely obstructed or strangulated. Since it is next to impossible to determine that there is no bowel in the sac, except by operation, and since mechanical obstruction of the bowel is ultimately as dangerous as strangulation, we believe that all painful hernias should be treated as if they are strangulated. We believe that all painful hernias accompanied by abdominal pain and other symptoms of obstruction should be treated as strangulated hernia by operation without attempting taxis.

In 1758 Dr. P. Pott[3] said:

> Tumor of the groin or scrotum attended with pain not only in the part, but all over the belly creating a sickness and inclination to vomit, suppression of stools, some degree of fever; these are the first symptoms. If not relieved immediately the sickness becomes more troublesome, the vomiting more frequent, the pain more intense, the tension of the belly greater, the fever higher, and a general restlessness which is terrible to bear. When this is the state of the patient no time is to be lost. The remedy is operation. I have myself, seen a small portion of intestine become perfectly gangrenous in one day and night from its first expulsion.

In the vast majority of instances the point of strangulation is at the internal ring. In our series of 91 cases there were four recurrent hernias, and the site of strangulation was at the point of exit from the abdominal wall. There were two cases of the Richter type hernia in our group. This hernia is much more frequently seen as a femoral hernia. There were 17 direct hernias and 5 sliding hernias. There is little excuse for delay in the proper treatment of strangulated hernia. It has often been said that if a case of strangulated hernia be seen in the day time, the patient should be operated upon before the sun sets, and if seen at night, he should be operated upon before the sun rises.

"Taxis" is a term given to the procedure necessary to reduce hernias without operation. Attempts at reduction by the use of ice bags, elevation of the patient and morphine only cause unwise and dangerous delay. It is interesting to note the methods used in the days of Sir Ashley Cooper.[5] He states:

Taxis is done by making pressure on the tumor and at the same time placing the patient in that posture which gives as much room in the abdomen as possible, and relaxes the muscles and aperture. The best position for this purpose is supine, with the body moderately incurvated. This is effected by laying the patient on his back and putting one pillow under the pelvis and another under the shoulders. Both thighs should be elevated at right angles with the body, the knees should be brought so close together as only to admit the surgeon's arm between them. This is a most essential point since it relaxes the fascia and consequently the aperture through which the hernia first quits the abdomen. The surgeon then places himself on the right side of the patient, and embracing the tumor in his right hand, he passes it from side to side, thus endeavoring to get a small portion of it within the abdomen. Pressure should be maintained for a quarter of an hour. I have seen reductions take place in twenty minutes.

He then goes on to talk about slinging the patient by the knees.

If this fails, other means should be resorted to. Bleeding is one of them. The two best remedies are tobacco glyster and application of cold. Tobacco has been used both in the form of smoke and as a liquid glyster. To use smoke requires a complicated apparatus and has been discontinued in the hospitals. The liquid glyster is made by infusing a drachm of tobacco in 12 ounces of boiling water for ten minutes. As the effect of this potent remedy varies very much in different institutions, and perhaps according to the quality of the tobacco, it is best to inject only half the quantity. To those who have commonly heard of two drachms being thrown up at a time without bad consequences, this may appear a useless precaution, but instructed by personal observation, I can venture to assert that whoever practices this often, will meet with effects that will lead him to repent his rashness.

I once saw a man with whom a tobacco glyster had been used in the quantity of two drachms without reduction in the tumor who, when put upon the operating table, his pulse was so low his countenance so depressed and his body covered with sweat, that he was ordered back to bed and on carrying him thither he expired.

A girl had a single drachm injected. It produced most violent pain with vomiting, in which was thrown up matter which strongly smelt of tobacco and she died thirty-five minutes after the glyster had been administered.

It is sometimes permissible to use taxis. In our series, every patient had taxis attempted before coming to the hospital. This method of reduction of strangulated hernia is accompanied by certain dangers. The greatest dangers of this method are: (1) reduction of the hernia en masse with the persistence of the symptoms of strangulation; (2) rupture of the intestine within the sac; (3) reduction of gangrenous intestine; (4) forcing infected material into the abdominal cavity. In our cases, two were reduced en masse. Taxis is permissible (1) if attempted gently for a short time; (2) if, for some good reason, operative treatment is contraindicated; and (3) if the hernia has not been down more than one or two hours. Taxis should never be attempted (1) on the hernia that has been down for several hours; (2) when the onset has been acute and the symptoms severe; or (3) when previous attempts have failed. The patient should be closely observed even if reduction has been accomplished, for if the symptoms of strangulation persist, operation is imperative. There is no doubt that attempts at taxis increase the mortality rate.

Nason and Mixter[4] state that certain conditions must be present before reduction en masse can occur. (1) The neck of the sac must be small enough to retain and incarcerate the contents. (2) The neck of the sac must be sufficiently mobile and detached from the internal ring. When strangulation occurs at the external ring, reduction en masse cannot occur. The vast majority of strangulations occur at the internal ring, which may explain the rarity of this condition. Pearse states that the mortality of these cases is 40 per cent, and that reduction en masse occurs one in 13,000 persons.

In dealing with strangulated hernia and gangrenous bowel, three pathological entities must be treated: (1) intestinal obstruction with its dehydration, toxemia and biochemical upset; (2) the gangrenous loop of bowel, which, if not removed from the peritoneal cavity, will perforate and give rise to peritonitis; and (3) the hernia itself, which should be the last concern.

Regarding the advisability of operation, Dr. P. Pott[3] said:

One evil is very frequently the parent of others. If I might presume to give my opinion on the subject, I should say that the operation should always be performed as soon as possible after it appears that all rational attempts by large and free bleeding, warm baths, glysters (enemas) are found to be ineffectual and the symptoms increase rather than decrease. While such means are made use of and the necessary handling for reduction becomes more painful, for if it be delayed and the parts are not found quite gangrenous are we sure that the removing of the strictures will at this time appease the symptoms? Mortification may ensue though it be set free in the belly. Though I am perfectly satisfied that the case of strangulated hernia is as I have presented it, the disorder of the intestine is originally produced by the stricture made on it by the borders of the opening, and the gut is perfectly sound and free from disease before it becomes engaged in the stricture. I think it right to acquaint the uninformed reader that it is still the opinion of some ingenious men that the disease is originally in the gut and that the stricture is an accident arising from the inflammation and distention of it, or in other words, that the intestine is first inflammed and by means of the lateration produced by such inflammation became too large for the tendinous aperture which therefore makes the stricture on it and which they think is the reason why the chirurgical operation is often unsuccessful.

The mortality rate will vary in direct relation to the length of time between the onset of strangulation and its release. The mortality rate, as quoted by Gatch and Montgomery,[6] varies from Frankau's[7] series of 45 per cent to some smaller series of 100 per cent. Gatch also quotes Alexander,[8] who, before this Academy in 1913, stated that the results of treatment of strangulated hernia had been the same for 20 years previously, and Gatch further stated that apparently the mortality rate is almost the same now as it was in the pre-antiseptic days. Reduction before gangrene develops has the most favorable prognosis. If gangrene has occurred, the prognosis is best when omentum alone is involved. In the resection cases, those with the small intestine involved, have the lowest mortality and it is highest when colon resection is necessary. Watson states that in strangulated hernia when operation is performed in the first 12 hours, the mortality rate is 5 per cent; within 12 to 14 hours, 10 per cent; and 24 to 48 hours, 25 per cent.

The changes in physiology resulting from the intestinal obstruction should be corrected before operation is begun. Each patient was given an intravenous of 2 per cent glucose in normal saline before or during the operative procedure. Those in shock were given blood or plasma. Our patients had been allowed to rest in bed, with the hips elevated, and given morphine and

Table 1. Time Lapse from Admission to Operation

Operation Time	Number of Cases	Percentage of Mortality	Deaths
Under 2 hrs.	21		
3- 6 hrs.	49	2.1	1—Shock, 10 hrs.
7-12 hrs.	9		
13-24 hrs.	6	16.6	1—Shock, 8 hrs.
25-48 hrs.	0		
Over 48 hrs.	4	50.0	2—Heart Disease, 33 days Hemorrhage, 3 days
No Operation	2	50.0	1

atropine until seen by the surgeon. In one case, the hernia reduced itself, the patient refused operation and left the hospital. Twenty-one of our cases were seen by the surgeon and operation was begun in less than 2 hours; 49 were seen between 3 and 6 hours, making a total of 71 cases seen before 6 hours (Table 1).

The anesthetic of choice is, in our opinion, fractional spinal anesthesia. However, local anesthesia has its advocates, and Watson, for example, states that the mortality rate would decrease considerably if everyone used local anesthesia. Nevertheless, we believe that fractional or continuous spinal anesthesia has many advantages. The disadvantage is that there may be a tendency to do too much surgery. We should like to advocate here introducing the intraspinal needle and performing the operation under local. If anesthesia is needed, the anesthetic agent may be introduced through the intraspinal needle (already placed) in amounts just large enough to supplement the local anesthetic. In two of our cases, the spinal needle was introduced, but the spinal anesthesia was not needed. In our series, 60 or 65 per cent had spinal or fractional spinal anesthesia, 12 or 15 per cent had local anesthesia, and 20 per cent had inhalation or inhalation combined with spinal or local anesthesia.

The type of operation performed should depend on the patient's age, his general condition and the duration and degree of strangulation. The most pressing problem is the relief of strangulation—the repair of the hernia is secondary. In operation for strangulated inguinal hernia, the sac should be carefully opened before the constricting ring is divided, as the ring can be divided under direct vision. If the constriction is divided first, the involved bowel may slip back into the abdominal cavity before it has been examined. If so, one should attempt to recapture that portion of the intestine for examination. Small necrotic patches are inverted with Lembert sutures of No. 00000 chromic catgut. If there is a necrotic furrow at the site of stricture, it should be inverted by interrupted sutures of No. 00000 chromic catgut, producing a small intussusception. There were three such procedures in our series, one of whom died on the thirty-third day of arteriosclerotic heart disease (Table 2).

Table 2. Operation

Treatment	Number of Cases	Deaths
Operated	89	4
Repair only	70	2
Resection		
Omentum	9	
Appendix	4	
Bowel turned in	3	1
Bowel and anastomosis	3	

The viability of strangulated intestine is usually determined by color, reaction to stimulation, and consistency. Nonviable intestine loses its lustre, does not contract when flicked with the finger and feels like wet tissue paper. The viability can be determined definitely by the intravenous injection of fluorescein, as described by Hatfield, Buyers and Walkling.[9] This is done by the injection intravenously of 20 cc. of 5 per cent suspension of fluorescein in 5 per cent sodium bicarbonate solution having a pH of 5.7. Viable bowel begins to fluoresce in about 90 seconds when exposed to ultraviolet light viewed through a Woods type filter. Fifteen to 20 minutes of observation should be sufficient. This method was used in 3 of our cases in which the viability of the intestine was questionable. We believe that one should wait at least 20 minutes before sacrificing the involved intestine. Two of our patients were saved the extra risk of resection by this method. One patient in whom resection was done had the hernia repaired 14 days later. If the gangrenous bowel can be removed with reasonable safety, it should be done. If the risk of removing the bowel is such that the patient's life is further endangered, a multistage procedure should be carried out.

Resection with primary anastomosis has a mortality rate ranging from 20 per cent to 63 per cent. The side-to-side anastomosis used in our three cases is much safer than end-to-end anastomosis, as there is apt to be a difference in the circumference of the two ends. A side-to-side anastomosis can be performed above the site of strangulation, and the gangrenous loop may be allowed to remain and may be removed at a subsequent operation. When the intestinal loop is gangrenous and the patient in poor condition, the formation of a fecal fistula is the only safe procedure. This can be done in two ways: (1) by suturing normal bowel wall to the internal ring for fixation, surrounding the affected loop at the ring with iodoform gauze and open the bowel, or (2), if the bowel has not perforated, a second incision can be made above in the abdominal wall, the nonviable bowel pulled through and opened. This results in a double-barreled ileostomy which can be closed later. We are definitely of the opinion that too much surgery is the cause of many of the deaths from strangulated hernia and we should like to add a word of cau-

tion in dealing with the strangulated hernia with gangrenous bowel of more than 24 hours duration. We agree with Gatch and Montgomery[6] in that the Miller-Abbott tube is a great help in these cases, as the normal intestinal physiology returns sooner and the patient can be fed by mouth. Gatch states that in femoral hernia, the strangulated loop is always adherent to the femoral ring. The problem in inguinal hernia is quite different, because the point of constriction varies and the strangulated loop is much less apt to be adherent to the ring. Orchidectomy was necessary in one of our cases because of gangrene. Orchidectomy was done in four other instances, always with the signed permission of the patient. The advantage is that one may close the abdominal wall completely. While the repair of the hernia is secondary in the treatment of strangulated hernia, it will be seen in our series 87 of 89 cases operated upon had primary repair. There is a tendency among surgeons to approach the problem of the surgical treatment of hernia from the standpoint of the routine application of one of the standard operations. Many surgeons seem to be quite satisfied when the recurrence rate has remained more or less stationary.

The attempt at cure goes back at least 1,000 years before the Christian era. Hippocrates wrote of the use of plasters. Celsus in the first century advised ligation and removal of the unopened sac with the removal of the testis. Oribasius, in the fourth century, and Aeginata, 300 years later, freed and excised the sac and added the cautery to encourage sloughing of the sac. It is needless to say that infection and suppuration destroyed the surgeon's work and often his patient. In the nineteenth century, Pancoastin and Velpeau used iodine. The former by injection, the latter by applying it directly to the interior of the sac. Slightly over 100 years ago, Bonnet relied on sepsis to close the sac. Lister's discoveries rapidly increased the number of operations on hernia and reduced the mortality rate to a twentieth of the former rate. Almost every surgeon devised his own operation for hernia. Lucas-Championnière, who was the father of Asepsis, was the first to operate on hernia using boiled or sterilized instruments. Recurrences were still too numerous. Bassini and Halstead reported almost simultaneously in 1889 their methods of repair. This was the greatest advance in treatment since the beginning of surgery. The Halstead method differs only in detail from the Bassini method.

The countless operations for the repair of hernia that have been described since are merely modifications of these two. Wolfer used the rectus sheath or muscle reinforced Heselbach's triangle. Bloodgood used the rectus sheath sutured to the inguinal ligament. Ferguson[10] reinforced the structures in front of the cord and repaired the internal ring. MacArthur[11] suggested in 1901 living suture derived from the external oblique to unite the internal strips of fascia lata between the conjoined tendon and inguinal ligament to oblique and conjoined tendon to the inguinal ligament. Gallie in 1921 used repair the posterior wall of the inguinal canal. Pitzman apparently was the first to appreciate the importance of the transversalis fascia in repair. Seelig

and Chouke[12] showed that muscle sutured to fascia resulted in poor union and suggested that fascia be sutured to fascia. In 1939 McVay[13] brought before us the fundamental error in the Bassini operation. The inguinal ligament apparently does not stand the pull of muscles sutured to it. Therefore, he suggests that the results of operation would be better if the transversalis fascia be sutured to Cooper's ligament covering the pubes as far lateral as the femoral vein. This work of McVay in 1939 and Anson and McVay[14] in 1938 has been the greatest advance in the technic of hernia operations for many years. It is well known that repair by any standard method has a recurrence rate of from 2 to 20 per cent. It has also been shown by Longacre[15] in an analysis of 428 cases of inguinal hernia that the recurrence rate with nonabsorbable suture was less than half that when absorbable suture has been used, i.e., silk 2.16 per cent and catgut 9.2 per cent. In spite of all the evidence available, there are still many surgeons who use absorbable suture material in the repair of hernia. There is evidence at hand that cotton suture material is as good as silk and has less infection rate. In a study of 500 cases of hernia at the Pennsylvania Hospital, the catgut repaired cases had a recurrence rate just twice that of repaired cases by silk and cotton. We agree with Zollinger and Flynn[16] that patients with herniorrhaphies performed with cotton sutures have a much smoother convalescence and wounds are much less painful with less induration than those sutured with catgut. Swenson and Harkins,[17] in speaking of tissue reaction while discussing operations on recurrent inguinal hernias, state that in all cases where absorbable sutures had been used scarring was so extensive that it was difficult to recognize and dissect free the essential structures for an adequate repair, while in those cases in which cotton or silk had been used, scarring was minimal and fascial layers were easily discernible, greatly facilitating the repair of the defect.

In strangulated hernia, the sac is disposed of in any of the conventional methods. The Bassini repair has been used in our cases to the extent of 67.8 per cent. In 1940, Zimmerman, in an article on repair of hernia, stressed the important differences between indirect and direct inguinal hernia in reference to repair. He stated that the small indirect inguinal hernia is essentially the result of a dilated internal ring. All that is necessary for repair is to close the internal ring more tightly about the cord. The large indirect hernia in which the internal ring is much enlarged presents the same problem as does the direct hernia. The essential problem is "repair" of the defect in the fascial floor of the canal due to insufficient support from absence of overlying muscles. The repair logically should consist of the repair of the fascial defect with fascia. Instead of using fascial grafts from above such as rectus sheath or using fascial sutures, he proposed the use of a portion of the external oblique aponeurosis as the reinforcing material.

Our last three cases have been repaired by the McVay technic which we think is superior for all types of groin hernia, i.e., indirect, direct and femoral.

Harkins et al.[25] accept the theory that the transversalis and the internal

oblique fibers do not normally attach to Poupart's ligament, but rather to Cooper's ligament. They have used a repair which restores the normal attachments to Cooper's ligament. No fascia is sutured to Poupart's ligament. In 131 herniorrhaphies, only one recurrence was found. Swenson and Harkins[17] reported in 1943 that there had been no direct recurrence in 37 cases operated upon and followed over a period of 3 years. This procedure is a modification of the Lotheissen operation for femoral hernia. If the transversalis alone appears strong enough, it alone is used as the upper leaf of the repair. If not, the internal oblique aponeurosis is used. No muscle is used. The lower leaf is Cooper's ligament. The transversalis fascia or internal oblique fascia is sutured to Cooper's ligament between the spine of the pubes and the femoral vessels. This effectively closes off the femoral canal and reinforces the weakened floor of the inguinal canal. It may be difficult to bring the internal oblique aponeurosis down without tension and if so, a relaxing incision is made by cutting through the internal oblique aponeurosis just lateral to where it joins with the external oblique to form the linea alba. These sutures are of silk and are tied separately and tied together. In our cases No. 40 cotton thread was used. We have been so struck by the efficiency of this method that we are now using it on all our cases of groin hernia.

In 89 cases of strangulated inguinal hernia, there was a mortality rate of 4.5 per cent in the cases operated upon. In six cases containing gangrenous intestine, there were no deaths as the result of the operation. Three were sutured, three were resected. One death occurred 33 days after operation from arteriosclerotic heart disease.

Finally it will be seen from the foregoing that the treatment and the results of treatment in strangulated inguinal hernia still leaves much to be desired. We believe better results can be obtained by strict attention to the details mentioned. Fairbank, an English surgeon, once said, "You can judge the worth of a surgeon by the way he does a hernia."

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ANNUAL ADDRESS FOR 1946
THE PATHOGENESIS AND TREATMENT OF PULMONARY EDEMA IN RELATION TO SURGERY*

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The classical experiments of Starling[54] demonstrated that the fundamental factors concerned in the movement of fluid between the blood and the tissues are: the hydrostatic pressure in the capillaries, the semipermeable capillary membrane and the colloid osmotic pressure of the blood. As the salt concentration in the extracellular fluids and the blood plasma is the same, it is the colloid osmotic pressure of the plasma proteins which offers the opposing force to the hydrostatic pressure in the capillaries. It was the later work of Landis[34,36] with his direct measurements of intracapillary pressure, which gave additional evidence in support of this hypothesis. Cannulation of lymphatic vessels and chemical analysis of the lymph obtained from various parts of the body by Drinker[15] has thrown additional light upon the composition of the extracellular fluid. The conclusions which have now been reached by all students of the subject are that Starling's fundamental thesis is valid and that capillaries in general, although they leak small amounts of protein (with the exception of capillaries in the renal glomeruli, choroid plexus and ciliary body) are relatively impermeable to colloids and thus succeed in retaining fluid in the blood stream.

Many circumstances can affect this balance between the capillary pressure and the colloid osmotic pressure of the plasma. The commonest of these factors is an increase in venous pressure, which is immediately reflected by an increase in capillary pressure. The contrary, of course, is not true with changes in arterial pressure. The filtration of fluid from the blood capillaries into the extracellular spaces, once initiated, is restrained by the development of tissue pressure.[37] This tissue pressure results from the accumulation of fluid in tissue spaces which are surrounded by relatively unyielding structures, such as sheaths of muscles, fascial layers and the skin itself.

The integrity of the capillary wall is an essential requirement for the maintenance of these normal relationships. The capillary wall may be injured by many noxious agents, the commonest of which is undoubtedly trauma. A diminished supply of oxygen to the capillary wall has also been shown by Landis[35] to affect immediately and markedly the ability of the capillary wall to retain colloids in the blood stream.

ANATOMIC AND PHYSIOLOGIC CHARACTERISTICS OF THE LUNGS IN RESPECT TO EDEMA

A discussion of pulmonary edema must begin with a consideration of the peculiar anatomic and physiologic arrangements in the lungs. These extraordinarily efficient organs are designed for exchange of gases between the blood and the outside atmosphere. In a space roughly 10 inches high and of a girth somewhat less than the classic 36 inches more than 90 square meters of surface are provided for the exchange of gases between the blood and atmospheric air.[17] This is roughly an area of 31 x 31 feet. Having mechanically attempted to provide a surface for this gas exchange in a small animal (the cat) by using the inner side of a cylinder 8½ inches in diameter and 22 inches long, an area roughly of 2 x 2 feet,[24] I have been led time and again to marvel at the efficiency of the lung which provides such an enormous aerating surface in such a small space. This is accomplished by the spongelike construction of the lung which fills every nook and cranny of the thorax. The bronchi arborize rapidly into many fine branches, ending in a noncartilaginous bronchiolus varying from 0.6 to 0.3 mm. in diameter. At this point, the columnar ciliated epithelium changes to a cuboidal-type of cell. From this terminal bronchiolus, the respiratory bronchioles arise, approximately 0.3 mm. in diameter. The bronchioles eventually terminate in alveolar ducts measuring about 0.2 mm. in diameter. This ductus alveolarus opens out into pulmonary atria, which have out-pouchings known as "sacculi alveolares" and "alveoli pulmonum."[44] These irregularly shaped air pockets, in which the air ducts terminate, provide an extraordinarily large surface area in a very limited space.

The pulmonary alveoli are lined with a very thin single layer of flattened cells. Between these delicate membranes lining adjacent alveoli lies the closely knit meshwork of pulmonary capillaries. These alveoli communicate with one another through many small circular openings which have been designated as alveolar pores.[38] The walls of the capillaries and this thin layer of epithelium, which is closely applied to the capillaries, is all that separates the blood from the air in the alveolus. Consider how different this structure is from that in capillaries elsewhere in the body, where the capillaries lie cheek by jowl with the packed cells of the organs they supply with blood. In the lungs the capillaries are surrounded by the nonresistant air. Between the lungs and other tissues, there is another structural difference which has a bearing upon the absorption of excessive amounts of extracellular fluid. The lymphatics of the lung do not extend beyond the ducti alveolares, and no lymphatics are present in the walls of the air spaces distal to these structures.

What are the functons which this remarkable organ, the lung, is called upon to perform? At all times the lungs must accommodate the same quantity of blood per minute which flows through all the rest of the body. It is probably the most vascular organ per gram of tissue. Every other organ takes only a fraction of the total output of the heart, while the lungs accommodate the entire output. Under basal conditions, this amounts to between 3 and 4 liters a minute. A red cell requires between 5 and 10 seconds in transit through this giant sponge. The average amount of blood in the lungs under basal conditions is between 500 and 800 cc. The lungs thus

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contain at any one time approximately 1/12 of the total blood volume. The pulmonary capillaries are 0.5 to 1.0 mm. in length and 10 microns in diameter. With pulmonary congestion, the capillaries may enlarge from 10 to 30 times their previous size.

During violent exercise, the lungs are able to accommodate a cardiac output up to 37 liters a minute; in other words, 10 times the normal resting output. The volume of air which is moved in and out of the lungs under these circumstances will change from a resting volume of 8 liters to 125 liters per minute.[16] This increase in the movement of air in and out of the lungs must match the increased blood flow in order to meet the requirements for oxygen under these circumstances.

With violent exercise, one encounters the phenomenon of pulmonary congestion and early edema in healthy humans. All of you will remember, after violent exercise, such as a grueling race, the panting respirations which frequently follow with the coughing up of clear salty fluid. Those who are accustomed smokers will probably remember that smoking a cigarette after violent exercise will frequently provoke coughing. This is undoubtedly a result of the extreme pulmonary congestion which occurs under these conditions of stress and represents one form of pulmonary edema, which will be discussed in more detail later.

FACTORS WHICH CONTROL THE MOVEMENT OF FLUID THROUGH THE CAPILLARY WALL

There are three chief factors which govern the movement of fluid into or out of the blood vessels; they are: (1) the hydrostatic pressure within the capillaries, (2) the colloid osmotic pressure of the blood and (3) the semipermeable capillary membrane.

There is a fourth factor which occasionally operates to produce an excessive amount of extracellular fluid, rich in protein. This factor is obstruction of lymphatic channels. Under normal conditions, in all the tissues of the body, with the exception of the lungs, the following sequence of events takes place when blood passes through the capillaries. At the arterial end of the capillary, the hydrostatic pressure is slightly greater than the colloid osmotic pressure. Water, electrolytes and dissolved gases pass out of the blood plasma through the capillary wall in this region. A very small amount of protein is contained in this fluid. At the venous end of the capillary, the hydrostatic pressure is somewhat less than the colloid osmotic pressure of the blood. This results in the movement of water, salts and dissolved gases from the extracellular tissue spaces into the blood. Probably little, if any, of the protein which has leaked out of the arterial end of the capillary is returned to the blood stream here. This protein, which has been concentrated by the reabsorption of water and salts into the blood, readily enters the lymphatic vessels in the tissue spaces and thus is returned to the blood stream through the lymphatic ducts.

The situation in the lung differs from that described above in several important characteristics. The colloid osmotic pressure of the blood in the pulmonary capillaries is as high as elsewhere in the body, 25 or 30 mm. Hg., but the hydrostatic pressure of the blood in the pulmonary capillaries is considerably lower than that in the peripheral capillaries. It has so far been impossible to measure directly the pulmonary capillary pressure under physiologic conditions. The reason for this is that the natural movements of the chest wall and diaphragm and an intact thorax must be present to maintain a normal state in the pulmonary capillaries. By indirect means, the pressure has been variously estimated to be in the neighborhood of 10 mm. Hg. The pulmonary arterial pressure and the pulmonary venous pressure have both been measured in the closed thoraces of animals. The pulmonary arterial pressure is approximately one-third that in the large systemic arteries. The pressure in the pulmonary veins is very low. Recently, in the human being, pulmonary arterial pressures have been measured in the intact closed thorax by catheter technic,[12] and in the open thorax by introducing a needle directly into the pulmonary artery.[7] The pressures so determined are of the same order of magnitude as those recorded in animals. The pressure in the human pulmonary artery is normally between 35 and 40 mm. Hg. On the basis of these and other observations, it is reasonable to conclude that the pulmonary capillary pressure is in the neighborhood of 10 mm. Hg. The situation thus makes for dryness in the lungs, because the colloid osmotic pressure of the blood is so much greater than the capillary pressure. Under normal conditions there should be little, if any, passage of water and salt solution into the extracellular space between the pulmonary capillary and the alveolar wall. The tendency would be rather for the immediate absorption of any fluid between the capillary and alveolar walls, because of the low capillary pressure. Thus, there can occur a considerable increase in pulmonary capillary pressure without the appearance of edema. This state of affairs affords great protection to the vitally important gas exchange in the lung, which is rapidly and disastrously interfered with by the presence of fluid in the alveoli.

The classical experiment which has often been quoted to illustrate this mechanism for dryness was reported by Colin in 1873.[11] He poured 21 liters of water into the trachea of a horse over a period of 3½ hours, without producing any ill effect. Winternitz and Smith[62] made similar observations in dogs, introducing large amounts of salt solution into the trachea. This method of administering parenteral fluid to a dehydrated patient might be of interest to some enterprising clinician. I am not aware that it has yet been employed in humans.

Four factors, which do not operate in other parts of the body, influence the formation and removal of extracellular fluid in the lungs. These are (1) the oxygen supply to the pulmonary capillaries and alveolar epithelium, (2) the absence of lymphatics in the alveolar walls, (3) the negative intrathoracic pressure and (4) the absence of the restraining effect of surrounding solid

tissues on the formation of fluid. In all the tissues of the body except the lung, capillary endothelium receives its oxygen supply from the blood within its walls. On the other hand, the pulmonary capillaries and the epithelium lining the alveoli are normally dependent for their oxygen supply upon the alveolar air. Miller[44] has shown that the lymphatics in the lung do not extend beyond the ducti alveolares. The absence of lymphatics in the alveolar walls prevents their participation in the removal of early collections of extravascular fluid in this region. Drinker[17] remarks on "the inadequacy of the lymphatic system in the lungs to keep pace in terms of removal with a reasonably steady and rapid production of excess tissue fluid." The same investigator has shown again that on compression of the pulmonary veins there is an increase in the flow of lymph from the lungs and that, when the pressure is released, the lymph flow falls but not to a normal level for some time, indicating that the excess fluid requires an appreciable length of time to be absorbed by both blood and lymph capillaries. Thus, although there is a considerable protective mechanism in the lungs making for dryness, once pulmonary edema has appeared either from capillary damage or from increased capillary pressure, the mechanism for the reabsorption of that fluid is not strikingly efficient.

The negative intrathoracic pressure of —5 to —10 mm. Hg. favors the outward filtration of fluid from the pulmonary capillaries. For practical purposes, it can be added to the hydrostatic pressure of the blood in the pulmonary capillaries. Estimating the latter at 10 mm. Hg. and the former as averaging 7 mm. Hg., it diminishes the forces making for dryness to 11 mm. Hg., the difference between 17 mm. Hg. and 28 mm. Hg., the colloid osmotic pressure of the blood. There are certain clinical conditions in which the respiratory movements are markedly exaggerated. Under these circumstances, the negative intrathoracic pressure on inspiration is greatly increased, a condition which makes for pulmonary congestion and edema. Crowden[14] has demonstrated by x-ray that Muller's experiment (attempted inspiration against a closed glottis) increases the amount of blood in the lungs, whereas Valsalva's experiment (attempted expiration against a closed glottis) decreases it. A vicious cycle occurs once pulmonary edema has begun. Anoxemia results from the edema, and the anoxemia, in turn, increases the rate and depth of respiration. This increases the negative intrathoracic pressure, resulting in a greater tendency to edema. The clinical significance of deep labored respirations in pulmonary edema will be discussed later under treatment.

In practically all the tissues of the body except the lung, the blood capillaries are surrounded by solid masses of cells. These, in turn, are frequently encased in capsules or sheaths of fibrous tissue. This environment naturally offers some resistance to the accumulation of fluid in the tissue spaces. This factor of tissue pressure is difficult to measure directly. Meyer and Holland[43] found a pressure of 7 cm. of water in the skin and 3 cm. of water in the subcutaneous tissue. Krogh, Landis and Turner[33] found that venous pressures

below 15 cm. of water failed to produce significant filtration of fluid into the tissue spaces. Landis and Gibbon,[37] using a pressure plethismograph found that no significant filtration occurred from the capillaries into the tissues below 10 cm. of water pressure in the vein. They further showed that as fluid accumulated in the tissues of the human forearm, there was a decreasing rate of filtration at any given venous pressure. Thompson et al.[56] found that fluid was lost from the blood on quiet standing but that this loss ceased before edema appeared in the legs. This was probably due to the dual factors of tissue pressure and concentration of the blood colloids from the loss of water and solutes from the plasma. These studies show that as fluid accumulates in the tissues the increase in tissue pressure retards the further accumulation of fluid.

In the lung the situation is wholly different. The blood capillaries lie between the alveolar walls which consist only of an extremely thin, delicate, double layer of flattened epithelial cells. Beyond these cells lies the alveolar air, which obviously offers no resistance to displacement by fluid. When the alveoli are full of fluid, the only resistance to further filtration from the capillaries is the hydrostatic pressure of the fluid in the bronchi. This complete absence of the restraining influence of tissue pressure upon the accumulation of fluid in the lungs very probably accounts for the rapidity with which pulmonary edema has been observed to occur.

CLINICAL CONDITIONS IN WHICH PULMONARY EDEMA OCCURS

The clinical conditions in which pulmonary edema arises can be grouped best under the following heads: increased pulmonary capillary pressure, increased negative intrathoracic pressure, increased permeability of the pulmonary capillary wall, decreased colloid osmotic pressure of the blood and, finally, lymphatic obstruction. I have listed these factors in the probable order of their clinical importance. More than one is usually present when pulmonary edema occurs clinically. The first four concern the edema arising from the blood vascular system and the last from the lymphatic system. They will be considered in the order of increasing importance.

Lymphatic Obstruction. Because lymphatic vessels are so numerous and ubiquitous, it is comparatively rare to find the accumulation of protein-rich fluid in the tissues due to lymphatic obstruction. The commonest examples are the brawny edema of the legs, known as elephantiasis, which is the end result of chronic obstruction of the lymphatics from infection, and lymphedema of the upper extremity, due to obstruction of the lymphatics by tumor growth in the axillary lymph nodes, secondary to carcinoma of the breast. It is also sometimes seen in the upper extremity, following radical mastectomy from interruption of the lymph channels draining the upper extremity.

It is possible that the counterpart of elephantiasis or lymphedema of an extremity exists in the lungs, but, to my knowledge, it has never been described or recognized. The supply of lymph vessels in the lungs is far

richer than that of the kidney, spleen or liver. This abundance of lymphatic channels accounts for the rarity of their complete obstruction from a pathologic process. Because of the rich intercommunication in the mediastinum between the lymphatic vessels of the right and left lungs, it is unlikely that obstruction of either the right lymphatic duct or of the thoracic duct would, in itself, produce lymphedema of the lung. Pathologic states obstructing both these ducts must be exceedingly rare. A block of the lymphatic vessels in the hilum of a lung from involvement by silica particles, tumor cells or infectious processes, such as tuberculosis, could conceivably be expected to produce lymphedema of the affected lung. However, such lymphedema, if it occurs, is of no clinical importance because the primary process overshadows these secondary effects.

Decreased Colloid Osmosic Pressure of the Blood. Pulmonary edema, in the absence of edema elsewhere in the body, probably never occurs solely as a result of decreased colloid osmotic pressure of the blood. The reasons for this are obvious. In the systemic blood capillaries, the hydrostatic pressure in the vessels equals the colloid osmotic pressure. Any marked decrease in the latter may be expected to produce promptly an increased outward filtration of fluid through the capillary wall. For example, in congestive heart failure, Moore and Van Slyke[46] found that edema was almost invariably present when the total plasma protein was below a level of 5.5 Gm. per cent and the serum albumin below 2.5 Gm. per cent. The lowered protein concentration reduces the colloid osmotic pressure of the blood, leaving a balance of forces in favor of the outward movement of fluid from the capillaries into the tissue spaces. A comparable reduction in the colloid osmotic pressure of the blood would not be expected to produce pulmonary edema because of the safety factor making for dryness in the lungs (approximately 11 mm. Hg.).

There are, undoubtedly, numerous conditions in which a decreased colloid osmotic pressure of the blood is a secondary factor of importance in the production of pulmonary edema. To my knowledge, it is never the primary cause in the absence of edema in other parts of the body. States of hydremia, such as occur clinically in glomerulo-nephritis, the toxemia of pregnancy, beriberi and such as can be produced experimentally by massive intravenous infusions, are examples of such conditions.

Increased Permeability of the Pulmonary Capillary Wall. The balance of forces making for dryness in the lungs is immediately upset if the integrity of the semipermeable membrane of the capillary is impaired. This membrane is not easily injured alone except by local infectious processes such as pneumonia. If toxins which affect the capillaries generally are produced in the body, the capillaries in the lung will share the injury. But in the lungs the forces for dryness will prevent the early appearance of pulmonary edema. Thus, Moon produced shock and death in dogs by the introduction of muscle substance into the peritoneal cavity by burns, intestinal obstruction and injections of sodium glycocholate, bile, sodium phenobarbital and histamine.

In the late deaths produced by these means, there was evidence of general capillary injury throughout the body and, also, pulmonary edema. Where death occurred rapidly after these procedures, pulmonary edema was absent.[45]

The best clinical example of toxins affecting the permeability of the pulmonary capillaries alone occurs in pneumonia. Here bacterial toxins increase the capillary permeability. There is an outpouring of protein-rich fluid and cells, resulting in the familiar consolidation of a lobe of the lung. This process is similar to that following infections in other tissues, which injure capillaries in the affected region and result in a transudation and exudation of fluid.

Inhalation of toxic fumes or poison gases is another familiar, although fortunately rare, example of direct injury of the pulmonary capillaries. It is probable that people who are burned in a fire do not inhale the flame as far down as the alveoli. The upper respiratory passages are burned, but the deeper portions of the lung are affected only by the toxic fumes and smokes, which penetrate to the alveoli. The extremely early pulmonary edema, which occurred in two cases in the Cocoanut Grove fire, was probably produced by toxic fumes.[41] Pulmonary edema, occurring several days after a severe burn, may be the result of the severe injury to the mucous membrane of the tracheobronchial tree and other complicating factors.

The pulmonary edema resulting from the inhalation of poisonous gases was intensively studied in World War I and in World War II. Of the lung-irritant gases, such as chlorine, chlorpicrin, lewisite, mustard and phosgene, the latter is the best example of a pure lung irritant. In the usual concentrations, it does not irritate the skin and mucous membranes as do mustard and lewisite. A discussion of the effects of poison gases does not lie within the scope of this paper. However, the extensive research which has been directed toward the discovery of an effective treatment has been helpful in the evaluation of methods of treatment of pulmonary edema due to other causes.

There is only one chemical substance, so far as I know, that can be absorbed into the general circulation and produce a fatal pulmonary edema, without any demonstrable injury to capillaries elsewhere in the body. In 1943, the McKenzies,[40] reported that thiourea, in addition to causing thyroid enlargement and a lowered metabolic rate, produced a rapid and fatal pulmonary edema in adult rats. They further demonstrated that there was no relation between the effect on the thyroid and the pulmonary edema. Richter, in 1945,[51] reported that alpha-naphthyl-thiourea kills rats and dogs by its action on the pulmonary capillaries, producing a fatal pulmonary edema. Drinker[17] has confirmed these observations on this extraordinary compound and measured the marked increase in lymph flow from the lungs following the administration of this drug in dogs. Alpha-naphthyl-thiourea exerts this effect, whether given parenterally or by mouth. At autopsy, these animals exhibited extensive edema of the lungs and some pleural effusion. Urine was excreted normally during the experiments, the myocardium was normal,

and there was no excessive fluid in the pericardial sac. The peculiar susceptibility of the pulmonary capillary endothelium to this drug is an amazing but incontrovertible fact.

Many years ago, Landis[35] demonstrated that anoxia increased the permeability of systemic capillaries. Drinker[17] has pointed out that the endothelium of the pulmonary capillaries is dependent upon the alveolar air for oxygen because the blood entering the capillaries is venous in character. However, under resting conditions, the mixed venous blood in the pulmonary capillaries contains 70 per cent oxyhemoglobin, an amount which should be sufficient to prevent an increase in permeability from anoxia. Henry[31] studied the effect of anoxia upon the capillary permeability of the human arm. He found that at an altitude of 18,000 to 20,000 feet, with arterial oxyhemoglobin values of 60 to 70 per cent, there was no increase in fluid loss from the capillaries at any given venous pressure. Experimentally, Warren[58] has shown that, in dogs with controlled artificial respiration, changing the inspired air from 21 volumes per cent oxygen to 10 volumes per cent produces an immediate and marked increase in the flow of lymph from the lungs. This effect is not due to any change in the respiratory rate or depth, as these were kept constant during the experiment. As Warren was exposing dogs to approximately the same partial pressure of oxygen (75 mm. Hg.) as Henry exposed his human subjects, the evidence is conflicting. However, it is probable that pulmonary edema due to decreased oxygen tension in the inspired air is rarely if ever seen in patients. The symptoms of cerebral anoxia which appear at high altitudes are well known, and yet no instance of pulmonary edema under these conditions has been reported. The nerve cells of the brain are evidently more susceptible to lack of oxygen than are the pulmonary capillaries. Gibbs et al.[27] have produced unconsciousness in human volunteers from breathing air containing only 4 volumes per cent of oxygen. No mention of the occurrence of pulmonary edema was made in any of these experiments.

Perhaps the commonest type of cerebral anoxia seen in surgical practice is that occurring during an improperly administered anesthetic or where there is some obstruction to the respiratory passages. The milder forms are manifested by personality changes and disturbance of vision, and the more severe forms, by prolonged unconsciousness and death. All this occurs without pulmonary edema. Thus, it seems evident that a marked diminution in the oxygen content of the alveolar air will produce death from the effects of anoxia upon the brain and other vital organs long before deleterious effects upon the pulmonary capillary endothelium become evident in the form of pulmonary edema.

Increased Hydrostatic Pressure in the Pulmonary Capillaries. Clinically, the commonest cause of pulmonary edema is an increase in the hydrostatic pressure in the pulmonary capillaries. In the systemic circulation, it is well known that changes in the arterial pressure are not necessarily reflected in capillary pressure, whereas an increased venous pressure produces immediately an increased capillary pressure.[36] Obviously, similar hydrostatic principles obtain in the pulmonary circulation. Any obstruction to the outflow of blood from the pulmonary veins will result in an engorgement of the pulmonary vascular bed with an increased capillary pressure, provided that the inflow from the right heart remains constant.

One of the most amazing mechanisms in the human body is the absolute equality of output from the right and left sides of the heart from minute to minute. Consider, if you will, the shape and irregular structure of the two great pumping chambers of the heart, the right and left ventricles. The wall of one, the left ventricle, is composed of muscle tissue which is three times as thick as the muscle tissue surrounding the right ventricle. The cavities of each ventricle are irregularly cone-shaped, and projecting into the cavities are ridges and columns of muscles known as the *trabeculae carneae*. Extending into the ventricles from the margins of the openings into the auricles are the broad, thin leaflets of the tricuspid valve on the right and the mitral valve on the left. Consider the work which the two ventricles must perform. The right ventricle expels blood against a resistance of 35 or 40 mm. Hg., while the left ventricle contracts against a resistance three times as great. Is it not in the nature of a miracle that, with such irregularly shaped cavities working with such dissimilar loads, the ventricles deliver equal amounts of blood with such precise constancy?

Let us consider for a moment what would happen if the right ventricle were to expel, with each contraction, 3 cc. more blood than the left ventricle. Under basal conditions in an average-sized adult, the volume of blood delivered from each side of the heart with each beat is approximately 60 cc. Our hypothetical 3 cc. difference would represent only a 5 per cent increase in the output of the right ventricle. Yet in ten minutes there would be 2,100 cc. more blood contained in the pulmonary vascular bed than was there before. This extra amount of blood in the pulmonary circuit is approximately one-third of the blood volume. Obviously, such a condition is not compatible with life. We can only conclude, therefore, that even such a slight discrepancy in output never occurs for that length of time. This illustration can be expressed in another way: a one per cent difference in output or a 0.6 cc. difference in stroke volume, persisting for as long as 50 minutes, would produce similar results.

The explanation for this extraordinary phenomenon lies in the physiologic characteristics of muscle tissue. Starling[48] has studied these characteristics in the heart and expressed them as "the law of the heart." The "law" is that, within certain limits, "the energy set free at each contraction of the heart is a simple function of the length of the fibers composing its muscular walls." Thus, under normal conditions, a greater diastolic filling or an increase in arterial pressure will result in stimulation of the heart fibers to perform increased work.

Clinically, pulmonary edema most commonly occurs as a result of a breakdown in the equality in output between the right and left heart. Mitral

stenosis and insufficiency, aortic insufficiency, hypertension or coronary artery disease may all result in a failure of the left ventricle to keep pace with the right. Such left-sided heart failure is known clinically as "cardiac asthma." The term was first used in 1832 by James Hope,[32] who wrote an excellent description of the condition. Experimental confirmation of the fact that failure of the left heart to keep up with the right results in pulmonary edema was reported by Welch[61] in his classical experiments in 1878. He showed that compression of the left ventricle or partial obstruction of the aorta resulted in pulmonary edema in animals.

The best recent account of the pathologic physiology was given by the late Soma Weiss in 1942.[59] He pointed out the following:

(1) The pulmonary circulation can fail when the greater circulation is adequate. Thus, dyspnea, orthopnea, pulmonary edema and low vital capacity can be associated with a normal cardiac output, normal blood pressure and venous pressure, and normal utilization of oxygen by the tissues. (2) Thickening and rigidity of the alveolar sac wall occurs with pulmonary congestion resulting in an emphysemalike state. (3) Pericapillary edema may be present without alveolar edema. Thus, patients with left ventricular failure may have dyspnea, orthopnea and low vital capacity without rales. (4) Left ventricular failure does not mean that the output of the two ventricles is continuously unequal. Such a condition can exist only for a few seconds or minutes. Then the output of the two ventricles equalizes, but pulmonary engorgement persists until the left ventricle pumps out more blood than the right for a short time. (5) Left ventricular failure can occur with high, low or normal blood pressure.

Although the commonest cause of pulmonary engorgement and increased pulmonary capillary pressure is left heart failure, there are a number of clinical conditions in which states of hydemia occur with increased circulating blood volume. These conditions do not produce pulmonary edema in themselves, but, associated with left heart failure or decreased colloid osmotic pressure of the blood, they are important contributing factors. Thus, Edeiken and Griffith[19] report a case of cyclic pulmonary edema at the menses in a patient with mitral stenosis. They found that there was a 20 per cent increase in the patient's blood volume during her menstrual periods. The diuresis produced by mercupurin at the time of her menses prevented the attacks, and the patient was eventually permanently relieved by irradiation of the pituitary gland. As the authors point out, this was undoubtedly a case in which pulmonary congestion already existed because of mitral stenosis, and the increased circulating blood volume at the time of her menses was the precipitating factor. Bramwell and Jones[9] report two patients dying from pulmonary edema in midpregnancy, who probably fall into the same category. They believe that the pulmonary edema was due to left auricular failure associated with the increased cardiac output and blood volume of pregnancy.

Conditions in which there is an excessive amount of fluid in the body,

such as glomerulo-nephritis[42] and eclampsia, are often complicated by acute pulmonary edema. Teel et al.[55] have reported that pulmonary edema occurs in 30 per cent of all patients with eclampsia. They report six typical cases of cardiac asthma. All these patients had hypertension and marked peripheral edema. In one patient in addition, the plasma protein level was very low. The attacks of pulmonary edema came on suddenly and progressed rapidly while the patients were at bed rest under treatment. The authors suggest that the edema fluid is mobilized while the patient is at bed rest and that the resultant hydremia, plus the excessive burden on the left ventricle from hypertension, produces the pulmonary edema. In this connection, Selye's[53] observations are of interest. He reported that in bilaterally nephrectomized rats pulmonary edema induced by adrenalin does not occur in the first 24 hours if the rats are exercised, whereas the rats that were not exercised all showed pulmonary edema. It is possible that in the rats that were exercised, a sufficient amount of excess water was vaporized from the respiratory tract to prevent pulmonary edema. This observation is another instance of the association of pulmonary edema with hydremic states.

Excessive amounts of fluid in the vascular system or in the tissues may be produced by the injudicious use of intravenous infusions, as well as by disease. Pulmonary edema occurs not infrequently these days with the widespread use of intravenous fluids in postoperative patients. It is more apt to occur with the use of fluids which are retained in the vascular system, thus producing an increased blood volume. The Gibbons[26] reported a case of a 71-year-old woman who had hyperthyroidism and congestive heart failure. A thyroidectomy was performed and several days postoperatively a plasma infusion was given because of hypoproteinemia. The infusion was given slowly, but it had to be discontinued after 300 cc. because of the presence of dyspnea and loud rales throughout the chest. Two days later another plasma infusion was given, but again it had to be discontinued because of signs and symptoms of pulmonary edema. Luisada and Sarnoff[39] produced severe pulmonary edema in 16 out of 18 dogs by giving huge intravascular infusions (2.3 times the estimated blood volume). The fluids used were Tyrode's solution, physiological saline, bovine albumin solutions and oxygenated dogs' blood. The pulmonary edema was most marked with the use of the blood and least marked with the solutions that were not retained in the vascular system. These observations probably also represent the type of edema due to increased pulmonary capillary pressure occasioned by an increased blood volume.

The Negative Intrathoracic Pressure. The negative intrathoracic pressure is a factor which affects the balance of forces governing the movement of fluid through the pulmonary capillary endothelium. It is considered a subheading under increased capillary pressure because it is additive to that factor in the production of pulmonary edema. Normally, it varies between —5 and —10 mm. Hg., but this figure can be greatly increased with labored respiration and may become a factor of paramount importance. Some very

beautiful observations on the effects of exaggerated breathing have been made by Harrison and his co-workers.[30] They found that in both normal individuals and in cardiac patients, exaggerated breathing increased the absorption of oxygen from inspired air. As the arterial blood was normally saturated to begin with, the only explanation is that the blood flow through the pulmonary circuit is increased under these circumstances. Some of the cardiac patients responded to forced breathing with a diminished vital capacity and, occasionally, pulmonary edema. In the cardiac patients they observed that the deep breathing, which followed attacks of coughing, may initiate pulmonary edema. Experimental evidence for these observations has been offered by the same authors.[29] In dogs with intact chests, the introduction of 25 cc. of blood into the ligated left pulmonary artery, while the left pulmonary veins were tied, led to a well-marked increase in the respiratory rate and ventilation. They conclude that "the pulmonary afferent fibers of the vagus nerve are extremely sensitive to pulmonary congestion. A very small amount of excess blood in the pulmonary vascular bed causes a well-marked reflex stimulation of breathing." Thus, pulmonary congestion, brought on by deep breathing, stimulates further the respiratory movements and a vicious cycle results.

The question of the relation of bilateral vagotomy to pulmonary edema in animals will not be discussed here. Conflicting results have been reported in different species and with different experimental procedures. However, the observations of Farber[21] are of some interest in connection with the effects of respiratory movements on pulmonary edema. He gave large intravenous infusions of physiological saline to rabbits. This caused rapid, shallow breathing but no pulmonary edema. In another group of animals, he sectioned both vagus nerves; this resulted in slow, deep respiration. Administering the same volume of salt solution intravenously then resulted in pulmonary edema. It is quite possible that the character of the respirations contributed to this result.

TREATMENT

In the treatment of pulmonary edema, most therapeutic measures can be applied with equal effectiveness, regardless of the etiological factor, or factors, involved. The obvious exception to this statement is the pulmonary edema due to direct injury to the capillary endothelium, of which the most striking example is phosgene poisoning. Despite the extensive research which has been done on this subject, none of the therapeutic measures to be discussed are particularly effective in this condition. The commonly used measures in the treatment of pulmonary edema are venesection, or peripheral venous stasis, morphine, oxygen and, more recently, positive pressure breathing. Other less important or unestablished measures will be mentioned briefly.

The oldest and most time-honored method of treating pulmonary edema is by venesection, and it rightfully retains its position as effective therapeusis

in left heart failure. The more modern method of peripheral venous stasis embodies the same principle. This is accomplished by applying blood pressure cuffs to all four extremities and inflating them to a pressure a little below the diastolic blood pressure. Thus, blood is trapped in the veins of the extremities. The rationale of venesection, or peripheral venous stasis, is obvious and is well expressed in the words of Weiss and Robb:[60] "In order to be compatible with life, the duration of the acute failure of the left ventricle can last but a short period of time, in the order of seconds or minutes. Following this, the vital balance between the ventricles, as far as blood flow is concerned, is re-established but with continued disturbance within the pulmonary circuit. The blood in the lungs remains trapped and, therefore, the dyspnea continues after the ventricular balance is re-established. . . . For the relief of the attack, it is essential, therefore, that the left ventricle be capable of throwing out more blood from the lungs than the right throws in." The simplest method of accomplishing this is to diminish temporarily the return of venous blood to the right heart. This may readily be accomplished by venesection or peripheral venous stasis. Although this therapy is particularly beneficial in cases of left heart failure, it is also effective in conditions of general circulatory engorgement for obvious reasons. By reducing the circulating blood volume in the vascular system as a whole, it reduces the engorgement of the blood vessels in the lungs.

The second oldest therapeutic measure is the exhibition of morphine. In the labored breathing, and often tense anxiety, which is associated with pulmonary edema, the use of sedation for symptomatic relief naturally enters the mind of the therapist. The drug has been used on a pragmatic basis for many years. Recently, Harrison et al.[30] have given objective evidence of its therapeutic effectiveness. They have shown that morphine relieves the dyspnea, decreases the total pulmonary ventilation and increases the vital capacity. These effects are produced by diminishing the exaggerated respiratory movements which tend to increase pulmonary congestion and edema. The authors have reported an instance of one patient with pulmonary edema who had an arterial oxygen saturation of 56 volumes per cent. After the administration of morphine, the arterial oxygen saturation rose to 80 volumes per cent, and the patient's pulmonary edema disappeared. Reed and Teel[50] have recently shown the efficacy of morphine and venesection, or peripheral venous stasis, in patients with cardiac asthma and acute pulmonary edema complicating the toxemias of pregnancy.

The third therapeutic measure, which is commonly employed in pulmonary edema, is the inhalation of oxygen. When the patient is cyanotic, such therapy seems obviously indicated. Drinker[16] believes that at this stage the use of oxygen is far less effective than if it were employed earlier. He is convinced that the accumulation of fluid in the alveoli injures the capillary wall, in the direction of increased permeability, by cutting off the oxygen supply of the alveolar air from the pulmonary capillary endothelium.

The experimental evidence in support of this contention is not, to my

mind, clear-cut. Clinically, to be sure, oxygen should always be administered to a patient who shows any cyanosis and probably should always be given in the presence of engorgement of the pulmonary vascular bed, as evidenced by dyspnea, orthopnea, diminished vital capacity, etc. The administration of oxygen, however, is probably a less effective therapeutic measure than sedation, which diminishes exaggerated respiratory movements, and venesection, or peripheral venous stasis, which diminishes the output of the right heart.

It will have occurred to many of you that a method which would eliminate or reduce the negative intrathoracic pressure, which occurs with inspiration, and would, at the same time, increase the air pressure in the alveoli, thus offering slight resistance to the transudation of fluid, might be of value in the treatment of pulmonary edema. This has been termed "positive pressure" breathing. One of the earliest instances of the use of positive pressure in pulmonary edema was reported by Norton in 1897.[47] He applied intermittent positive pressure through a laryngeal tube in a patient who was moribund from edema of the glottis and of the lungs. The procedure was kept up for 15 minutes and was probably life-saving, although 48 hours elapsed before the pulmonary edema completely disappeared. In 1909, Emerson[20] observed that the normal respiratory variations in blood pressure were exactly reversed in animals when artificial respiration was carried out by the intermittent insufflation of air under positive pressure into the trachea. Furthermore, he made the enlightened observation that pulmonary edema in cats could be controlled by positive-pressure artificial respiration. In attempting to apply this observation to patients with pulmonary edema, he became confused and suggested that artificial respiration, administered by pressing upon the chest, might be of value. Such a procedure, of course, is in no way comparable to the intermittent insufflation of air into the lungs under positive pressure.

It was Poulton,[49] in 1936, who first reported the employment of positive pressure in the treatment of pulmonary edema in patients with left-sided heart failure. The positive pressure used amounted to between 5 and 15 cm. of water. It was given throughout both phases of respiration. He cautions against the use of too high a pressure, particularly if heart failure is generalized, because of the decrease in cardiac output, which results from the diminished venous return to the heart. Beecher et al.[6] also caution against the employment of positive pressure breathing in the presence of shock from hemorrhage because this procedure will further diminish an already dangerously low blood pressure. Clinically, of course, the occurrence of pulmonary edema in shock from hemorrhage does not occur except possibly as a terminal event.

In 1938, Barach[4] reported the value of positive pressure respiration in the treatment of acute pulmonary edema in patients and demonstrated that the pulmonary edema produced by adrenalin in rabbits could be prevented by the application of positive pressure respiration. He has devised a mask for clinical use which makes it possible to exert positive pressure during the expiratory phase of respiration. He has also described a helmet apparatus

which can be employed to administer positive pressure during both phases of respiration.[5]. Ansbro[1] discussed the rationale of positive pressure respiration, enumerating the following effects: (1) Positive pressure decreases the elevated negative intrapulmonary pressure in obstructive dyspnea. (2) The lumen of the smaller bronchioles becomes widened. (3) It diminishes the amount of blood trapped in the lungs by retarding the entrance of blood into the right heart. (4) It directly opposes the transudation of fluid from the pulmonary capillaries. He makes an analogy between artificial positive pressure respiration and the unconscious puffing of the cheeks and closing of the mouth seen in tired runners after great physical exertion or in excited people who have just escaped great danger. The effect of these involuntary acts is to increase the intrapulmonary pressure during expiration. He cites two instances in which patients with pulmonary edema were not relieved by the administration of oxygen but who were relieved when the oxygen was administered under pressure.

Positive pressure respiration has also been used clinically where there has been an injury to the pulmonary capillary endothelium. Carlisle,[10] in 1943, reported favorably on the use of oxygen and positive pressure during the expiratory phase of respiration in a large number of patients exposed to toxic industrial gases. Most of these patients were not suffering from frank pulmonary edema. Hardy and Barach[28] reported a favorable effect in an 11-year-old boy with pulmonary edema following poisoning by chlorine gas. Inhalation of 40 per cent oxygen in a tent did not control the edema, whereas 50 per cent oxygen with 4 cm. of water pressure on expiration was effective. In some unpublished observations Longcope and Luetscher treated 5 patients with pulmonary edema. In 4 of these patients, edema was due to phosgene poisoning; in the fifth, to chlorine. In all these patients, they were able to demonstrate a progressive improvement as evidenced by a rise in the systemic venous oxygen saturation with the employment of 40, 60 and 100 per cent oxygen, with a positive pressure of 6 to 12 cm. of water, applied during the expiratory phase of respiration. They remark upon the striking general improvement in the condition of these patients and the prompt disappearance of rales during pressure breathing. The increase in the arterial oxygen saturation with pressure breathing was attributed to the freer movement of the air in the lung because the increase in the partial pressure of oxygen was so small as to be negligible. It should be mentioned that favorable results with positive pressure breathing have not been obtained in animals exposed to phosgene poisoning.[8]

Helium mixed with 20 per cent oxygen has been suggested by Barach[2,3] for the treatment of patients with respiratory obstruction. Helium is of value in these conditions because its density is less than that of nitrogen and, consequently, less pressure is required to force such a gas mixture through a narrow orifice than is required with air. Breathing this mixture decreases the activity of the respiratory musculature and elevates the greatly decreased intrathoracic pressure, which occurs with respiratory obstruction. Where

there is no respiratory obstruction, helium and oxygen are of value in violent dyspnea, because under these conditions, where large volumes of air are moved rapidly, the smaller bronchioles offer appreciable resistance. Hence, the use of helium might have its place in the treatment of pulmonary edema by quieting respiration in a similar fashion to morphine.

Barach[5] has recently reported a mechanical device, within which patients may be placed for hours and in which no detectable respiratory movements of the chest or abdomen occur. Adequate ventilation of the blood flowing through the lungs is achieved by varying the pressure of the air in the lungs. Simultaneous variations of air pressure outside the body are also produced. When the pressure of the air surrounding the mouth and nose is increased, air will enter the lungs. When it is decreased again to atmospheric pressure, air will escape from the lungs. This will occur just as well whether there is any movement of the diaphragm and chest wall or not. Indeed, these respiratory movements of the chest and abdomen are prevented by an ingenious arrangement which produces a pressure over the chest and abdomen exactly equal to that in the lungs. The apparatus was devised for the treatment of patients with pulmonary tuberculosis in order to put the lungs completely at rest. I do not know whether such an apparatus has been employed clinically in the treatment of pulmonary edema. If it were so employed, it should produce valuable information upon the effect of the respiratory movements on pulmonary edema. Drinker[16] has shown that the respiratory movements are essential in producing a normal lymph flow from the lungs. Whether such cessation of respiratory movements might have a retarding effect upon the removal of edema fluid from the lungs remains to be determined.

Finally, if the apparatus for the artificial maintenance of the circulation devised by Gibbon[23] proves adaptable to clinical use, it could be used with value in pulmonary edema. Part of the blood could be withdrawn from the venous side of the circulation, oxygenated and reinjected into a systemic artery. This would decrease the amount of blood returning to the right heart and thus relieve the pulmonary congestion.

THE RELATION TO SURGERY

With advances in the art and science of anesthesia and a more widespread knowledge of fluid and electrolyte balance, protein and vitamin requirements, far more extensive surgical operations are being performed now than twenty years ago. A larger number of people are now living in the later decades of life. With the greater incidence of malignant disease in old age, it is natural to find that operations are not only more extensive but also are performed on older people. This age group is more prone to the cardiovascular disorders of coronary artery disease and hypertension. It behooves us all as surgeons, then, to be thoroughly familiar with the syndrome of left-heart failure and to be prepared to deal with it intelligently and energetically.

The widespread use of intravenous fluids, which has almost entirely replaced hypodermoclysis as a source of parenteral fluid in postoperative patients, has brought with it additional problems and dangers. One of the dangers is in overburdening the circulation in elderly people. Fluids such as blood and plasma must be used more cautiously from the standpoint of pulmonary edema than fluids such as dextrose solution, which pass easily into the tissues of the body and are readily excreted by the kidneys. Postoperative gastric dilatation, by its mechanical effects upon cardiac action, may be a significant factor in initiating pulmonary edema. The case reported by Eason and Karp[18] is an example of this, and in the patient reported by Fisher,[22] gastric dilatation may well have been a factor.

Finally, the subject of pulmonary edema is probably of greatest interest to the thoracic surgeon. He performs long operations with the chest open, using positive pressure respiration. Intravenous fluids are almost always necessary both during and after operation. Not infrequently, large portions of functioning pulmonary tissue are removed. Half of the pulmonary tissue is removed in pneumonectomy and considerably more when both lower lobes are removed for bronchiectasis. Whenever any pulmonary tissue is removed, an added burden is imposed on the lung that remains. Because of the reserve capacity of the lungs, such a reduction in functioning tissue may be without appreciable effect under conditions of rest. With stress, such as the result of exercise, the reduced capacity of the lungs becomes evident. Again, if intravenous fluids are administered too enthusiastically, particularly plasma and blood, pulmonary engorgement and edema may result. Gibbon et al.[25] have shown that in cats the removal of 71 per cent of the functioning pulmonary tissue is compatible with life. Some pulmonary engorgement results but not pulmonary edema. However, when an amount of plasma or blood which is well tolerated by normal animals is administered to those with only 29 per cent of functioning lung tissue remaining, pulmonary edema and death result. Thornton et al.[57] have pointed out that large blood transfusions are well tolerated by dogs following the resection of lung tissue if certain precautions are observed, and it is a common clinical experience that large amounts of blood may be given without ill effect during thoracic operations in which pulmonary tissue is resected.

It should always be borne in mind that any extensive resection of pulmonary tissue, such as pneumonectomy, will produce some engorgement in the remaining lung. As pneumonectomies for carcinoma are most frequently performed in elderly patients, the possibility of left-heart failure should always be remembered. Pulmonary edema from any cause is more prone to occur in someone with one lung than with two.

Crafoord[13] has made the interesting observation that pulmonary edema not infrequently occurs in elderly patients about the third or fourth day after pneumonectomy and that such edema can often be relieved by aspiration of fluid from the pleural cavity on the operated side of the chest. The explanation of this therapeutic effect probably is that the mechanical pressure of the

fluid upon the heart interferes with its action and with the entrance of blood from the pulmonary veins into the left auricle. I have had the opportunity of observing the occurrence of pulmonary edema in the remaining lung of an elderly patient four days after pneumonectomy for carcinoma. The patient was comfortable when sitting upright, a position which diminishes the return of blood to the right heart. When lying flat in bed, being bathed by a nurse, the patient coughed continuously, bringing up white frothy material. Rales could be heard throughout his remaining lung. In the erect position, the coughing ceased, but rales could still be heard over the dependent portions of the lung. Aspiration of bloody fluid from the operated side of his chest and replacement with air resulted in the complete disappearance of rales and the patient could lie flat in bed thereafter without signs of pulmonary edema.

I have purposely avoided a discussion of the influence of nervous factors on pulmonary edema. The experimental and clinical reports on this subject are conflicting. However, certain extensive operations within the thorax may markedly interfere with the innervation of the lungs. I refer to such operations as resection of the lower three-quarters of the esophagus with esophagogastrostomy anterior to the aortic arch. This procedure, in which the esophagus is mobilized above the aortic arch, may result in almost complete denervation of the lungs. Both vagi, of course, are divided and removed with the esophagus, and where the growth is extensive and the dissection difficult, both the pulmonary branches of the vagi and the sympathetic innervation of the lung may be interfered with.

Afferent and efferent impulses pass over both the parasympathetic vagal fibers and over the sympathetic nerves. Whether an almost complete denervation of the lung may result in pulmonary edema remains to be determined, although Rienhoff[52] has reported performing total denervation of both lungs by resecting the posterior pulmonary plexus without the occurrence of pulmonary edema.

Many problems in the pathogenesis and treatment of pulmonary edema remain to be solved. The surgeon who concerns himself with geriatrics, or who opens the thorax, must of necessity be thoroughly versed in the physiologic mechanisms involved in the movement of fluid through the pulmonary capillary wall.

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ANNUAL ADDRESS FOR 1947

CANCER OF THE COLON AND RECTUM

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Cancer of the colon and rectum is one of the most preventable and curable of malignancies and is by far the most curable of the common tumors of the gastro-intestinal tract. This statement is made with several good reasons in mind. Colonic cancer is preventable in a high percentage of cases because we know more about the lesions that lead to this type of cancer than we do of most malignancies. Treatment of these lesions is a logical and successful prophylactic measure. Colonic cancer should be one of the most curable of malignancies because (1) it produces symptoms that are highly typical and suggestive; (2) its presence may be detected by relatively simple diagnostic procedures; (3) the tumor may be removed without producing any marked disturbance of body physiology; and (4) with modern anesthesia, preoperative preparation and postoperative care, operation for cancer of the large bowel can be performed with a minimum of risk.

PROPHYLAXIS OF CANCER OF THE LARGE BOWEL

There is good evidence that most cancers of the large bowel develop from adenomatous polyps. The high incidence of malignant degeneration and of multiple malignancies in the colon of patients with diffuse polyposis or with multiple polyps is well known. The association of benign polyps and full-blown cancer is a common finding. Early carcinomatous change is seen frequently in a portion of an adenomatous polyp, the remaining part of which is benign. The frequency of polyps is surprising when figures such as Feyrter's[1] are considered. In 1,800 autopsies, he found 1,110 colons that contained polyps. He showed that polyps increased in frequency with advancing age as follows:

AGE	
45 to 54	⅓ of patients had polyps
55 to 64	½ of patients had polyps
65 to 74	⅔ of patients had polyps
75 to 87	¾ of patients had polyps

By far the majority of polyps of the large bowel are located in the rectum and sigmoid, which is also the most common site for cancer. Miller, Day and L'Esperance[2] found polyps in 6.4% of 7,494 patients on routine proctoscopy. The relative importance of proctoscopy over barium enema study in 479 polyps is emphasized by the fact that only 2 per cent of the total were demonstrated by roentgenographic findings alone. They point out that many small polyps that have undergone malignant degeneration may not have

ulceration producing bleeding on slight trauma, and, as a prophylactic measure, they recommend that every individual over 45 should have a routine proctoscopic examination.

The usual symptoms that suggest the presence of a polyp are indefinite lower left sided discomfort and blood in small but definite amounts in the stool. On proctoscopic examination the finding of blood clots or blood streaks in the mucus adherent to the bowel wall should make one suspicious of the presence of a polyp in the area above that visualized by the proctoscope. A barium enema study, especially using the double contrast technic, frequently will show the polyp in outline. Repeated studies may be necessary before the polyp can be demonstrated. The finding of one polyp should lead to a search for others, because more than one polyp is found in at least 50 per cent of the cases. The treatment of colonic polyps is simple, successful and safe. Those located in the area that can be visualized through the proctoscope can frequently be destroyed by electrocoagulation or, if they are pedunculated, they can be removed by an electrocoagulating snare. Polyps in the area of the sigmoid or above can be removed by simple colotomy or by resection and anastomosis if there is a question of malignancy.

A second prophylactic measure against cancer of the bowel deals with the chronically irritated colon of the patient with ulcerative colitis. The incidence of carcinoma in the chronic ulcerative colitis varies in reported figures. Cattell has pointed out the high incidence of cancer in ulcerative colitis of more than 9 years' duration. This shows the necessity of frequent examination of these colons by roentgenography, and, when the patient comes to surgery, we feel that a colectomy should be done either as a primary or subsequent procedure, not only as a treatment of the ulcerative colitis but also as a prophylaxis against malignant degeneration.

THE DIAGNOSIS OF CANCER OF THE COLON AND RECTUM

The cure of cancer of the colon and rectum depends to a large extent upon early diagnosis, because the curability of malignancy of the bowel depends largely upon whether or not the patient is operated upon while the growth is still a local one which can be completely removed.

Early diagnosis requires first an education of the laity concerning symptoms that are signs of large bowel cancer. Too often the symptoms (change in bowel habit, constipation or diarrhea, indefinite abdominal pain or discomfort, blood and mucus in the stools) are disregarded or looked upon as a natural change that appears with advancing age. Or patients often become reticent about an examination of the rectum or bowel because of a false modesty or a fear of being hurt.

Early diagnosis requires a knowledge of the early symptoms and an index of suspicion sufficient to make the attending physician investigate the patient thoroughly. The critical diagnostic methods, digital examination, proctoscopy, barium enema, and a stool examination, are simple, easily available and will give an accurate diagnosis in at least 90 per cent of the cases.

Cancer of the Right Colon

Cancers of the cecum, ascending colon and hepatic flexure represent about 11 or 12 per cent of malignancies of the large bowel. In their early stages, they produce chronic digestive disturbances and right-sided abdominal pain or discomfort in from 80 to 90 per cent of the cases. These symptoms are often mistaken for those of other more common right-sided lesions, such as cholecystitis or subacute or chronic appendicitis. Mayo[3] and Ransom[4] point out that from 10 to 15 per cent of patients with right colon tumors have been subjected to appendectomy within the duration of the symptoms of the malignant process. This emphasizes the necessity for exploration of the right colon when operating for a so-called subacute appendicitis in a patient of 40 or beyond.

The second group of symptoms produced by right colon cancers is weakness, weight loss and fatigability, associated with a severe secondary anemia. An unexplained anemia in a patient past 40 should lead to a consideration of right colon malignancies as a cause.

The third common symptom is the presence of a slightly tender mass in the right abdomen.

Although a change in bowel habit or in the character of the stools is usually considered a common symptom in bowel malignancy, these symptoms may be absent in at least half of the cases of right-sided tumors. When they are present they are significant, but their absence does not exclude the presence of a right colon cancer.

The critical diagnostic procedures are the palpation of a right abdominal mass and the demonstration of a defect in the right colon by barium enema study.

The roentgen diagnosis is accurate in approximately 90 per cent of the cases, but the interpretation of the findings in the cecal area may be difficult and require repeated examinations.

Cancer of the Transverse Colon

Tumors of the transverse colon represent about 10 per cent of bowel cancers. They produce symptoms more likely to focus attention on the large gut. Pain of intermittent or colicky type relieved by the passage of gas or stool indicates the common obstructive nature of these tumors. The attacks of pain often appear when peristalsis has been stimulated by the ingestion of food. A change in bowel habit, increasing constipation or diarrhea, associated with bloating, "gas" or borborygmus are other early symptoms. In about half of the cases a tumor may be palpated when the patient first comes for examination.[5] Blood in the stools, loss of weight, anorexia and nausea are frequent but less diagnostic symptoms.

The critical diagnostic method is the barium enema study.

Cancer of the Descending Colon and Sigmoid

In the descending colon (10 per cent) and sigmoid (22 per cent) are found about a third of the cancers of the large gut. In this area the intestinal contents become more solid, and, since the typical cancer is one which encircles and obstructs the gut, the symptoms are usually quite characteristic. Pain of dull aching type associated with acute exacerbation of crampy discomfort is the common complaint. Increasing constipation and, in a fair percentage of cases, complete obstruction may be the presenting symptoms. Change in the character of the stools, blood, mucus or liquid evacuations may be other early symptoms which are frequently neglected or misdiagnosed as hemorrhoids, mucous colitis, and the like. On abdominal examination a mass may be palpated, and in about a third of the cases, there is an associated tenderness in the lower left abdomen. In a few cases an extrarectal mass may be palpated on digital rectal examination. The diagnosis may be strongly suspected if blood-streaked mucus is present in the bowel coming from above the area that can be inspected through the proctoscope.

The barium enema study is the critical diagnostic method.

Cancer of the Rectum and Rectosigmoid

Cancer of the lower bowel from the anal opening to the promontory of the sacrum represents about half (47 per cent) of all cancers of the large bowel. The group of symptoms produced by these tumors is easily recognized if one considers that the ulcerating cancer is developing in the narrowest part of the bowel, in a part of the bowel where the colon contents are most solid and formed. Because the lesion is constricting, increasing constipation occurs, often associated with what the patient terms "diarrhea." The diarrhea is simply the evacuation of irritating mucus and necrotic discharge from the ulcerating tumor that collects in the ampulla of the rectum. In a typical case, the patient may volunteer the information that as soon as he arises, he has to pass a liquid mucous stool. After breakfast he may have a normal movement, and then several times during the day a mucous stool, often associated with considerable tenesmus. Trauma to the carcinomatous ulcer by the passage of the intestinal contents over it, causes oozing or frank bleeding. The amount and color of the blood varies with the degree of bleeding and the time the blood remains in the rectum.

These two symptoms, a change in bowel habit and bleeding from the rectum, spell cancer in capital letters, especially in the patient past 40 years.

Cancer of the rectum and rectosigmoid should be diagnosed on the first visit of the patient to the doctor in almost every case. A simple digital examination of the rectum is sufficient to make the diagnosis. This is an office procedure that requires no apparatus except a glove or finger cot. The diagnosis may be confirmed by visualization of the tumor by the proctoscope. By these two simple procedures a definitive diagnosis can be made in 100 per cent of the cases. A biopsy may be taken if desired, but it is the rare case in which the diagnosis cannot be made by inspection and palpation. More reliance

should be placed upon the clinical examination than upon the pathologic report. The finding of anal lesions such as fissure, fistula or hemorrhoids, does not alter the necessity for a complete examination of the rectum and rectosigmoid.

Cancer of the rectum and rectosigmoid does not require and should not have a barium enema study for its diagnosis. This is an area in which the barium filling of the bowel does not readily show defects in the bowel outline, and frequently a negative x-ray report and a lack of a digital examination of the rectum have led to delay in diagnosis and treatment. It is my belief that no patient should have a barium enema study who has not first had a digital and proctoscopic examination of the rectum and rectosigmoid.

TREATMENT OF CANCER OF THE COLON AND RECTUM

Before discussing specific types of treatment it may be well to place before you certain principles concerning the treatment of cancer of the large bowel.

1. Surgical extirpation of all the cancer is the only known way of producing a cure. Fortunately, extirpation of large parts of the colon is not only compatible with life but also with normal function. In order to remove all the cancer, the bowel containing the tumor must be removed as well as the paths of lymphatic spread.[6] Since the lymphatics lie close to the blood vessels of the colon, removal of the lymph nodes and channels means also ligation of the blood supply to a large area of bowel. Hence, to remove all of the tumor and the area of lymphatic spread requires the removal of a much longer section of bowel than simply that containing the carcinoma.

2. When obstruction is a finding in cancer of the bowel, the obstruction should be relieved before making a radical attack on the tumor. To attempt radical removal of an obstructing cancer of the bowel is to invite disaster. Fortunately, this complication of colon cancer is found usually in tumors of the distal bowel, so that relief of the obstruction can be accomplished by fecal diversion with a transverse colostomy or a decompression by cecostomy without interfering with a later radical resection of the tumor. A Miller-Abbott tube is usually not effective in decompression of large bowel obstruction.

3. When all of the tumor cannot be removed, because of liver metastasis or local irremovable spread, the most effective palliation is obtained by a resection of the necrotic, bleeding, obstructing tumor mass. Even though a cure is not possible, removal of the carcinoma frequently gives the patient many months and, at times, years of useful life.

4. Operations for cancer of the large bowel can be performed more safely if the patient is adequately prepared for operation. Adequate preparation may be divided into two parts, general and local. The general or systemic preparation implies a restoration of fluid and protein balance, a restoration of normal blood volume and blood count, measures which should be a part of the preparation of any patient for major surgery.

The local preparation consists of measures to produce an empty bowel at operation. These include a low residue, high caloric diet, and daily enemas for 3 to 4 days before operation. Some surgeons advise daily saline cathartics instead of enemas, but they are not recommended because they tend to deplete the patient.

A consideration of bowel antiseptics is in order in a discussion of colon surgery. Aureomycin, terramycin, streptomycin, sulfasuxidine and sulfathalidine are all used. They are valuable in that they reduce the bulk and solidity of the stools, and therefore help to produce an empty bowel. There is no question but that they reduce the bacterial content of the stools if given for 3 to 4 days before operation. In our experience, however, local or peritoneal bacterial contamination is not a factor in the morbidity or mortality in operations for cancer of the colon unless there is a continuing leak of intestinal contents due to a technical error.

5. Preoperative intestinal intubation is an effective way to avoid the most frequent postoperative complication—intestinal obstruction. The tube should be introduced and allowed to pass well down the gut before operation. This aids in handling the small gut at operation. Postoperatively, continuous suction on the tube removes swallowed air and decompresses the gut until peristalsis is resumed. The tube should remain in place until the passage of gas indicates a return of normal peristalsis.

6. Adequate anesthesia supervision and adequate blood replacement are important factors in safe colon surgery. To be adequate, the anesthesia must produce a quiet flaccid abdomen; but just as important as the anesthesia is its supervision, to prevent falls in blood pressure and periods of anoxemia which endanger the recovery of the patient.

The transfusion of blood during the operation is not only a method of maintaining blood pressure but is also important in replacing blood as it is lost during the operative procedure. Thus the patient leaves the operating room with the same oxygen-carrying hemoglobin that he had when he came to it.

7. Careful postoperative supervision permits early recovery and does much to prevent postoperative complications. The important points in the postoperative care may be enumerated briefly as follows:

(A) Maintain adequate fluid intake parenterally until peristalsis returns. The fluids used may be 5 per cent glucose solution, saline solution or solutions of amino acids. Usually, 3,000 cc. daily is sufficient, divided as follows:

5 per cent glucose—1,500 cc.
Saline—500 cc.
Amino acids—1,000 cc.

(B) Give penicillin prophylactically—300,000 u. daily. This reduces the danger of pulmonary and other infections and makes a smoother recovery.

(C) Get patient out of bed early. Early resumption of normal activity

improves the metabolic function and helps to prevent pulmonary and thrombotic complications.

(D) Resume normal diet as soon as patient desires. Usually a solid diet will be well tolerated by the third or fourth day. There is no virtue in a liquid or soft diet after peristalsis is resumed and the intestinal tube has been removed. The patient's nutrition and morale both benefit by a normal diet high in calories and proteins, but perhaps low in residue.

In an experience with 246 cases of cancer of the colon and rectum, all but 13 were subjected to laparotomy. On these 13 cases, 9 were cases in which a carcinomatous polyp was removed by electrocoagulating snare, 3 cases of bleeding inoperable carcinoma of the rectum treated by electrocoagulation, and 1 case treated by local excision.

Of the 233 cases subjected to laparotomy, the tumor was resected in 197 (84 per cent of the operated cases); in 31 cases (13 per cent), a colostomy or ileotransverse colostomy was performed; and in 5 cases an exploration and biopsy was performed. There were 17 operative deaths in the 246 cases treated, a mortality of 6.9 per cent.

In 99 cases (40 per cent of the total number), palliation was all that could be done for the patient. This means that in 2 out of every 5 patients with carcinoma of the colon and rectum, the tumor had already metastasized to the liver, or there was such widespread local invasion that there was no hope for cure. In 60 (or 3/5 of these cases) it was possible to resect the primary growth as a palliative procedure. Such an operation relieves the patient of the danger of impending obstruction and removes the bleeding infected ulceration which plays a great part in the anemia and protein depletion which otherwise occurs.

Operations for Cancer of the Right Colon

Thirty carcinomas of the right colon were divided as follows: cecum, 18; ascending colon, 8; and hepatic flexure, 4. All of the patients were operated upon, but operation for cure could be expected in only 14 cases (46.7 per cent). In 16 cases (53.3 per cent) it was recognized that operation was for palliation only. The growth was resected in 26 cases (83.3 per cent). In 22 cases the resection was accompanied by a primary open end-to-end anastomosis between the terminal ileum and transverse colon. There were no deaths or complications attributable to this procedure and we look upon this type of operation as the operation of choice for malignancy of the right colon. In none of these cases was a complementary jejunostomy performed. There were 4 operative deaths (13.3 per cent), all of which occurred in patients operated upon for palliation.

Operations for Cancer of the Transverse Colon, the Left Colon and the Sigmoid

There were 77 carcinomas of the transverse colon, left colon and sigmoid. All of the patients were operated upon, for cure in 45 (59.7 per cent), and

for palliation in 27 (40.3 per cent). The tumor was resected in 70 patients—90.0 per cent of the cases. In 49 of the resections a primary end-to-end anastomosis was performed—66.6 per cent of the resected cases. In 12 cases (17 per cent) the Mickulicz type of operation was employed. A previous colostomy or cecostomy had been employed for the relief of intestinal obstruction in 4 cases. In none of the cases was a cecostomy or colostomy performed complementary to a resection and anastomosis. A resection with primary anastomosis is the ideal operation for this part of the colon. There were 3 operative deaths, an operative mortality rate of 3.9 per cent. There were no deaths attributable to the type of operation employed.

Operations for Cancer of the Rectosigmoid and the Rectum

There were 144 cases of carcinoma of the rectosigmoid and rectum. Of these, 88 cases (54 per cent) could be operated for cure, but in 56 cases (46 per cent) only palliation could be expected. The tumor was resected by a local operation (electrocoagulating snare) in 10 (7 per cent) of the cases. A radical operation for removal of the tumor was performed in 106 (74 per cent) cases. In 28 (19 per cent) cases, no attempt was made to resect the tumor. In 71 (70 per cent) cases, a Miles type of abdominoperineal resection with end colostomy was performed, and in 24 (16.7 per cent) cases, an anterior resection with primary anastomosis of rectum to sigmoid was employed without complementary colostomy or cecostomy. We believe that this type of operation may be employed in many cases of carcinoma located above the peritoneal reflection and that it is particularly indicated when liver metastasis is present so that palliation only can be expected. In the 144 cases there were 10 deaths, a hospital mortality of 7 per cent.

Time does not permit, and I am not prepared to give a discussion of the prognosis in cancer of the colon and rectum, but in a general way it may be said the five-year survival rate of resected cases is approximately 45 to 50 per cent in carcinoma of the rectum and between 55 and 60 per cent for cancer of the remaining colon.

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STUDIES ON THE FORMATION OF PERITONEAL ADHESIONS

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During the past few years, a number of the problems of intestinal obstruction have been the subject of studies carried on in the Harrison Department of Surgical Research at the University of Pennsylvania. Although this work is still in progress, this seemed an appropriate time to summarize the data obtained thus far on the problem of adhesions. Many individuals have taken part in the work, and Doctors Jacob Chandy, Clifton Portnoff, Keith Reemstma and George Jackson have carried on various phases of it more or less independently.

Intestinal obstructions due to bands and to adhesions are usually considered together because at times it is impossible to distinguish them. In the clinical series reviewed by Reemstma at the Hospital of the University of Pennsylvania, 48 of 60 cases had had a previous operation. In the report of Koch[1] from Lund, Sweden, only two-thirds of the patients had undergone a previous laparotomy. The same author gave the incidence of obstruction due to adhesions following various operations shown in Table 1. He arbitrarily excluded cases developing within three weeks of operation because of the factors in the early postoperative period which tend to produce paralytic ileus. He noted that obstruction due to adhesions almost never followed operations confined to the biliary tract. This is corroborated by the experience at the Hospital of the University of Pennsylvania, where only one such case was encountered in ten years.

TABLE 1. INCIDENCE OF OBSTRUCTION DUE TO ADHESIONS FOLLOWING VARIOUS OPERATIONS (Koch)

	PERCENTAGE
Appendectomy without drainage	0.2
Appendectomy with drainage	2.0
Gynecological laparotomies	0.3

When we compare the occurrence of obstructing adhesions with the incidence of all adhesions following laparotomy, it becomes evident that obstruction is a comparatively rare complication of adhesion formation. W. L. Green,[2] writing in the Journal of the Indiana Medical Association in 1946, cites a personal series of 50 patients in whom he had performed a laparotomy after a previous operation. Adhesions were found in 48 patients, or 96 per cent.

The tendency for adhesions not to cause obstruction is suggested also

TABLE 2. INTESTINAL OBSTRUCTION WITH ADHESIONS. Hospital of the University of Pennsylvania (1935-1945—60 cases). Time interval: last operation and onset of illness

TIME INTERVAL (LAST OPERATION AND ONSET)	NUMBER OF CASES	PERCENTAGE OF TOTAL CASES (48)
3 days to 6 months	12	25
6 months to 2 years	14	29
2 to 5 years	7	15
5 to 35 years	15	31
Average interval, 5.4 years	48*	100

*Only 48 of the 60 patients in this series had had previous surgery.

by the time interval frequently noted between the original operation and the onset of acute obstruction. Koch[1] found an average of almost four years. The figures from the Hospital of the University of Pennsylvania are shown in Table 2. The average interval was 5.4 years.

On clinical grounds then, it would appear that the incidence of adhesions was high but that they resulted in obstruction in only a small proportion of instances and then often only after long intervals from the probable time of their formation. Mortality figures collected from the literature during the past three decades are summarized in Table 3. Not only has the mortality been high but improvement has not been as rapid as in many other types of surgery.

TABLE 3. REPORTED MORTALITY IN ACUTE OBSTRUCTION DUE TO BANDS AND ADHESIONS*

AUTHOR	YEAR	NUMBER OF CASES	MORTALITY
Braun	1922	160	36%
Moss and McFetridge	1927	46	65%
Moss and McFetridge	1932	91	35%
Miller	1929	98	57%
Vidgoff	1930	170	38%
Cornell	1930	110	42%
Holden	1930	64	22%
Koslin	1932	26	35%
Rochkind	1938	108	32%
Scudder et al.	1938	573	37%
F. Koch	1939	156	25%

*Adapted from Fredrik Koch, Acta. Chir. Scandinav., Supp. 88, 90:1, 1944.

Laboratory experiments in the Harrison Department were begun in 1945, not with the aim of finding an agent that would prevent adhesions, but in the hope of reaching a sounder understanding of the factors which provoke them so that surgical technic could be better planned to minimize adhesion formation.

Among the questions to which answers were sought were the following: (1) How much mechanical trauma is necessary for the production of adhesions and is the response subject to considerable individual variation? (2) Are mild degrees of thermal trauma likely to produce adhesions? (3) Are mild degrees of chemical trauma likely to produce adhesions? (4) Which suture materials are inert and which, if any, may lead to adhesion formation? (5) Does the placement of sutures influence adhesion formation? (6) Which glove powders cause adhesions and what quantities are required? (7) How easily are adhesions produced by injections of bacteria? (8) Does blood in the peritoneal cavity cause adhesions or prevent them? (9) Are breaks in the serosa better left alone or should they be reperitonealized by suture? (10) Will heparin, papain, intraperitoneal saline solution, or gelatine solution prevent adhesion formation? (11) Do adhesions persist indefinitely and are those which result from talc more likely to persist than those resulting from mechanical trauma? (12) How does the omentum become attached to a point of injury?

The Wistar rat was selected for these experiments. These animals were available with a minimum of individual variation and they could be used in sufficiently large numbers to obtain statistically significant groups when this seemed important. The data obtained has been arranged as it bears on each of these twelve questions, and the methods used will be referred to subsequently. Adhesions forming to laparotomy wounds were discounted. It was difficult to control the technical factors accurately enough to eliminate them completely.

Table 4 shows results with a variety of types of mechanical trauma. In general, the depth and area of the injury seemed to be the determining factors in the proportion of adhesions formed. These adhesions were usually single and consisted of an attachment of either the omentum or the mesosalpinx (or mesotestis) to the area of injury.

TABLE 4. ADHESION FORMATION: EFFECT OF MECHANICAL TRAUMA

	NUMBER OF RATS INJURED	ADHESIONS TO INJURED AREAS AFTER 3-6 DAYS	PER CENT
Careful serosal stripping from all of cecum	20	0	0
Gauze scarification 10X	10	3	30
Gauze scarification 20X	25	19	76
Multiple Allis forceps bites—cecum	25	19	76
Payr clamp—cecum	31	29	94

In regard to thermal injury, some preliminary experiments on the effect of heat were done by Portnoff[3] who found that application of compresses at 55°C. to the bowel of the rat produced adhesions in one of nine animals.

Several chemical irritants were tested by Chandy.[4] I shall refer to only two—tincture of iodine and sodium hydroxide. Iodine has been implicated as a cause of adhesions by Figueroa[5] in 1942. In the rat, Chandy[4] found that

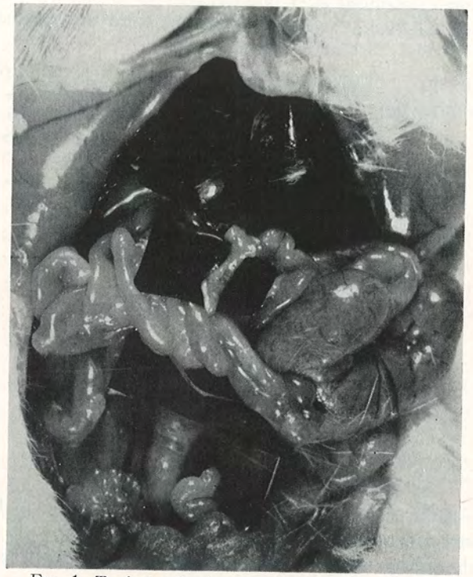

FIG. 1. Typical adhesions produced in the peritoneal cavity of a rat by the injection of 0.05 cc. of 3 per cent sodium hydroxide. Black paper has been placed behind the adhesions for photographic purposes.

0.05 cc. of a seven per cent solution produced adhesions in three of ten animals and that the injection of larger amounts resulted in a mortality of over 40 per cent. Sodium hydroxide, while not of much clinical interest, was useful because the method could be standardized accurately and this, in turn, made possible certain observations on the rate of adhesion formation and on the duration of adhesions resulting from its use. See Table 5. Representative adhesions due to 0.05 cc. of three per cent sodium hydroxide are shown in Figure 1. At autopsy, it appeared that the formation of adhesions occurred, by and large, where they were needed to prevent peritoneal soiling. Sections of bowel, injured by 2 per cent sodium hydroxide, showed the injury to be confined largely to the serosa, and no adhesions formed. With stronger solutions, which produced adhesions, it frequently was found that forcible separa-

tion of the adhesions would tear out a segment of bowel wall which appeared to be devitalized.

In starved rats, in which sodium hydroxide was tolerated so poorly that 40 per cent of the animals died even with 0.05 cc. of the one per cent solution, adhesions formed in all of the survivors, although this dosage produced none in healthy animals. Using 0.05 cc. of the 3 per cent solution, it was found that of 15 animals sacrificed after one day, one-third had adhesions. On the second day, one-half had adhesions, and on the third day, all had adhesions.

TABLE 5. ADHESION FORMATION: EFFECT OF SODIUM HYDROXIDE (Chandy)

CONCENTRATION	AMOUNT INJECTED	NUMBER OF RATS	PER CENT DIED	PER CENT OF SURVIVORS FORMING ADHESIONS
10 per cent	0.15 ml.	1	100	
	0.10 ml.	1	100	
	0.05 ml.	1	100	
5 per cent	0.15 ml.	1	100	
	0.10 ml.	6	66	100
	0.05 ml.	10	30	100
	0.03 ml.	25	20	100
	0.02 ml.	30	0	90
3 per cent	0.05 ml.	30	3.9	100
2 per cent	0.05 ml.	15	0	53
1 per cent	0.05 ml.	10	0	0

From all of this data on injury to the bowel, it is evident that there are wide individual variations, a few animals forming adhesions in response to a fraction of the injury necessary to produce adhesions in all animals. None of the mechanical methods of producing trauma caused adhesions to form in every case.

The influence of suture materials on adhesion formation was considered next. To avoid the concomitant influence of needle wounds in tissues, series of rats were run in which six-inch lengths of various materials were rolled into a ball and placed free in the peritoneal cavity by laparotomy. The results are summarized in Table 6. These findings agree with the findings of many earlier workers in the field and serve to re-emphasize the role of catgut in the formation of adhesions. In this connection, it is well to recall the work of Gilson and Gratia,[6] who showed that rabbits could become sensitive to catgut and that subsequent use of catgut in the abdomen might result in intense reactions with adhesion formation and even intestinal obstruction. Such catgut sensitivity was believed by Kraissl and Meleney[7] to be a factor in premature absorption of sutures resulting in wound dehiscence. Vaccaro and his associates[8] skin tested large groups of children. Unoperated groups with and without an allergic background were compared with similar groups that had

TABLE 6. ADHESION FORMATION: EFFECT OF 6-INCH LENGTHS OF VARIOUS SUTURE MATERIALS AS FREE FOREIGN BODIES IN THE PERITONEUM OF RATS

	NUMBER OF RATS	AUTOPSY FINDINGS AFTER 5 DAYS
Silk No. 00	5	The silk is encapsulated by the omentum in all cases.
Silk No. 3	5	The silk is encapsulated by the omentum in all cases.
Cotton—fine	5	The cotton is encapsulated by the omentum in all cases.
Cotton—coarse	5	The cotton is encapsulated by the omentum in all cases.
Catgut—plain No. 000	5	Extensive matting of the bowel and omentum.
Catgut—plain No. 3	5	Extensive matting of the bowel and omentum.
Catgut—chromic No. 0	5	Extensive matting of the bowel.

been operated on. The latter showed a moderately higher incidence of positive reactions to catgut. Stainless steel and tantalum wire caused the least reaction of the various materials used. In several rats such wire was found lying free, having gravitated, as a rule, to the scrotal pouches in the males. In so far as these observations can be applied to man, they suggest that catgut is not an ideal suture material for reperitonealizing areas in which one is particularly anxious not to get adhesions.

Not only is the suture material a factor, but the manner in which it is used is a factor. Strangulation of tissue, of course, leads to necrosis, and the necrotic material in itself is a foreign body and stimulates adhesion formation. However, the depth at which a suture is placed in bowel wall seems to be a factor. Thus, if silk is introduced subserosally and the ends are clipped off, no adhesions are likely to form. On the other hand, if such a suture is introduced more deeply, so that it penetrates the muscular layer and is tied, adhesions usually result. Whether or not there are bacteriologic factors that enter into this has not yet been determined.

The role of glove powders in adhesion formation was studied next. This is an old subject and one to which we have all listened. Years ago, lycopodium

TABLE 7. ADHESION FORMATION IN THE RAT: EFFECT OF GLOVE POWDERS

POWDER	AMOUNT IN MG.	NUMBER OF RATS	PER CENT DEVELOPING ADHESIONS
Talc	10	4	75
	25	5	100
Bio-Sorb	50	6	0
Modified Bio-Sorb	25	4	100
	50	2	100
	200	4	100
Modified Bio-Sorb with MgO	50	2	100
	200	4	75
Potassium bitartrate	50	5	0

spores were used as a dusting powder but they were shown to be a definite cause of adhesion formation. Talc was substituted, but the evidence is now overwhelming that it too causes adhesions. Furthermore, of all the methods we have employed experimentally, the introduction of talc is the one that produces the most widespread matting together of the bowel loops, simulating the picture one sees from time to time in patients with postoperative obstruction. Of the many authors who have written on this subect, Seelig[9] has pro-

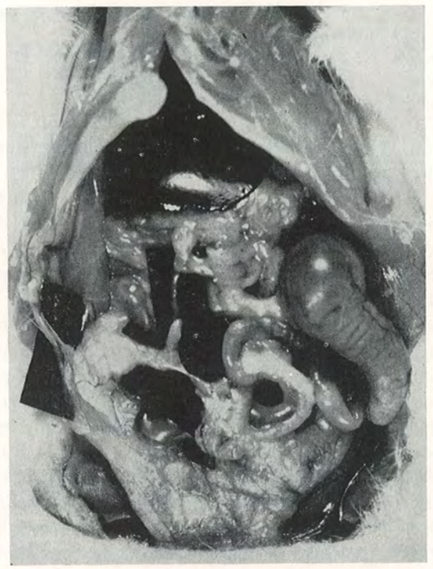

FIG. 2. Intraperitoneal adhesions in the rat which resulted from the introduction of 10 mgm. of glove talc.

duced perhaps the strongest evidence. He has shown that talc will produce adhesions in the experimental animal and that the amounts that can be introduced by gloves, if a finger tip gets punctured, are sufficient to produce adhesions experimentally and he has demonstrated crystals, having the characteristic appearance of talc under polarized light, in adhesions resected from patients who have been operated upon previously.

We are not in a position to say how large a role talc plays in clinical adhesions but the evidence strongly suggests that it is a factor and quite possibly an important one. The experimental evidence is striking. Table 7 tabulates the results of experiments with glove powders in the rat and compares the results with talc, potassium bitartrate, and with a modified starch

powder sold by Johnson and Johnson under the trade name of "Bio-Sorb." Representative adhesions produced by 10.0 mg. of talc are shown in Figure 2.

Potassium bitartrate is ideal from the standpoint of adhesions, but it is very hard on rubber gloves and has a somewhat gritty feel as compared to talc. Seelig[9] maintains that autoclaves operated at exactly fifteen pounds per square inch of steam pressure shorten the life of the gloves 50 per cent. At

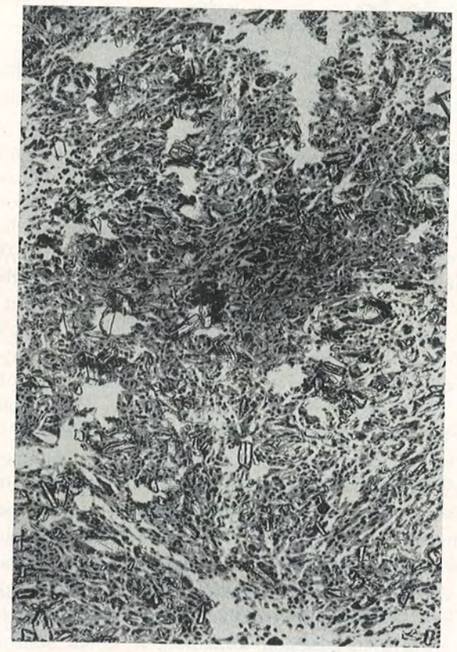

FIG. 3. Photomicrograph of a section through an intraperitoneal adhesion showing talc crystals.

the Hospital of the University of Pennsylvania, in the operating-room autoclaves, which operate at 17 pounds per square inch, the gloves were usually ruined after two sterilizations.

Bio-Sorb does very well from the standpoint of adhesions but forms a slightly sticky layer on the gloves which makes it somewhat difficult to thread sutures. Its physical characteristics in the dry state are excellent, and it is probably the best material available today. Johnson and Johnson submitted several modifications of this material but in each instance when the physical characteristics of the material were improved it began to produce adhesions. The gelatine powders which we tried caked on autoclaving. Figure 3 shows

Table 8. Study of the Influence of Oversewing on the Formation of Adhesions After Serosal Injury

	Number of Animals	Percentage With Adhesions
Denuded Area Oversewn	48	79
Denuded Area Left Bare	47	31.9

a photomicrograph through an adhesion produced by talc. The crystals of magnesium silicate are easily seen. Seelig[10] terms this "silicosis of the abdomen."

Infection is commonly thought of as an important factor in adhesion formation. In most clinical peritonitis there are foreign body factors such as necrotic tissue, alimentary-tract contents, etc., which may also act as stimuli for adhesion formation. To avoid these secondary factors as far as possible, Chandy[11] injected cultures of *E. coli* and *Streptococci,* 50,000,000 organisms each, intraperitoneally in each of five rats. After five days, the peritoneum was clean and no adhesions were found. Five other rats then received this dose every 48 hours for six doses, and at sacrifice five days after the last dose, two of the five had a few adhesions. When the single injection experiment was repeated on three starved rats, they promptly died of peritonitis—an interesting confirmation of the findings of Dr. Paul Cannon[12] on the relationship between nutrition and susceptibility to infection.

While the experiments suggest that infection is not so important a factor in the formation of adhesions as we have sometimes thought, it is important to remember that the rat has a high species resistance against peritonitis.

The introduction of 2 cubic centimeters of rat blood in each of the five rats did not produce adhesions, and the introduction of 2 cubic centimeters of such blood one-half hour after injection of an adhesion-producing dose of sodium hydroxide, did not prevent their formation. If blood clotting was an important element in adhesion formation, it would seem probable that a greater effect would have been observed in the first set of experiments.

One practical point on which we have attempted to get information, is whether it is better to reperitonealize areas where the serosa is broken or whether it is preferable to return such areas of the bowel to the peritoneal cavity with the raw area exposed. The results to date indicate that it is better not to suture.[13] Of course, this assumes that there is no perforation. See Table 8.

A few of the agents proposed for the prevention of adhesions were tested. Of these, the most widely used is probably heparin. Its use was suggested by Lehman and Boys[14] in 1941 and in 1945, in discussing a paper by Massie, Dr. Lehman[15] stated that "the experimental results have been consistently conclusive."

Table 9. The Fate of Adhesions Produced by Two Standardized Procedures Without Treatment

Procedure and No. of Rats	No. of Days After Trauma	Adhesions Present	Adhesions Not Present	Autopsy Observations	Pathologic Section
Chemical					
36	3	36		Adhesions not quite firm—holding well	Large number of cells, including few fibroblasts
20	4 & 5	20		Adhesions well formed	Large number of cells, including few fibroblasts
10	10	10		Adhesions quite firm	Capillary growth, with fibrous tissue
10	15	10		Adhesions quite firm	Capillary growth, with fibrous tissue
10	20	7	3	Adhesions very strong and band-like	More fibrous tissue
10	80	3	7	Areas well healed. Adhesions band or cordlike	Mostly fibrous tissue
Mechanical					
10	4 & 5	10		Adhesions well formed	Large number of cells, including few fibroblasts
5	60	2	3	Adhesions strong and bandlike	More fibrous tissue

In the rat this did not prove to be the case. In small doses, adhesions formed as usual in response to a standard stimulus, and as the dosage of heparin was increased more and more rats died of hemorrhage.

Additional data has been obtained which shows the same type of result. Boys and Lehman[16] recommended heparin particularly to prevent the reformation of adhesions divided at a second operation. We have not used it in this manner because the control results in such experiments in rats were radically different from those which Boys and Lehman[16] found in the dog.

Other materials which were tested in our laboratory include physiologic saline solution, gelatine solution, fibrin foam, fibrin film, gelatine sponge, papain and hyaluronidase. None of these materials prevented adhesions satisfactorily. A hyaluronidase preparation lowered the incidence of adhesions somewhat following Allis clamp injuries, but attempts to extend this to Payr clamp injuries gave negative results. The Allis clamp injuries do not give adhesions consistently, and I do not feel confidence in the results obtained with this spreading factor.

TABLE 10. PERSISTENCE OF ADHESIONS IN THE RAT

STIMULUS	NUMBER OF RATS	INTERVAL	PER CENT OF ADHESIONS	RESULT
0.05 cc. 3% NaOH	36	3 days	100	Adhesions not quite well firm but hold rather well
0.05 cc. 3% NaOH	20	4-5 days	100	Well-formed adhesions
0.05 cc. 3% NaOH	10	15 days	100	Well-formed adhesions
0.05 cc. 3% NaOH	10	20 days	70	Seven had adhesions Three had no adhesions
0.05 cc. 3% NaOH	10	80 days	30	Three had adhesions Seven had no adhesions
Allis clamp injury	10	4-5 days	100	Ten had adhesions
to cecum.	5	60 days	40	Two had adhesions; three had no adhesions
Payr clamp	15	89 days	75	Fine adhesions remained
Talc, 10 mg.	5	20 days	100	Extensive adhesions still present
Talc, 5 mg.	15	92 days	46.7	Fine adhesions remained

Sometimes, one has the experience of doing a secondary laparotomy after drainage of an appendiceal abscess and finding remarkably few adhesions. This naturally raises the question of the permanence of adhesions. In the rat, adhesions evoked by at least three stimuli were followed for periods of over 60 days. The results are shown in Tables 9 and 10. The adhesions remaining were often much attenuated.

The last group of experiments were carried out in an effort to answer the old question of how the omentum reaches and surrounds an area of inflammation or injury. For this purpose, Plexiglas windows were inserted in abdominal wounds in rats and carefully sutured in place. Dr. Jackson[17] found it possible to keep such animals alive as long as seven days by sealing the skin to the Plexiglas with collodion. A crushing injury was produced in the cecum of the rat. This quickly became hemorrhagic, and the cecum was placed where the injured area could be seen easily through the window. For eight hours or more the area rode freely up and down with respiratory movements. Between twelve and 21 hours, grayish exudate could usually be seen extending out from the area of injury. At 26 hours in a typical experiment, this grayish exudate was denser and the omentum was adherent to it. Gradually, contiguous portions of the omentum became adherent to the exudate and tended to engulf it. Mallory's trichrome stain suggested that the grayish material was fibrin. These observations suggested that the omentum moves passively with respiration and peristalsis until one part of it accidentally comes in contact with a sticky exudate from the area of injury. Then it continues to be moved passively, but this point of attachment remains and soon other points of contact are formed. As more and more attachments are formed the omentum comes to engulf the exudate and eventually to cover the area of injury. To what extent the fibrin in the exudate contracts, drawing the omentum and injured bowel together we do not know, but it seems likely that this may occur. Neither do we know to what extent the omentum may share in production of exudate, but this seems quite possible also. Such a mechanism would help explain the capacity of the omentum to surround and encapsulate foreign bodies, a silk suture, for example.

TABLE 11. INTESTINAL OBSTRUCTION WITH ADHESIONS. Hospital of the University of Pennsylvania (1935-1945—60 cases)

TYPE OF OPERATION	PREVIOUS OPERATIONS: NUMBER OF CASES	PREVIOUS OPERATIONS: PERCENTAGE OF TOTAL CASES (48)
"Pelvic"	10	21
Appendectomy	13	27
"Pelvic" and appendectomy	8	17
Cholecystectomy	5*	11
Gastric resection	2	4
Laparotomy	2	4
Splenectomy	2	4
Colectomy	2	4
Adrenalectomy	1	2
Repair, perforation of sigmoid	1	2
Drainage, retroperitoneal abscess	1	2
Repair, perforated ulcer	1	2
	48†	100

*Four of these 5 patients also had had gynecologic surgery.
†Only 48 of the 60 patients in this series had had previous surgery.

A review of our clinical material suggests that advances in surgical care have finally reduced the operative mortality of obstruction due to adhesions. Table 11 shows the antecedent operations in the group. Of the 60 patients, 48 had been operated upon before. Seventeen had been operated upon before for adhesions. Of the 60 cases, 43 were acute and 17 chronic. There were three deaths within 30 days of operation, or an operative mortality just under 7 per cent.

Certainly the picture has improved from the standpoint of operative mortality when one compares it with earlier series (Table 3). In general, the simplest procedures were carried out which seemed to offer immediate relief. Since an average of 5.4 years intervened between the previous operation and

the operation for the relief of obstruction, it is obviously premature to gauge the end results, and no attempt has been made to do so. The fact that only 17 of 48 previous operations were done for obstruction suggests that the procedures used were frequently effective, and that if adhesions did reform they were often located more favorably.

In a series of 20 rats in which adhesions were produced by crushing the cecum, simple division of the adhesion was carried out at a second laparotomy. At a third laparotomy only eight, or 40 per cent, had reformed an adhesion. In these, the adhesion was again divided and at a fourth laparotomy only five, or 25 per cent of the original series, had reformed the adhesion. This would indicate that simple division of adhesions was worthwhile though not certain.

In summary, experiments have been carried out in rats which indicate that there are marked individual differences in the formation of adhesions, but that mechanical injury had to be severe, with crushing of all layers of the cecal wall, to produce adhesions regularly. Chemical injury too had to be deep enough to damage the deeper layers of the intestinal wall to produce adhesions regularly.

Catgut resulted in marked and widespread adhesion formation.

Silk and cotton were encapsulated by the omentum when free.

Wire sutures caused the least reaction.

Subserosal silk usually caused no adhesion.

Seromuscular silk sutures caused adhesions fairly regularly.

Among glove powders, talc produced adhesions regularly in amounts as small as 10 milligrams. Bio-Sorb seldom produced adhesions in amounts of 25 milligrams, and potassium bitartrate produced none—even in amounts as large as 200 milligrams.

Adhesions produced by talc often resembled the general matting together of the intestines seen in some of the worst cases of adhesion formation in man.

Injuries limited to the serosa usually heal without adhesion formation and, in the rat, the reperitonealization of such breaks with fine silk sutures resulted in the formation of more adhesions than when this step was omitted.

Heparin, papain, saline and a number of other substances failed to prevent the formation of adhesions after standard injuries.

Adhesions do disappear with the passage of time, in some animals, but this did not approach 100 per cent within three months in the rat.

Observations through a Plexiglas window suggest that the omentum reaches an area of injury by random passive movements during which it is caught by sticky material containing fibrin, which exudes from the area of injury.

A review of a series of 60 cases of obstruction due to adhesions indicates that the operative mortality, which remained high in most series reported up to 1945, has been markedly reduced. No new solution for the problems of the patient with the matted abdomen is as yet available, but it is believed that further attention to technical details may render the occurrence of these cases much less frequent.

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ANNUAL ADDRESS FOR 1949

RELIEF OF INTRACTABLE PAIN IN INOPERABLE CANCER

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Pain presents an urgent problem in the treatment of any type of disease. Pain is the symptom that brings the sufferer most promptly to his physician demanding that immediate action be taken to relieve his distress. Unfortunately, the pain accompanying the spread of cancer can rarely be checked by an attack on the original lesion. The pain is caused by infiltration of the growth beyond its initial site into adjacent areas, with compression of sensory nerves. The cancer has spread beyond the possibility of surgical removal or roentgen-ray control. The cancer will kill the patient. He is in pain. How can his last months of life be made less tragic and distressing?

Drugs in the form of the opium derivatives have been the time-honored answer to this problem. But opium gives only intermittent surcease of pain. The duration of relief depends on the amount administered and the intervals at which it is used. Furthermore, a tolerance to opium soon appears, and larger doses are necessary, given more frequently. Again opium causes constipation, weight loss and often nausea. And opium does not completely relieve pain.

Two methods are available to check intractable pain accompanying inoperable cancer. The afferent pain pathways leading from the region to which the pain is referred may be sectioned by rhizotomy or cordotomy. The area is rendered anesthetic if rhizotomy is performed, or analgesic if the anterolateral pathways in the spinal column are sectioned by cordotomy. Or those parts of the frontal lobes in which perhaps pain sensation is interpreted as pain can be ablated by prefrontal lobotomy. Following frontal lobe section the sufferer unquestionably can feel pain but he does not interpret it as a distressing sensation and pays little or no serious attention to it. Admittedly, our knowledge of the psychophysiology of the frontal lobes is very meager. But a limited experience with section of specific association fibers running into this area shows that this maneuver seems to render the patient oblivious to pain, which hitherto even large doses of opium had failed to check.

Both these methods for relief of pain involve the patient in further surgery. Cordotomy or prefrontal lobotomy are relatively minor procedures and can be carried out under local anesthesia with no more than mild mental distress and discomfort to the patient. Nevertheless, an operation is an operation. Are we justified in subjecting these pain-wracked sufferers, dying from inoperable cancer, to further surgery? Obviously, the course of the disease will in no wise be influenced. These people, however, will be much more comfortable until they die. The decision, of course, is one of expediency. If the patient is suffering intolerable pain, in spite of generous doses of opium,

he should be told that the possibility exists for great relief of his distress if he will undergo a relatively simple surgical procedure. He should be told that the price he must pay for relief by cordotomy is 45 minutes of discomfort in the operating room, followed by stiffness and soreness of his neck for the next 72 to 96 hours. If a bilateral cordotomy is necessary for bilateral pain, a mild weakness in either leg may be noted, and, most serious, a 20 per cent chance of loss of sphincter control exists. Sphincteric difficulties are the hazard following cord section. Many patients with prostatic, vesicle, cervical or uterine cancer already have some difficulty with urinary control because of the involvement of the bladder wall or the sphincteric nerve supply by direct infiltration or pressure of enlarged lymph nodes on the presacral nerves. Another disadvantage of cordotomy is that after pain has been relieved by cord section, the problem of reducing the amount of opium the patient is receiving may be difficult, especially if large quantities have been given for a long time. Withdrawal symptoms may appear, the distress produced by cutting down the opium may scarcely seem justified, and smaller doses at less frequent intervals may be necessary to keep the patient comfortable. What can be accomplished by rhizotomy and cordotomy and the price the patient must pay for pain relief is known through 20 years of clinical experience.

Prefrontal lobotomy for the relief of pain is a relatively new maneuver. Apparently this procedure has these advantages. Cancer is a fluid disease and may metastasize contralateral to or above the level of analgesia produced by cordotomy. If pain is relieved by prefrontal lobotomy, extension by metastasis of the cancer which seemingly should produce distress in other areas, fails to elicit from the patient further complaints. Secondly, frontal ablation seems to check the desire for further morphine. The problem in carrying out a prefrontal lobotomy is to know just how much of what part of the lobes should be removed. Too wide an incision will cause marked personality changes and a dull, listless patient, not complaining of pain, to be sure, but also not taking much interest in his surroundings. Bladder and rectal incontinence accompanies prefrontal lobotomy in direct relationship to the amount of frontal lobes removed. The greater the indifference of the patient to his pain and his surroundings the more difficulty will be found in persuading him not to void wherever he happens to be.

Considering now the relief of pain by section of afferent fibers, the head and neck from the vertex to the level of the clavicle can easily be rendered anesthetic by section of the sensory root of the trigeminal, the glossopharyngeal and the upper four or five cervical posterior roots. The face and mouth, together with the frontal and maxillary sinuses, can be freed of pain by blocking the appropriate branch of the trigeminal or by severance of its posterior root. Nasopharyngeal pain or pain on swallowing is relieved by glossopharyngeal section. Distress in the supraclavicular or posterior cervical region is checked by interruption of the appropriate cervical posterior roots.

A painful cancer of the face, mouth or tongue may be handled in one of two ways, depending upon the position and extent of the lesion. If it is situated within an area supplied by afferent fibers from a single branch of the trigeminal, that branch may be blocked with alcohol. When the growth is localized and its subsequent removal seems possible, alcohol injection for nerve block is to be preferred to root section. A successful alcohol block produces anesthesia for only a year to eighteen months. Following local excision of the cancer, the patient will not be annoyed by permanent anesthesia of the face which always follows complete root section. Furthermore, successful alcohol block, while painful, involves the patient in no operative risk. Unfortunately, however, if on the surface, the cancer may involve the skin area through which the injection is made or, if deep, may so distort the anatomy of the nerve that an accurate block is impossible.

But when the cancer of the face, tongue or sinuses is sufficiently extensive to include areas supplied by two or more branches of the trigeminal, root section is indicated. This involves the patient in a relatively minor surgical procedure which may be carried through entirely under local anesthesia without more than momentary distress at the instant of actual section of the trigeminal root. If the growth involves the lower lip, tongue or floor of the mouth, the ipsilateral external carotid should be ligated. This accomplishes three purposes: (1) It makes excision of the lesion less bloody. (2) By control of the middle meningeal artery, the approach to the sensory root is simplified. (3) The skin incision for approach to the vessel should be so placed that the superficial branches from the cervical nerves supplying the skin beneath the line of the jaw are picked up and severed. This maneuver, plus root section, assures complete desensitization of the trigeminal area since these cutaneous nerves establish a definite overlap with the mandibular branch of the fifth.

Not only is the pain produced by the cancer relieved by section of these various nerves but the operative field is anesthetized with complete comfort to the patient after excision of the lesion. He can now sleep and eat without serious discomfort; he regains weight and will accept any amount of local treatment necessary because it is not painful.

Face, mouth and tongue cancer presents a special problem. It is visible to the patient. The education of the public on the subject of cancer is now so widespread that the patient is usually fully cognizant of his condition. He is naturally gravely alarmed, and miserable because of the pain. He wants to be treated, but if the treatment aggravates his pain as it so often does, he wonders whether it is worth while. If his pain is abolished by blocking afferent pathways, he will accept the maximum amount of treatment. A local recurrence may be handled and excised without complaint. And not infrequently because the therapy is not painful and can be carried to any extent, a complete cure is accomplished.

Pain on swallowing, commonly encountered in cancer of the tonsil or base of the tongue, requires glossopharyngeal section. Pain in the neck, due

to pressure from metastasis to the glands in this area, necessitates section of the upper cervical posterior roots. Pain deep in the ear, which fortunately only infrequently accompanies cancer, cannot be controlled by section of any nerve in our experience.

Glossopharyngeal section, with or without posterior-root section, involves the patient in a serious operative procedure. Whether the suboccipital exposure be carried through in the prone or sitting position, a general anesthetic will be necessary when the glossopharyngeal nerve or the posterior roots are cut. Local anesthesia may be used up to this point. A little intravenous pentothal is sufficient to carry the patient through the nerve section without distress. But this is major surgery, relatively more severe and dangerous than trigeminal root section. These patients are usually elderly, debilitated by cancer and pain. The advantages to be gained by any surgical procedure in this type of patient must be carefully weighed against the risks involved.

Pain due to invasion of the brachial plexus by metastasis from breast cancer or involvement of the posterior roots between cervical five and thoracic two by vertebral invasion has been difficult to control. The arm is almost always involved in the radiation of the pain. Rhizotomy brings relief, but, since root section results in suppression of all modalities of sensation, the arm and hand are numb and useless to the patient. For this reason, rhizotomy for pain in the arm (or for that matter in the leg or any region which the patient uses purposefully) is a thoroughly unsatisfactory procedure. The dead limb quickly becomes a source of annoyance, paresthesias develop and the situation is not improved.

A high cordotomy at the level of cervical one or two performed by a bold and lucky operator will produce analgesia in the arm and hand without rendering the limb useless by eliminating touch and sense of position. But since the cordotomy is performed only four segments above the area affected, the incision into the cord must be wide and deep. The medulla is close to the first cervical segment. Phrenic involvement with interference with the function of the diaphragm may result, together with damage to adjacent pyramidal tracts.

If prefrontal lobotomy is to be of value then these cases where pain is referred to the arm and hand are those in which it is to be hoped that it will be successful. Certainly cordotomy is not an adequate solution to this problem.

Pain of any type referred to any area from the ensiform process to the toes may be relieved by section of the anterolateral columns in the spinal cord on one or both sides. Accurate section of these tracts results in loss of pain and temperature sensations only. Consequently, the legs and sphincters function normally, since touch and position sense are not impaired. But the section must be accurate, for pain sensation will not be completely abolished unless all afferent fibers are severed.

Furthermore, since the pyramidal tracts lie closely adjacent, deviation of

the incision into the cord may injure them and result in ipsilateral weakness of the leg. When bilateral cordotomy is performed the chance of technical error and consequent neurologic deficit is doubled. Following bilateral section, a full 20 per cent of the patients have sphincteric difficulty, usually retention. As has been mentioned, this loss of control is not due entirely to the operation, since in many cases the cancer has already invaded the bladder and crippled the function of the sphincters. However, about a third of these patients recover control of urination within a week or two. Nevertheless, this deficit is the distinct hazard following bilateral cordotomy.

A unilateral cordotomy should never result in bladder difficulty unless alcohol has been previously introduced into the lumbar subarachnoid space in an effort to relieve pain. Bladder control seems to be bilateral. Alcohol is always injected on the same side as the pain to block adjacent lumbar posterior roots. But the function of the anterior roots on that side controlling the sphincters may be impaired unavoidably. The sphincters are still under contralateral control and no deficit appears. But the pain is not relieved and cordotomy is suggested. The anterolateral column, of course, must be sectioned contralateral to the side of the pain, since the pain fibers decussate in the substance of the cord. This is done and the remaining bladder fibers are sectioned, with resulting loss of sphincter competence.

Since cordotomy is performed for relief of pain, a very careful history of this pain is necessary, particularly as to position and radiation. While in many instances the pain is bilateral when the patient is first seen, many complain only of unilateral pain. However, careful questioning almost always reveals a major, intense and distressing pain on one side and some pain at least on the opposite side. If the major complaint is relieved by contralateral cordotomy, the opposite side in six to eight weeks will become the site of as great or even greater distress. Should a bilateral cordotomy therefore be done as an initial procedure? Experience seems to show clearly that cordotomy first on one side, and then, six to eight weeks later if occasion demands, on the opposite side reduces materially the percentage of bladder involvement. However, in certain instances where the bladder sphincters are already incompetent, immediate section of both sides of the cord should be performed. While the separation of the cordotomy into two procedures will spare the sphincters, nevertheless, this requires two operations. A patient may become discouraged if the original operation relieves only part of his distress and the second session may be refused. If only a unilateral cordotomy is done in a patient with major pain on one side but with some contralateral distress, the situation must be explained to him carefully and the possibility of the need for a second procedure must be emphasized.

The original technic for cordotomy described cord section at the level of the first to the fourth thoracic segments. In this region the laminae are not thick, the cord is easily accessible, the dentate ligament is a well-formed definite landmark, useful for cord rotation and determining the posterior level of the incision and, lastly, the spinal roots are widely spaced and do not

interfere with rotation of the cord or cover in the area to be incised. Furthermore, if the pyramidal tract is damaged, only the lower extremities are affected. However, after high thoracic cordotomy, a burning sensation may remain in the analgesic area. Considering this to be sensation carried over the sympathetic afferent pathways, the point at which cordotomy was performed was changed to the fifth to eighth cervical segments to cut the afferent pain fibers above the point where they might be joined by the sympathetic fibers. In this region, however, the roots are large and closely placed; consequently, rotation and section of the cord is more difficult. Cervical two to five has been selected as the better area for the incision. Technically, this region is easy to approach, the cervical laminae are thin and easily removed, and nerves small and widely spaced, and the dentate sufficiently large to be readily seized and used in rotation. Furthermore, one of the disadvantages of cordotomy is the drop in pain level during the first two or three weeks postoperatively. This is probably due to trauma and edema about the section. As the swelling subsides, fibers not actually severed recover function and the sensory level may drop six or eight segments. Therefore, the higher in the cord the afferent fibers are cut, the higher will be the final permanent level of analgesia. If regression of the level occurs, it is less likely to reach such a low level that pain will recur. The only objection to the high cervical section is the possible paresis of the ipsilateral hand and arm if the pyramidal tracts are injured. Phrenic damage from a section at the third or fourth cervical segment is most unlikely.

Unilateral cordotomy requires only a unilateral laminectomy. The spines are removed only if a bilateral cord section is indicated. The prone or the sitting position may be employed. Local anesthesia is always used, and the patient is tested promptly for pain loss. The section should be made sufficiently deep at the third cervical segment to carry the analgesia as high as the third, second or possibly the first thoracic dermatomes. If both sides of the cord are to be cut simultaneously, the section should be made at the second and fifth cervical segments. If both anterior columns are incised at the same segment, a transverse myelitis can develop and has developed. Therefore, bilateral incisions must be staggered.

Whenever a patient complains of pain, the nature, cause and type of which is uncertain, spinal anesthesia may afford important information. If following this procedure, the area to which the pain is referred is anesthetic, and yet the pain is not relieved, a cordotomy is useless. Furthermore, spinal anesthesia will show the operator to what level his analgesia must be carried to afford complete relief. An incision into the cord of sufficient depth to produce analgesia to the same level as that following spinal anesthesia will insure relief of pain.

The last 100 cases of cordotomy at the University of Pennsylvania Hospital have been reviewed—fifty unilateral and fifty bilateral (Table 1). Since 1942, an additional 35 patients have had a unilateral or bilateral cordotomy performed for the relief of severe pain not due to cancer. The

TABLE 1. CORDOTOMY FOR 100 CANCER CASES

	UNILATERAL CASES	PERCENTAGE OF UNILATERAL CASES	BILATERAL CASES	PERCENTAGE OF BILATERAL CASES
Number of cases	50		50	
Complete relief	38	76	39	78
Unsuccessful	6	12	6	12
Deaths	6	12	5	10
Complications				
Weakness in extremities	1	2	4	8
Bladder difficulties			8	16
Reference of pain above level	2	4	3	6

results obtained in these cases are included to indicate the lasting improvement following a properly placed section (Table 2).

Clinical experience with prefrontal lobotomy is less extensive. The operative maneuver in experienced hands is easier than cordotomy. The patient is in a more comfortable position on the operating table and has little or no

TABLE 2. CORDOTOMY FOR 35 NONCANCER CASES

	UNILATERAL CASES	PERCENTAGE OF BILATERAL CASES	BILATERAL CASES	PERCENTAGE OF UNILATERAL CASES
Number of cases	29		6	
Complete relief	21	77	4	66⅔
Unsuccessful	8	23	2	33⅓
Deaths	0		0	
Complications	0		0	

actual pain. The problem is entirely that of learning from further experience exactly how much of what part of each frontal lobe should be removed. A unilateral frontal lobotomy, whether in the dominant or nondominant hemisphere or ipsilateral or contralateral to the side of the pain, is of no value. The removal of too large an area of the frontal lobes may produce a patient free of pain but heavy and dull, with marked personality changes and, consequently, probably incontinent. However, since pain relief is the purpose of the procedure, a wide section of association pathways is to be preferred.

Following a successful prefrontal lobotomy, these patients present a fascinating psychosomatic problem. They do not complain spontaneously of pain or ask for sedation. On direct question they may state frankly and with a smile that the pain still exists. On neurologic examination no loss of pain sensation, or any other modality of sensation for that matter, can be demonstrated in the area to which the pain is referred. Due to section of proper

frontal association pathways the pain simply ceases to be an annoyance to the patient.

Our experience with prefrontal lobotomy is relatively meager. In fifteen cases the maneuver has been used to relieve intractable pain. In ten cases the results were highly satisfactory; in four little or no relief of pain was produced; and one patient died (Table 3).

TABLE 3. PREFRONTAL LOBOTOMY IN 15 CASES

	NUMBER OF CASES	PERCENTAGE OF CASES
Complete relief	10	66⅔
Unsuccessful	4	26⅔
Deaths	1	6⅔
Complications		
Bladder difficulties	5	33⅓

These, then, are the procedures available for relief of intractable pain accompanying inoperable cancer. Unfortunately, as has been observed from the figures given, complete relief cannot be assured in every case. Even to obtain the relief that can follow these maneuvers, one and perhaps two surgical sessions are necessary. Nevertheless, intractable pain is an outrageously distressing accompaniment of incurable disease. If the patient appeals for some form of relief other than the morphine derivatives the methods here described should be outlined to him. If his pain is really unbearable he can obtain at least a very great measure of relief.

ANNUAL ADDRESS FOR 1950

EXPERIENCES WITH SIXTY-FIVE OPERATIVE CASES OF MEDIASTINAL LESIONS

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H. Taylor Caswell, M.D., John H. Hall, M.D.,
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The purpose of this paper is to present the diagnostic and the therapeutic aspects of mediastinal masses on the basis of 65 patients who were operated upon in Temple University Hospital from 1935 to June 1950.

At the beginning of this period, most physicians held the opinion that lesions of the mediastinum were extremely confusing diagnostically and that the benign were harmless and the malignant hopeless. X-ray therapy was the main method of attack. Fortunately, there were several courageous surgeons who had pointed out the contrary as witnessed by the reports of Bastianelli[3] in 1893 and of Frank[22] in 1906, of von Haberer[58] in 1917, of Harrington[29] and Mixter and Clifford[45] in the period between 1917 and 1929, and with a host of subsequent reports demonstrating increasing knowledge of the accessibility of the mediastinum by Eggers,[19] Heuer,[31] Crosby,[16] Haagensen,[27] Neuhof,[47] Hedblom,[30] Phemister,[50] Wangensteen[60] and others. More recently, the excellent reviews of Heuer and Andrus,[33] of Poer,[51] of Blalock,[7] of Kent, Blades, Valle and Graham,[36] of Thompson[55] and of Harrington[28] have all attested to the increasing number, which are not only discovered but are classified so that percentage of occurrence can be tabulated, diagnosis improved, and a better view of prognosis reached.

An idea of the incidence may be obtained from the fact that Heuer and Andrus' report[33] included 82 cases of their own and 613 collected, that of Blades[5] contained 25 teratoid tumors from the Barnes Hospital and 208 collected cases; while Hedblom[30] discussed 176 cases, and Kent et al.[36] studied 126 neurogenic tumors, of which 21 were their own. Harrington[28] has recently reported 168 personal cases of various types.

All observers have called attention to the fact that these tumors, even if benign, eventually produce serious, if not fatal, symptoms and that there is a definite tendency to become malignant, if they are not primarily so. In some groups, such as the neurogenic tumors, evidence of malignancy runs as high as 30 per cent. Thus, because of their tendency to malignant degeneration or of their continued growth interfering with the important functions of the thoracic viscera by displacement or compression, these tumors and cysts appear to be extremely significant.

Symptoms are absent in a moderate number of cases. However, in many there are symptoms produced by mild irritation or compression of somatic or sympathetic nerves, such as pain, Horner's syndrome, recurrent laryngeal palsy or phrenic nerve interruption. In addition, there may be cough from

impingement on bronchi, dyspnea due to lung compression, or the more significant pulmonary manifestations of bronchial obstruction. One of the most common complications is of infection or rupture, or both, of cystic masses in the mediastinum. Some of the neurogenic tumors grow by extension through foramina into the spinal column, producing compression or irritation of the cord, and some of the gastrogenic cysts cause vomiting, diarrhea, hematemesis or melena, and even death in infants. Malignancy is almost invariably indicated by the presence of superior vena caval obstruction, recurrent or phrenic nerve paralysis, or weight loss without evidence of infection, although there are rare exceptions. However, many malignant lesions do not produce such symptoms.

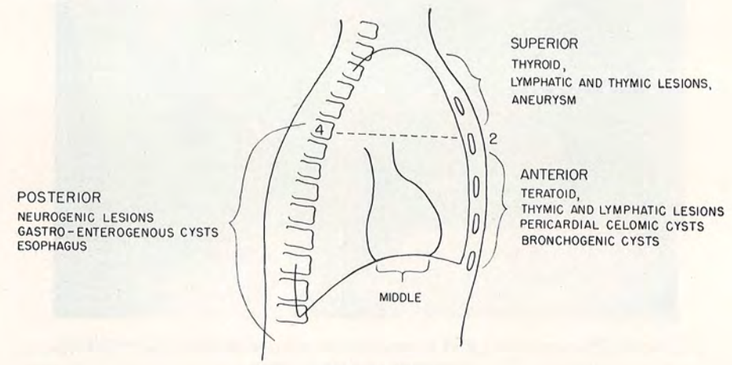


Figure 1

Physical signs suggesting the presence of mediastinal pathology are numerous, but frequently they are absent. Certainly venous distention or facial edema, lymphadenopathy, tracheal tug, a thyroid extending behind the sternum, enlarged or displaced mediastinal dulness, lung signs such as dulness or absence of breath sounds, hepatomegaly (nodular) and the presence of abdominal or pelvic masses suggesting a primary malignancy which may have metastasized into the mediastinum are significant if present.

X-ray examination of the chest is extremely helpful and is much more informative than physical examination. Postero-anterior, lateral and oblique views are required for a thorough study, and routinely barium should be swallowed to locate the esophagus and to identify the dilatation of achalasia. Planigraphy, bronchography and angiography may be useful.

Location is helpful (Fig. 1), in that the anterior mediastinum usually harbors teratoids, pericardial cysts, bronchogenic cysts and lesions of the lymphatic tissues, including the thymus. In the latter two there is a high percentage of malignancy. Practically all neurogenic tumors occur posteriorly, as do also the gastrogenic cysts and dilatations or tumors of the esophagus.

The superior mediastinum encompasses lesions of the thyroid, aorta and lymphatic tissues.

The contour of the shadow is often distinctive in that an indefinite border is indicative of infiltration and malignancy, while a sharply defined border is usually associated with a benign lesion. Nodular or bosselated contour suggests not only a solid tumor rather than a cyst but the possibility that it is also malignant.

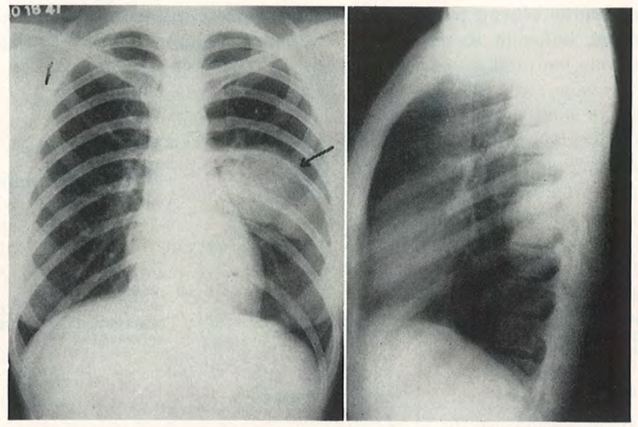


FIG. 2. Posterior location of tumor, with erosion, not invasion, of seventh rib by benign neurogenic tumor.

Bone defects can also be demonstrated by heavier roentgenographic penetration (Fig. 2). Intervertebral foraminal enlargement suggests an hourglass tumor which may have associated spinal cord symptoms. Ribs or vertebrae that are eroded indicate compression, usually from benign neurogenic lesions. Ribs or vertebrae that are invaded or destroyed strongly suggest malignancy.

Additional diagnostic steps include blood count and bone-marrow biopsy for possible leukemia, Wassermann, the judicious use of needle biopsy and, frequently, exploratory thoracotomy. A small dose of x-ray therapy in lesions which clinically suggest lymphoblastoma is sometimes diagnostically helpful, since rapid response corroborates the clinical impression.

Special classifications should be considered in more detail.

Lymphatic Lesions. Lymphoblastomas—leukemia, Hodgkin's disease and lymphosarcoma—accounted for some 22 per cent of mediastinal enlargements in eight years at Temple University Hospital. These lesions constitute a high percentage of the mediastinal enlargements in most of the reported groups. Heuer and Andrus[33] found 32 per cent in 145 cases. In most of

these, in addition to other evidences of the disease (such as superficial lymphadenopathy), there is rather distinctive infiltration in the anterior mediastinum of a thin, plaquelike induration of indefinite border, which is rather easily recognizable (Fig. 3). It is in such cases, particularly if superior vena caval obstruction is present, that x-ray therapy is justified diagnostically. However, two of our cases had no such distinctive findings

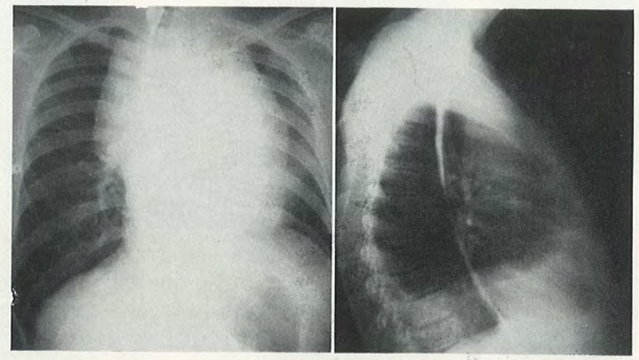


FIG. 3. A case of leukemia, demonstrating bilateral invasion extending laterally from mediastinum, confined to a rather thin portion of the anterior mediastinum as seen in the lateral view.

and were proven at exploratory thoracotomy (Fig. 4, *top*). These developed additional evidences of the disease as time passed but survived for three years after excision and eleven after exploration, respectively, under x-ray therapy in small, repeated doses (Fig. 4, *bottom*).

Thymic tumors have been reported on several occasions, with and without myasthenia gravis. In 1942, Poer[51] reported 41 tumors, plus 30 of enlargement not due to tumor, since which time Campbell[9] has reported 2, Turnbull[56] 1, Clagett[11] 5, Blalock[7] 2, plus 18 cases of other lesions of the thymus associated with myasthenia gravis, and Viets[57] has been quoted as finding 3 tumors in 14 cases, totaling some 54 tumors and 59 additional cases of enlargement or other abnormality. Thompson[55] states that there were at least 6 malignant thymic tumors which were observed in myasthenia gravis. Heuer and Andrus[33] state that the malignant thymomas were more common than the benign.

Our series includes three thymomas, of which two were malignant. One of these patients has remained well and free of tumor for two and a half years. The other patient's lesion was quite extensive and required a transverse, bilateral, fourth interspace and transsternal, Johnson and Kirby[35] incision, with resection of the left innominate vein at its junction with the superior vena cava (Fig. 5). She has remained well for six months. The

patient with the benign lesion showed no evidence of recurrence but continued to have cough and expectoration, which had been the primary symptoms. These were relieved two years later by removal of a mildly bronchiectatic left lower lobe. One additional patient with tumor and myasthenia gravis refused operation.

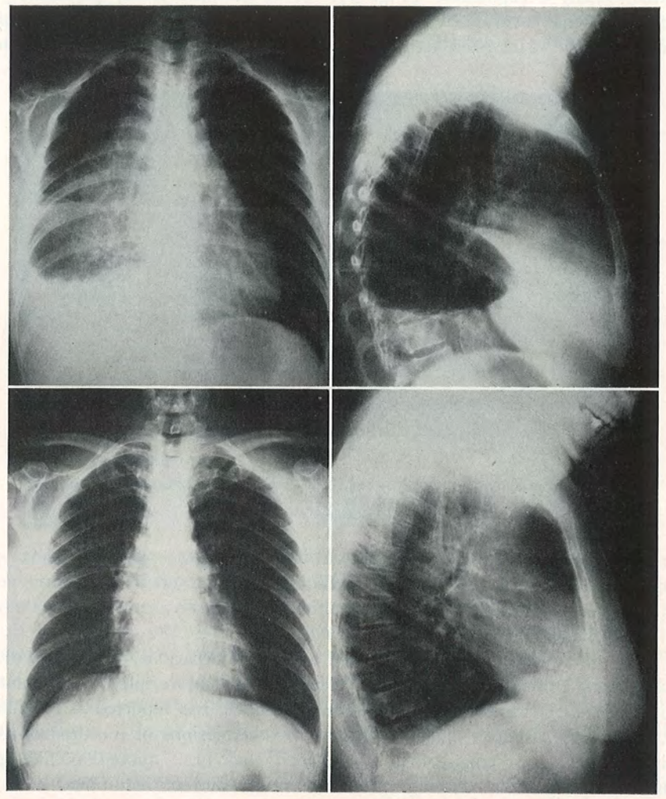


FIG. 4. (*Top*) Hodgkin's disease involving right middle lobe and mediastinum and infiltrating the zone of the diaphragm. (*Bottom*) Same patient 16 months later, having responded well to x-ray therapy in small doses. The patient was followed for 11 years, with repeated recurrences of lymphadenopathy, again controlled by small doses of x-ray.

The difficulty of classifying thymic tumors on a microscopic basis has long been recognized and may account for some of the unexpected results following removal. In any case, it seems quite justifiable to remove the lesion

if this is feasible, although x-ray therapy has some value since this is primarily lymphatic tissue. Certainly, in the lymphoblastoma group, surgery has little to offer beyond diagnosis, while irradiation is quite effective. Small doses, given when symptoms appear, seem to prolong the value of the irradiation therapy so that numerous cases have gone eight to twelve years under such treatment, although the average duration is nearer two to three years before demise. The use of the nitrogen mustards has increased the effectiveness of irradiation in some cases.

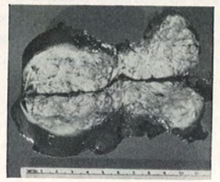
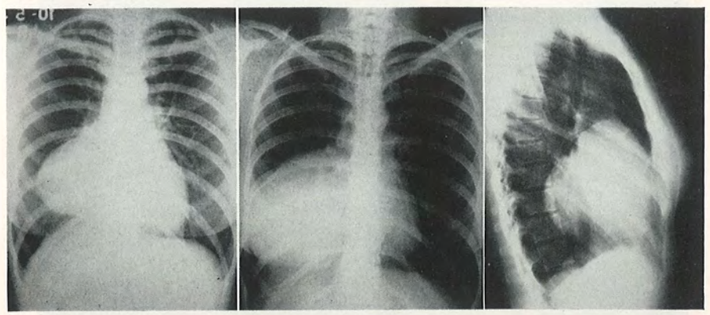


FIG. 5. (*Top*) Malignant thymoma in a 17-year-old girl, involving superior mediastinum and left innominate vein. (*Bottom*) Specimen from patient.

Teratoid Tumors and Cysts. Next to the lymphoma group, these vie with the neurogenic tumors as the most common lesions of the mediastinum. In 1944 Rusby[52] collected 251 cases. In 1949 Harrington[28] reported that 23.8 per cent of his 168 operative cases were teratoids, of which 15 per cent were malignant. These lesions range from simple, thin-walled cysts, containing thin, yellow fluid, to extremely organized affairs, sometimes called "fetus in fetu." Hedblom[30] classified them as (1) epidermoid-epithelium or skin with or without glandular structure of ectodermal origin; (2) dermoid —multicystic and lobulated with the above structures plus mesodermal elements such as cartilage, bone, teeth and muscle; and (3) teratoma—more or less solid and consisting of the above plus entodermal structures, such as

pancreas, intestine and thyroid, with occasional organized arrangements, such as coils of intestine.

Ewing[20] classified them as dermoid if only one germinal layer was represented, as teratoid if more than one germinal layer, and teratoma if they showed elements of all three germinal layers and formed a part of some structure of the body.



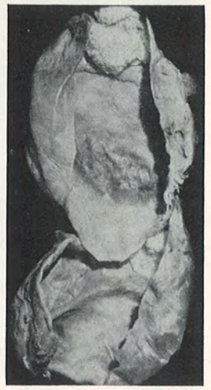


FIG. 6. (*Top, left*) Patient with dermoid cyst. (*Center and right*) Three years later. Note considerable increase in growth and the anterior location of such lesions. (*Bottom*) The cyst removed from patient.

Harrington[28] classes them all as teratoid tumors or cysts, since he feels that sufficient study of even the simpler types will demonstrate representation of all three germinal layers. With this conception Lawrence Wells Smith, former head of our Department of Pathology, agreed after multiple demonstrations of these findings.

They are obviously inclusion bodies, probably originating from the branchiogenic elements which are drawn into the thorax as the heart and diaphragm descend (Fig. 6).

Their location is in the anterior mediastinum, and Blades[5] found only

three of 233 situated posteriorly. What appears to be posterior, when large, may show its true location when decompressed by aspiration (Fig. 7). When x-ray study reveals them to be close to the base of the heart, our experience indicates that they are usually well organized as teratoids or teratomas and are frequently blended with the pericardium, requiring sacrifice of a portion

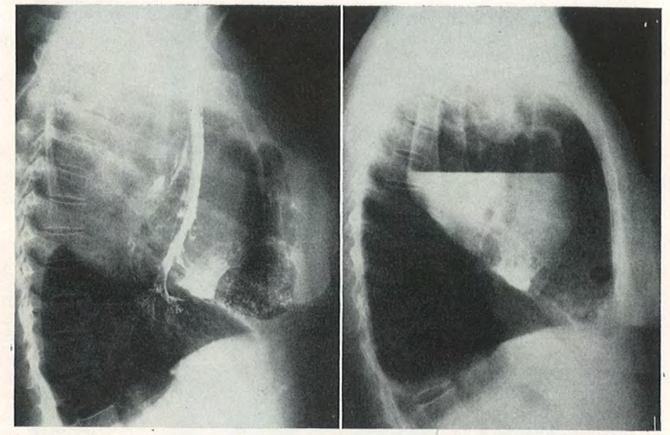


FIG. 7. Tremendous dermoid located in right chest. Note compression of the lung downward and backward, as evidenced by the lipiodol anteriorly and inferiorly located, which expanded into the apex after removal. In the view on the left one is unable to determine whether this is posterior or anterior in location. Three days later, after removal of approximately 1,000 cc. of turbid fluid with cholesterol crystals floating on its surface, the right view shows its anterior location, by the process of decompression.

of it for their complete removal (Fig. 8 A). Hedblom[30] called attention to 19 cases presenting in the suprasternal notch, and Rusby[52] to presternal and hour-glass transsternal arrangements, to intrapericardial and to diaphragmatic locations.

Most reported cases range from 20 to 30 years of age. Many cases are symptomless and are discovered in routine surveys or physical examinations. Because of the slow growth, symptoms rarely occur before puberty and are then insidious in 75 per cent but rather abrupt in the remainder, particularly following respiratory infection. Usually the symptoms are due to their large size, but small tumors can produce cough, pain, hemoptysis or combinations of the three. At times, a cyst can rupture into a bronchus, producing expectoration of peculiar material, including sebaceous matter and hair or pus. They may rupture into the pleura and produce empyema. According to Heuer and Andrus,[33] the most frequent complication is infection by way of the blood or lymph stream.

If left undisturbed these tumors almost invariably contribute to the death of the patient; they failed to do so in only 4.3 per cent of Rusby's series.[52] The average time from onset of symptoms to death in his cases was 1.8 years. Heuer and Andrus[33] found that 47 of 48 cases of untreated teratoid died of their tumor.

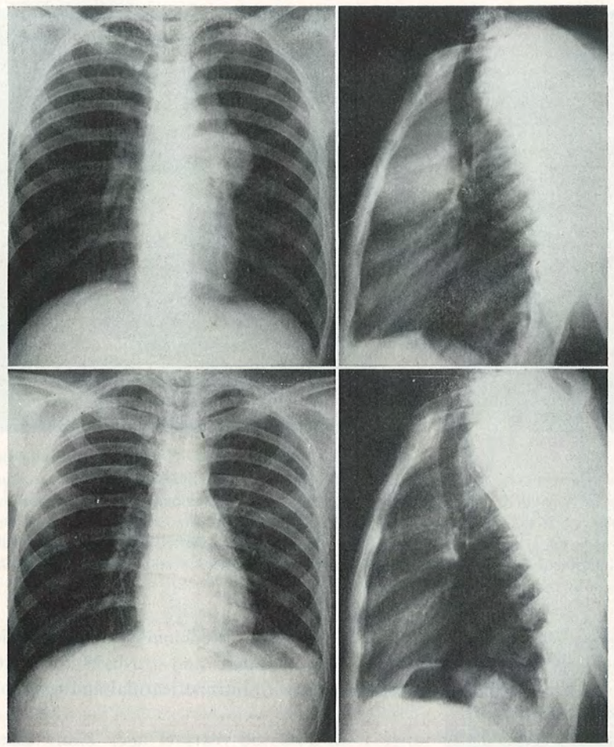


FIG. 8A. (*Top*) Anterior location blending with base of heart, suggestive of teratoid tumor and its intimate association or blending with the pericardium. (*Bottom*) Three months after removal of teratoid and a portion of pericardium.

Irradiation is of no appreciable value even in the malignant types, so that surgical excision in toto is the ideal method of treatment. In the older reports, mortality was about 10.7 per cent but in more recent ones it is only 2 to 3 per cent. Partial removal had almost twice the mortality and only half the number of cures, while external drainage had practically three times

the mortality and only 14 per cent of cures. There seems to be no proof that any malignant teratoid has ever been cured.

In our ten cases there were eight benign lesions and two mesenchymal sarcomas, which were classed as teratoids because of operative findings. Neither of the latter could be removed, and each lived about three years.

The benign lesions were: one infected cyst with symptoms of pulmonary suppuration (Fig. 9); four thin-walled cysts and three multiloculated solid and cystic tumors producing cough in four, pain in three and dyspnea or hemoptysis in one instance each. Only one of these had no symptoms,

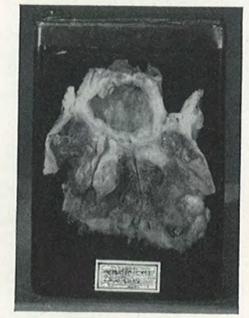


FIG. 8B. Teratoid tumor removed from patient.

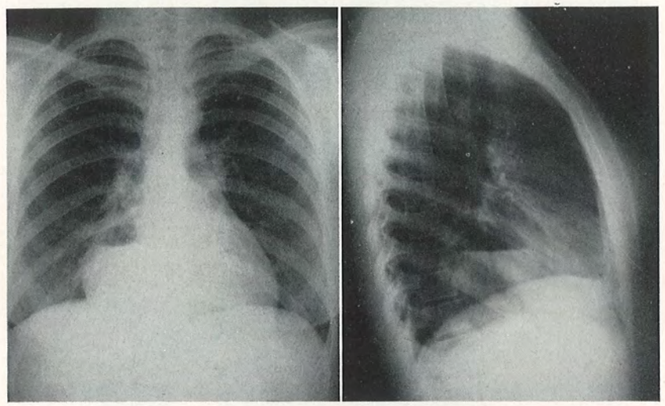
but the cyst was enlarging under her physician's observation. One was classed fetus in fetu (Fig. 10 A, B). Following a difficult dissection, there was one postoperative death from bilateral pneumothorax treated by inadequate drainage. Follow-up for from one to six years showed satisfactory progress in the rest.

Seven asymptomatic patients with positive x-ray evidence postponed or refused operation. We have been unable to follow these.

Neurogenic tumors are of leading frequence in more recent reports. They constituted a larger number in our group than any other single type. Heuer and Andrus[33] collected 108 cases; Kent, Blades, Valle and Graham[36] collected 105 and contributed 21. Godwin et al.[24] report 24, Efskind and Liavaag[18] 21, and Harrington[28] 51, which represented 30 per cent of his series. There are numerous smaller groups reported. Pathologically, these are classified under various headings, often with very slight differences in architecture. In general, there is a high incidence of malignancy, either primarily or due to degeneration of a previously benign neurogenic tumor. Percentages range from 6 to 19. In 850 neurogenic tumors from various parts of the body, Geschichter[23] found 41 per cent to be malignant.

The simplified classification suggested by Fisher[21] is (1) undifferentiated —such as sympatheticoblastoma; (2) incompletely differentiated—such as

sympatheticoneuroblastoma, pheochromoblastoma and astroblastoma; and (3) completely differentiated—such as ganglioneuroma, pheochromocytoma, astrocytoma and neurilemmoma or schwannoma. The extensive discussion on various types can be crudely simplified to the fact that tumors arise from either nerve cells on the one hand or supportive structure on the other, that they are either differentiated with a low potential of malignancy or undifferentiated with a high potential. The fact appears, from many studies, that different parts of the same tumor may show various combinations of these four.



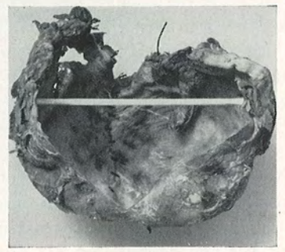


FIG. 9. Infected dermoid cyst of mediastinum. (*Bottom*) The ragged wall of cyst removed.

Of the benign tumors in the group of Heuer and Andrus,[33] 63 per cent were ganglioneuromas, 18.5 per cent neurofibromas and 17.5 per cent neurinomas. Of the malignant group, neurofibrosarcomas (neurogenic sarcomas) seemed to grow more slowly and metastasize much later than fibrosarcomas and were considerably less active than the sympatheticoblastomas, which metastasize early and widely. Unless metastases can be demonstrated, it is often extremely difficult to determine the presence or absence of malignancy until the tumor is removed. Seven of our 18 cases of neurogenic tumor were classified mesenchymal sarcoma, neurogenic sarcoma and malignant ganglioneuroma, as the styles changed in the Pathology Department. The benign lesions were: one neurilemmoma, two ganglioneuromas and eight neurofibromas. Possibly some of our two fibromas and of the 32 fibromas in the literature should be classified under the neurogenic tumors.

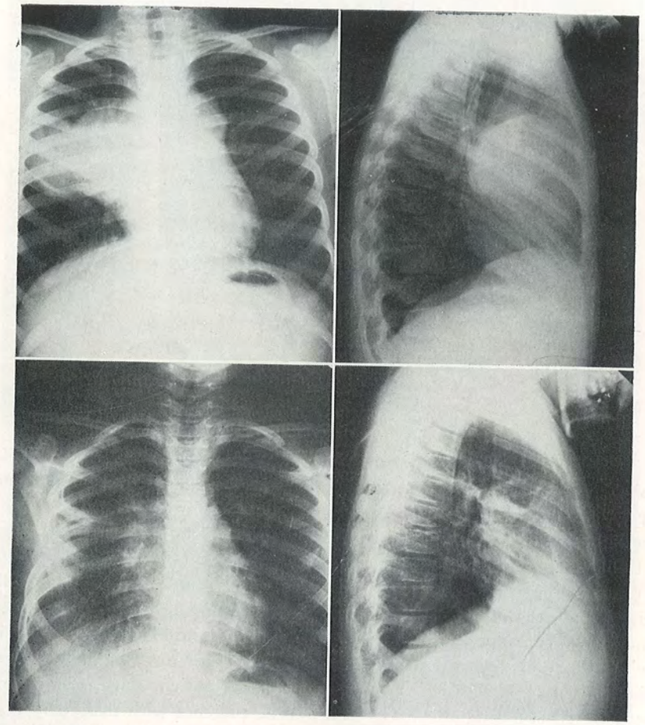


FIG. 10A. (*Top*) Lesion located anteriorly and intimately related to the pericardium, indicating teratoid or teratomatous tumor. (*Bottom*) Approximately 2 months postoperative, showing regenerated rib and slight persistent scarring.

These tumors are almost universally in the posterior mediastinum and present as smooth, rounded or oval and sometimes bosselated, shadows on the x-ray (Fig. 11 A, B). They are occasionally associated with von Recklinghausen's disease.

The symptoms are usually those of pain or disturbance of function of the affected nerves, with Horner's syndrome as evidence of the sympathetic disturbance in the upper or loss of sweating in lower locations of such lesions.

Bone erosion and hour-glass penetration of intervertebral foramina are common. At times, they reach large size, with consequent diminution in pulmonary expansion causing dyspnea. One of our patients was digitalized for months because of the great area of supposed cardiac dulness, the dyspnea and leg edema. All disappeared after operation (Fig. 12 A, B).

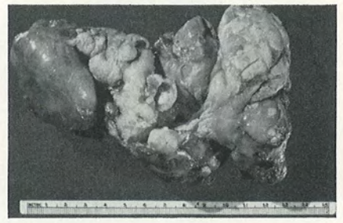


FIG. 10B. The teratoma removed. Skin, hair, bone, cartilage, rudimentary limb buds, sections of gastro-intestinal tract, pancreas, brain tissue with convolutions and peripheral nerves were all identified. The pathologic diagnosis was inclusio fetus in fetu.

The treatment consists of excision as soon as discovered, because of the probable increase in size or degeneration into malignancy. X-ray therapy has no appreciable effect on these and cannot be used as a substitute for surgery. The mortality of surgical removal should be low, although reports of the earlier cases reveal 30 per cent. The group of Kent[36] showed only 10 per cent mortality, that of Harrington[28] 6 per cent, and in our cases there were two deaths. One fatality followed exploration of an inoperable malignant ganglioneuroma; the other occurred at the end of removal of a huge malignant mesenchymoma from a six-month-old infant. Another inoperable lesion destroyed the patient after two years. A patient with incomplete removal lasted seven months. The other three patients with malignant lesions are alive and well one year, four years and ten years, respectively (Fig. 13). The last patient underwent removal of bilateral median nerve tumors, one of which was sarcomatous.

Of the eleven benign neurogenic tumors, one patient demonstrated two neurilemomas or schwannomas in the chest, three showed cystic degeneration and one was suspected preoperatively of being malignant because of associated pleural effusion.

The follow-up of from one to five years showing complete relief of the benign lesions is a happy contrast, emphasizing the value of early removal which may forestall malignant change.

Intrathoracic Thyroids. Intrathoracic goiters have been classified by Kocher[37] as those in which a portion remains permanently retrosternal;

Wakeley and Mulvany[59] describe them as those in which the majority or all of the swelling is within the thorax; and Clute and Lawrence[15] state that they extend to or below the arch of the aorta. By their definition, Clute

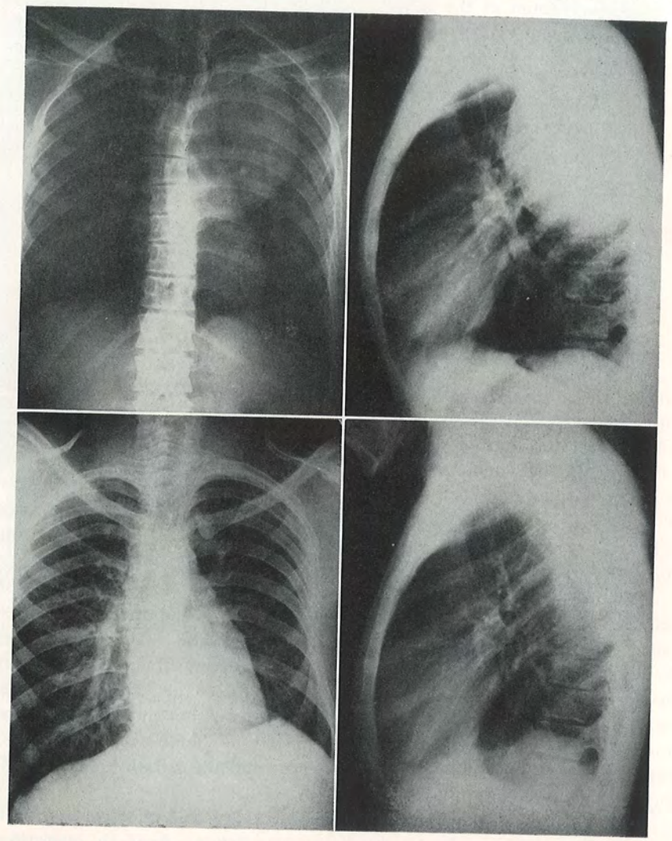


FIG. 11A. (*Top*) Typical smooth, oval outline and posterior location of neurogenic tumor. There is distortion of the alignment of the vertebrae, but no bone erosion, seen in the film on the left. (*Bottom*) Postoperative film.

and Lawrence feel that from 1 to 10 per cent of goiters are intrathoracic and if the substernal, retrosternal and subclavicular are included, the figures are from 12 to 30 per cent. Single adenomata are the usual causes of such extension, multiple adenomata the occasional, and the diffuse or exophthalmic type the extremely rare cause. Mora, Isaacs, Spencer and Edidin[46] reported

one case and collected six others of ectopic posterior mediastinal goitre. Sweet[54] reported another. One of ours fell in this category, and one other revealed malignancy.

The small outlet of the thorax, with its rigid bony margins, permits of considerable compression here, by even benign thyroid tissue, of trachea,

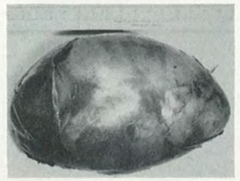


FIG. 11B. The large neurofibroma, with some of the pleura which covered it, removed from the patient.

esophagus and, rarely, of veins. Occasionally, recurrent nerve weakness without complete paralysis is observed. Usually, physical examination will indicate that the thyroid does extend behind the sternum, but occasionally this is found only upon x-ray examination as a superior mediastinal mass located anteriorly. Toxicity was present in seven of 20 cases reported by Wakeley and Mulvany.[59] Movement with deglutition, as observed fluoroscopically, or the appearance of the mass upon coughing are helpful in determination of the lesion.

Treatment consists of removal, which is naturally easier if the tumor has not been allowed to grow large. Lahey[39] advocates rupture of the capsule and decompression from within in the larger ones, and Adams[1] has reported the need for thoracic approach in three instances. Although the recurrent laryngeal nerve is usually pushed posteriorly, some of the retrotracheal masses may displace it anteriorly. It is usually difficult, if not impossible, to locate this nerve until the mass has been removed by blunt dissection from its intrathoracic position, when both the nerves and the parathyroids should be checked for injury.

Our ten cases include three combined cervical and thoracic approaches, one of which was for the intrathoracic mass left by previous operation in another state, when the cervical portion only had been removed. Following this the patient had required tracheotomy for a year. The tracheotomy was discarded when the thoracic adenoma was removed. He is well three years later. This patient, another with anterior location and the patient with the ectopic posterior mediastinal thyroid were each found to have small pedicles extending into the neck which could not be reached through the apex of the thorax and required cervical incision for that part of the blood supply. In one of these, the right recurrent laryngeal nerve was injured.

Cysts. There are two groups of cysts other than dermoids and the parasitic Echinococcus cysts which are rather intriguing. The one is of clear

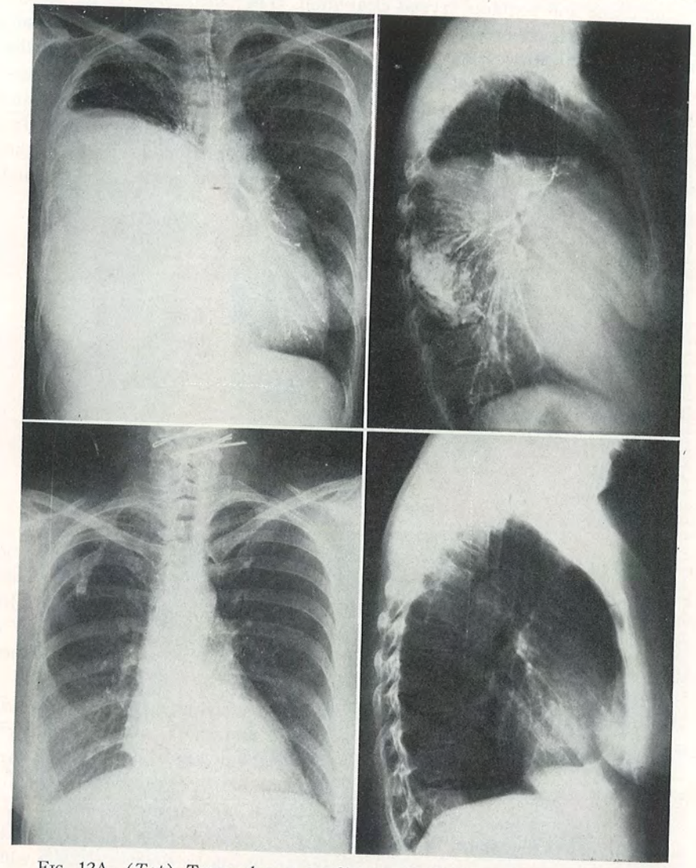


FIG. 12A. (*Top*) Tremendous neurofibroma displacing and compressing right lung, so that little lipiodol could be instilled in this side. (*Bottom*) After removal of large neurofibroma and disappearance of all symptoms, which had led to the diagnosis of congestive failure.

fluid, frequently called "spring water cyst," which is probably a pericardial celomic cyst, as suggested by Lambert,[40] who attributed the origin to failure of primitive mesenchymal lacunae, which form the pericardium, to fuse with others but instead to form independent cavities. A somewhat similar idea was presented by Lillie, McDonald and Clagett,[42] who expressed the

feeling that these cysts are remnants of the ventral parietal recess of the pericardial celom. It has a thin endothelial or mesothelial lining on loose fibrous tissue and contains crystal clear fluid. They usually lie in the cardiophrenic angle to either side, more commonly on the right, but occasionally are in communication with the pericardial cavity and constitute pericardial diverticuli. Dr. George Holmes[34] suggested that, when such a communication is present, a change in the patient's position will change the shape and size of the sac (Fig. 14). Even when the cysts are completely separated, they can be demonstrated as cysts by aspiration and instillation of air for radiographic demonstration of the thin wall and to obtain the typical limpid liquid,

FIG. 12B. The multilobulated and tremendous neurofibroma removed from the patient.

as occurred in two of our cases. Because of advanced myocardial disease one patient was not operated upon. The other is being observed after such demonstration. No case of malignant transformation has been reported. They are usually completely symptomless and rarely produce any evidence of enlargement. They do not demand removal unless symptoms or enlargement appear under periodic examination.

Before we knew how to diagnose them, two asymptomatic patients with such pericardial celomic cysts were explored and the cysts removed (Fig. 15). A pericardial diverticulum in another patient was excised during sympathectomy for hypertension. A sixth patient with pericardial diverticulum was demonstrated by positional change and was not operated upon.

In the other group are the bronchogenic or ciliated epithelial cysts and the gastro-enterogenous cysts or reduplications of the gastro-intestinal tract which are closely related. Of the former type, Heuer and Andrus[33] were able to collect 25 cases, Brown and Robbins[8] discussed twelve additional ones, and Maier[43] and Adams and Thornton[2] and a few others have reported small groups. Carlson[10] felt that these were the result of diverticuli of the entoderm and mesoderm from the foregut, secondary budding of the tracheal anlage or later, abnormal division of the respiratory buds or inverted closure of the communication between the trachea and esophagus. Besides ciliated columnar cells, they may contain other elements of the bronchi such as cartilage, mucous glands, fibrous tissue, elastic fibers, smooth muscle, nerves and vessels and may contain either fluid or air. Most of them seem to develop in the lung, where we have found several, but they are not rare in the mediastinum. We had one such case.

Many of these are asymptomatic while others, by size, location or infection, may cause cough or dyspnea.

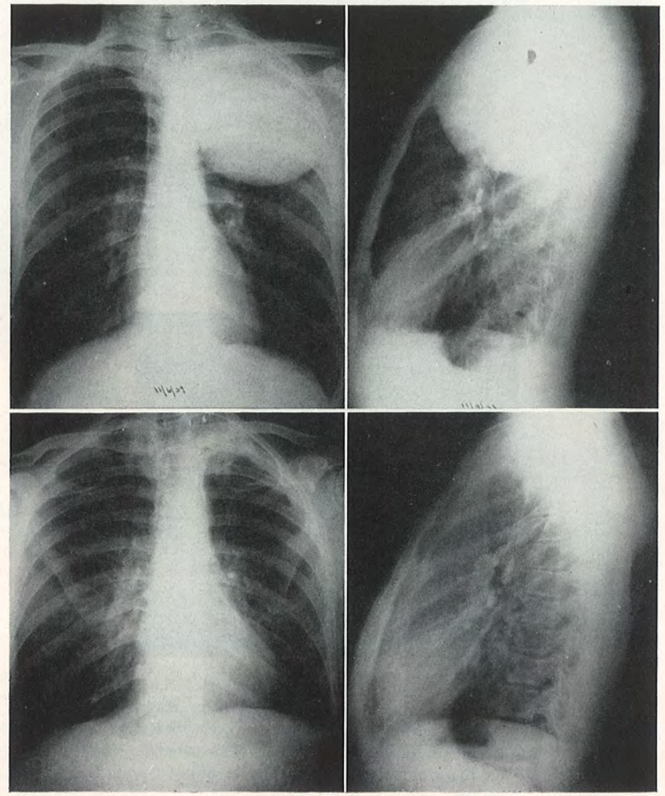


FIG. 13 (*Top*) Large neurogenic sarcoma, with great vascularity. This is too large to determine whether it is located anteriorly or posteriorly. Observation by x-ray and measurement for 6 months preceding operation showed no growth of the tumor. (*Bottom*) Postoperative, after two-stage removal, necessitated by hemorrhage encountered in the first stage. Bilateral median nerve tumors were excised locally with anastomosis of fasiculi involved. One of these tumors appeared malignant on microscopic study. The patient has remained well for 10 years.

Accessory lobes of the lung are closely akin to these cysts. In one asymptomatic case of ours, a tracheal lobe was removed.

Of the gastro-enterogenous cysts, Ladd[38] has reported five cases and Schwarz and Williams[53] two. Schwarz and Williams found five collected

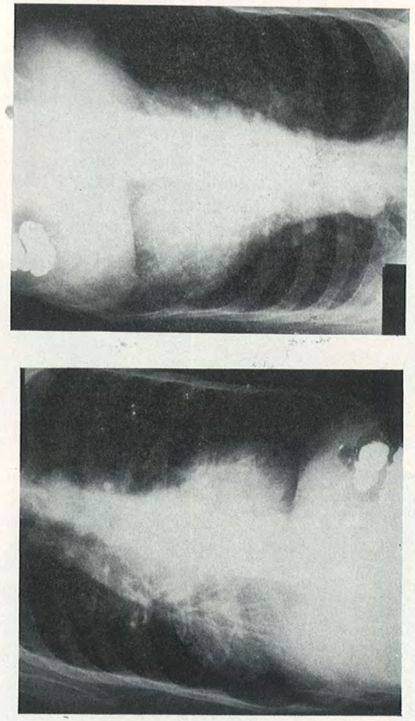
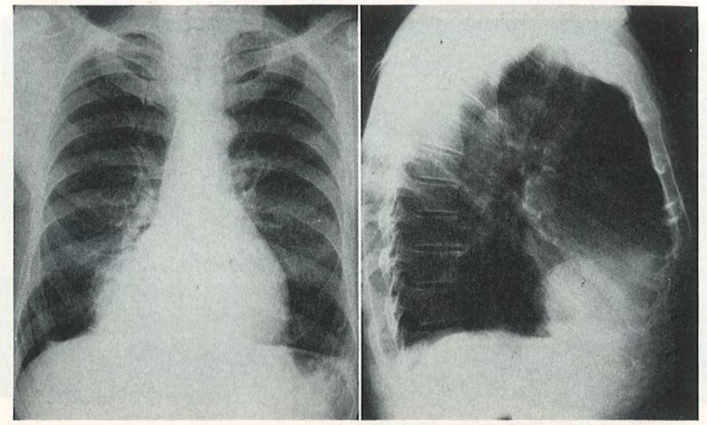


FIG. 14. Patient with right-sided pericardial diverticulum, demonstrating the Holmes' maneuver. With patient on left side (*top*), there is collapse of the diverticulum; with patient on the right side (*bottom*), wide filling is shown.

cases with evidence of secretory function in the cyst and seven with no activity. Some of the latter group contained also respiratory epithelium (as did one reported by Nicholls[49]) in addition to the gastric mucosa. They usually are lined by gastric mucosa but some have had also esophageal or intestinal mucous membrane. They may be in close association with the

esophagus or lie free in the mediastinum. In the case which Black and Benjamin[4] reported in 1936, the cyst extended through the diaphragm to communicate with the jejunum, forming a diverticulum—as did the one in our series. Our patient was reported before the Academy of Surgery of Phila-



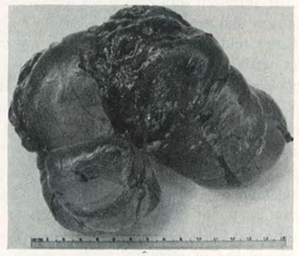


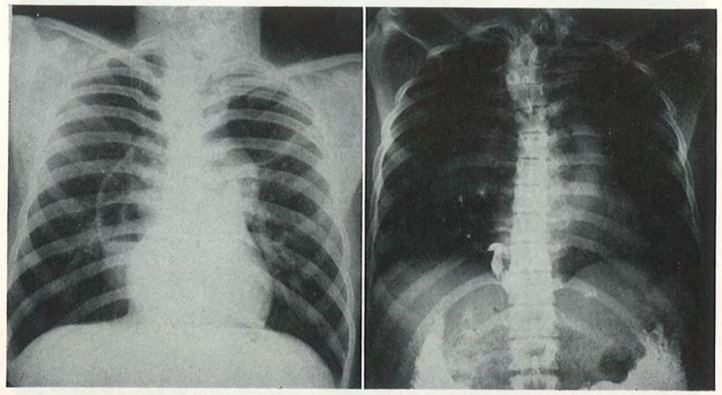
FIG. 15. The pericardial diverticulum in its usual location anteriorly and close to diaphragm on right side. (*Bottom*) The thin-walled cyst, filled with crystal-clear fluid, removed from patient.

delphia last year (Fig. 16). Gross, Neuhauser and Longino[26] reported three similar cases in 1950 and were apparently unaware of the previous report of Black and Benjamin.[4] Davis and Salkin[17] report one case of gastric mediastinal cyst and have collected 26 others. Surgery was tried in 17, with success in 10.

The symptoms are usually rather marked and are demonstrated early in life, frequently causing the death of the patient from inanition due to vomiting and other gastro-intestinal disturbances. Our patient had gone through a stormy course in infancy but had reached manhood, though there were still slight stigmata of maldevelopment.

Lipomas are infrequent, and Heuer and Andrus[33] have collected 13 cases protruding through the chest wall, five extending into the neck from the

mediastinum and 24 of the intrathoracic type, totaling 42 cases. They usually produce symptoms from pressure but occasionally become infected. Their consistency on x-ray is suggestive of fluid, and a boggy or buttery sensation is obtained on aspiration.



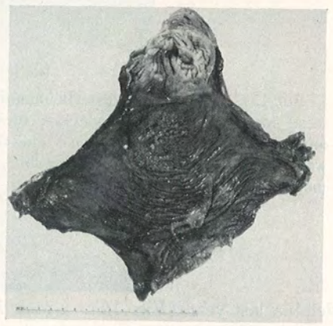
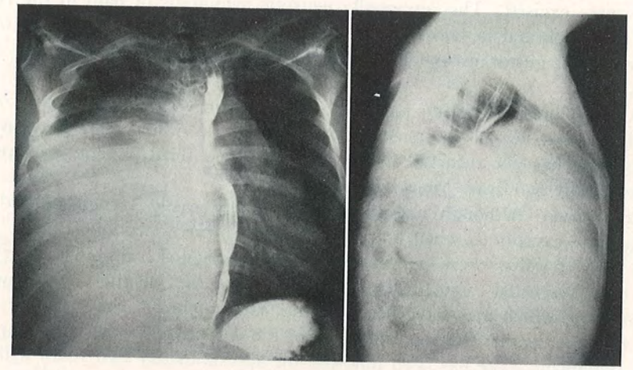


FIG. 16. Large diverticulum originating from the jejunum and extending through the diaphragm and mediastinum to be demonstrable in the middle right and upper left hemithorax with a fluid level near the right side of the pericardium. The film on the right shows barium entering the diverticulum and the sac so that some of it could be moved by postural change into the left upper portion of the sac. (*Bottom*) The large diverticulum of the jejunum extending through the mediastinum posterior to trachea and esophagus was removed with moderate difficulty down to just below the diaphragm, where it was resected, leaving a small diverticulum of the jejunum, which has caused no further symptoms. The wall contained mucosa of gastric and of enteric origin in different portions, as demonstrated in the open specimen above. The patient has been well for 3 years.

Although rarely malignant, they have, as a rule, contributed to the death of the patient unless removed, and considerable difficulty has been found in the removal of the large ones, although most of the reports are of earlier times before proper anesthesia and asepsis were available. Actually, the removal should be moderately easy, and mortality should be extremely low. Two instances of this type in our group were removed successfully.

Fibromas. Some 32 of these tumors were collected by Heuer and Andrus.[33] Clagett and Hausmann[12] have reported a huge one subsequent to this. In most instances these have appeared in the anterior mediastinum, but very large ones obscure their origin. The symptoms are not distinctive. In two of our patients, the fibroma was removed without incident. One required sacrifice of the right middle and lower lobes because of destruction due to



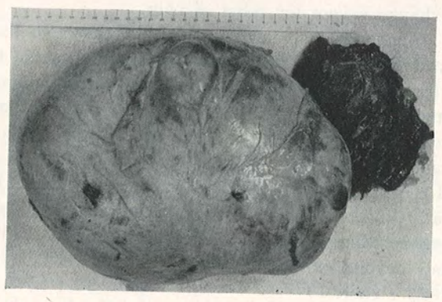


FIG. 17. Huge fibroma in the right hemithorax, causing marked damage to the right lower and middle lobes and so completely filling the hemithorax that the location of origin is not discernible in the x-ray. (*Bottom*) Specimen removed, with attached lower and middle lobes which had to be sacrificed.

compression of the bronchus, causing cough, dyspnea and hemoptysis (Fig. 17).

Lymphangioma. One asymptomatic lesion resembling neurofibroma on x-ray was excised from the posterior mediastinum.

Myxomas. One case of this type has been reported by Graham,[25] one by Heuer and Andrus[33] and one by Lemon.[41] The location has varied as have the symptoms, neither being distinctive.

One of our cases fell in this category. Cough, dyspnea, hemoptysis and sternal pain were his complaints. At exploration, the huge size of the lesion,

its firm adherence to the parietal pleura and the excessive bleeding resulting from attempts to separate a small area of adherence made us abandon the procedure. Only biopsy was accomplished. The patient lived for only three months after discharge. One extensive mesothelioma was explored and biopsied.

Chondrosarcoma. Three examples (one containing some osteoid tissue also) were excised. The supposedly neurotic symptoms of pain in the right neck and back and in both arms were completely relieved by excision from the vertebral gutter in one instance. The patient was alive and well two years later.

A huge tumor requiring removal of most of the sternum, adjacent costal cartilages, and medial portions of both clavicles, as well as the mediastinal component, has not recurred in ten months. In a third patient, recurrence caused death two years after removal.

Aneurysm. Although aneurysms are usually easily recognizable there are definite exceptions when no pulsations or a bizarre location occur.

Two such lesions were explored on erroneous diagnosis, although aneurysm had been suspected. One died of the rupture of the sac the day following exploration, which may have hastened this catastrophe.

One other patient having aneurysm of the descending aorta was wrapped with cellophane. She has been followed for approximately two years without showing much change except for relief of pain.

Discussion. In general, we have applied the principle that mediastinal masses are eventually harmful and potentially dangerous, that their character can be proven by appropriate diagnostic maneuvers, particularly exploration and that they must be actively treated, except for a rare and specified exception. We have been convinced of the value of this concept and of the rewarding salvage which it permits. The surgical mortality is low and still decreasing, and a more general application of these principles by the medical public can greatly improve the results by earlier attack. Mass surveys by x-ray discover many asymptomatic ones. Since all mediastinal masses which are not obviously beyond extirpation should be removed, exploration should be employed freely (Table 1).

Conclusions

1. A superficial review of the literature and our own experience indicates the danger of mediastinal lesions. The laissez-faire attitude of a large proportion of the profession concerning the asymptomatic cases should be abandoned.
2. Diagnostic procedures are clarified.
3. Our experiences with sixty-five operative patients are recounted.
4. Since pericardial celomic cysts usually can be diagnosed accurately, it is possible that the asymptomatic ones may be treated conservatively unless they grow larger.
5. The salvage in benign lesions is tremendous, and a few cases of malignancy may possibly be cured.

6. Malignancy was present in 27.7 per cent of the patients.
7. The mortality of surgical treatment is decreasing rapidly. In our series, 11 malignant lesions were excised and 7 were only explored and biopsied, while 44 benign lesions were excised and 3 were not removed. There was one operative death in each of the four groups, an over-all mortality of 6.1 per cent.

TABLE 1. MEDIASTINAL LESIONS TREATED SURGICALLY

	Benign	Malignant	Total	Died	Not Operated
Neurogenic	11	7	18	2	1
Thyroid	9	1	10		
Teratoid	8	2	10	1	7
Thymoma	1	2	3		1
Fibroma, lipoma and tuberculoma (2 each)	6		6		
Mesenchymal sarcoma		4	4		
Lymphoblastoma		2	2		
Aneurysm	2		2	1	
Pericardial celomic cysts	3		3		3
Gastrogenic (2); Bronchogenic, unclassified, tracheal lobe, lymphangioma and myxoma (1 each)	7		7		2
Totals	47	18 (27.7%)	65	4 (6.1%)	14

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TRANSACTIONS
OF THE
PHILADELPHIA ACADEMY OF SURGERY

THE YEAR 1943

Meeting of January 4, 1943, Cadwalader Hall, College of Physicians. The President, Dr. Robert H. Ivy, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

P. A. McCarthy, M.D., and Eli Saleeby, M.D. (By invitation) — Strangulated Obturator Hernia in a Patient Eighty Years Old.

Papers.

C. A. Whitcomb, M.D. (By invitation) — Carcinoma of the Lip. A Review of Fifty-six Five Year Cases.

Hubley Owen, M.D. — Zuckerman's Theory on the Effect of Blast.

Paper of the Evening.

Lt. Comdr. R. P. Kreuz and Otto Stader, M.D. (By invitation) — Application of the Stader Reduction Splint in the Treatment of Fractures and Other Orthopedic Conditions.

Joint Meeting of the Philadelphia Academy of Surgery and the New York Surgical Society on February 10, 1943, New York Academy of Medicine.

SCIENTIFIC PROGRAM

Bronson S. Ray, M.D. — Autonomic Nerve Supply to the Upper Extremity and Pupil as Demonstrated by Direct Stimulation of Anterior Roots at the Operating Table.

Discussed by:
Francis C. Grant, M.D.
Beverly Smith, M.D.
Benjamin Lipshutz, M.D.

ARTHUR H. BLAKEMORE, M.D. (By invitation) — The Severed Primary Artery in the War Wounded. A Non-Suture Method of Bridging an Arterial Defect.
Discussed by:
BENJAMIN LIPSHUTZ, M.D.
WILLIAM BATES, M.D.

JOHN E. SUTTON, M.D. — Postoperative Necrosis of the Liver. An Experimental Study.
Discussed by:
ADOLPH WALKLING, M.D.

WILLIAM DEWITT ANDRUS, M.D. and JERE W. LORD, M.D. (By invitation) and MR. PAUL STEFKO (By invitation) — The Effects of Pedicle Jejunal Grafts on Gastric Secretion.
Discussed by:
CALVIN M. SMYTH, M.D.
GEORGE J. HEUER, M.D.

JAMES A. DINGWALL, M.D. (By invitation) — Treatment of Burns with Sulfafilm.
Discussed by:
DR. SALTONSTALL
DR. BECKMAN
DR. BANCROFT
DR. WEBSTER
ALLEN O. WHIPPLE, M.D.

R. FRANKLIN CARTER, M.D. — Hepatocholecystenterostomy for Relief of Jaundice in Obliteration of the Hepatic Ducts.
Discussed by:
THOMAS A. SHALLOW, M.D.
S. DANA WEEDER, M.D.

RALPH COLP, M.D. — Modified Whipple Procedure for Adeno-Carcinoma of the Terminal Portion of the Common Bile Duct.
Discussed by:
DAMON B. PFEIFFER, M.D.
ALLEN O. WHIPPLE, M.D.

DEWITT STETTEN, M.D. — Volvulus of Stomach with Incarceration of Antrum in Diaphragmatic Hernia and Complete Acute Gastric Obstruction. Operative Recovery with Obliteration of Hernial Sac by Tamponade.
Discussed by:
S. DANA WEEDER, M.D.

Meeting of March 1, 1943, Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Presentation of a Patient.
DRURY HINTON, M.D. — Recurrent Dislocation of the Elbow.

Case Reports.
DRURY HINTON, M.D. — Pinning of the Neck of the Femur in a Man One Hundred Years Old.

WILLIAM T. LEMMON, M.D., and HENRY G. HAGER, JR., M.D. (By invitation) — Common Duct Stones Causing Jaundice Complicated by Hepatic Duct Stone, Cholelithiasis, Cholecystitis, Duodenal Ulcer and Duodenal Fistula. Recovery After Operation.

Paper.
HENRY SANGREE, M.D. (By invitation) — Urethral Plastic Operations for Male Infertility.

Paper of the Evening.
HAROLD L. FOSS, M.D. (By invitation) — Group Practice and the Rural Hospital.

Meeting of April 5, 1943, Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Papers.
S. BRANDT ROSE, M.D. (By invitation) — A New Blood and Plasma Filter.

JOHN S. LOCKWOOD, M.D. — The Use of Penicillin in Staphylococcic Infections.

JONATHAN E. RHOADS, M.D., and HAROLD A. ZINTEL, M.D. (By invitation) — The Use of Ammonium Chloride in the Treatment of Alkalosis in Surgical Patients.

Paper of the Evening.
DONALD GUTHRIE, M.D. (By invitation) — The Preoperative Preparation of the Thyrotoxic Patient.

Meeting of May 3, 1943, Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.
JAMES A. LEHMAN, M.D. — Brien King Operation for the Relief of Advanced Paralysis of the Larynx.

S. DANA WEEDER, M.D. — Intra-Abdominal Apoplexy.

Papers.

Robert H. Ivy, M.D., and Lawrence Curtis, M.D.	Deformities of the Lower Jaw with Special Reference to Protrusion and Retrusion.
Adolph A. Walkling, M.D., and Frank Allbritten, M.D. (By invitation) and Stanley Potter, M.D. (By invitation)	Experience with Biodyne in Skin Regeneration.

Paper of the Evening.

William L. Estes, M.D. (By invitation)	Partial Cholecystectomy and Indications for Its Use.

Meeting of October 4, 1943, Cadwalader Hall, College of Physicians. The President, Dr. Robert H. Ivy, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Patients.

Calvin M. Smyth, Jr., MD., and James F. Schell, M.D., and T. A. Ranieri, M.D. (By invitation)	End Results in Three Children with Major Third Degree Burns Treated by Three Different Methods.

Case Report.

J. Walter Levering, M.D.	Chylous Ascites.

Paper.

Damon B. Pfeiffer, M.D., and F. M. Simmons Patterson, M.D. (By invitation)	The Surgical Management of Acute Cholecystitis.

Paper of the Evening.

Lt. Col. J. Barrett Brown, M.C., U.S.A. (By invitation)	The Surgical Repair of Deep Burns.

Meeting of November 1, 1943, Cadwalader Hall, College of Physicians. The President, Dr. Robert H. Ivy, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

Eli Saleeby, M.D. (By invitation)	Teratoma of the Testicle with Metastasis to Epigastrium. Bilateral Orchidectomy. Recovery.

Presentation of Patient.

Lawrence Curtis, M.D.	Severe Injury to Soft Tissues and Bones of the Face.

Paper.

Hans May, M.D.	Closure of Large Defects of Lower Lip and Chin.

Annual Oration.

Frederick A. Bothe, M.D.	The Future of Surgery in Metabolic Diseases.

Meeting of December 6, 1943, Cadwalader Hall, College of Physicians. The President, Dr. Robert H. Ivy, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Patient.

John Brooke, M.D., and William L. Martin, M.D. (By invitation)	Hind Quarter Amputation for Osteo-Chondroma of Hip.

Paper.

Lt. Col. Harold J. Dunlap, M.C., and Capt. J. Edward Berk, M.C. (By invitation)	Hypertrophic Pyloric Stenosis in Adults with Report of Two Cases.

Case Report.

Thomas A. Shallow, M.D., and Sherman A. Eger, M.D. (By invitation)	Primary Carcinoma of the Third Portion of the Duodenum Successfully Resected.

Paper of the Evening.

Rudolph Jaeger, M.D. (By invitation)	Intervertebral Disc Injury with Herniation of Nucleus Pulposus, Illustrated by Colored Motion Picture.

REPORT OF THE SECRETARY FOR THE YEAR 1943

During the year of 1943, the Academy held seven stated meetings and one Conjoint Meeting with the New York Surgical Society. At the seven stated meetings there was an average attendance of thirty Fellows and thirty-two guests in addition.

The Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery was held in New York City at the New York Academy of Medicine on February 10, 1943. Following an excellent program, dinner for the combined Societies was held at the Harvard Club.

During the year there were eighteen papers, eight case reports, four presentations of cases. Twelve of these papers were presented by guests, six by Fellows. Seven of the eight case reports were by Fellows. There were seventy-seven discussers of these presentations. Three candidates were elected to Fellowship (Doctors Paul C. Colonna, Edward F. McLaughlin and Jonathan E. Rhoads). Two Active Fellows were transferred to the Senior List.

The Annual Oration was pronounced by Doctor Frederick A. Bothe on November 1st on the subject of "The Future of Surgery in Metabolic Diseases."

The Nominating Committee, consisting of Doctors Rodman, Muller and Eliason appointed by the Chair, presented the following nominations for officers to serve the year 1944:

President—Dr. Hubley R. Owen.
First Vice-President—Dr. John B. Flick.
Second Vice-President—Dr. Thomas A. Shallow.
Secretary—Dr. Calvin M. Smyth, Jr.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Robert H. Ivy and Dr. Eldridge L. Eliason.
Business Committee—Dr. Frederick A. Bothe, Chairman, with Dr. W. Emory Burnett and the Recorder.

Respectfully submitted,

CALVIN M. SMYTH, JR., M.D.
Secretary

THE YEAR 1944

Meeting of January 3, 1944, Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Report.
ADOLPH A. WALKLING, M.D., and CLARE C. HODGE, M.D. (By invitation) — Carcinoma of the Common Bile Duct. Excision and Reconstruction by the Use of a Vitallium Tube.

Case Report and Motion Picture Demonstration.
WILLIAM T. LEMMON, M.D., and SHERMAN A. EGER, M.D. (By invitation) — Carotid Aneurysm. Terminal Ligation Followed by Obliterative Endo-Aneurysmorrhaphy.

Paper.
W. EMORY BURNETT, M.D. — A System for the Prevention of Symptoms After Gallbladder Surgery.

Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery on February 9, 1944, Mitchell Hall, College of Physicians. The Presidents, DR. HUBLEY OWEN and DR. WILLIAM WHITE, presiding.

SCIENTIFIC PROGRAM

WARREN B. DAVIS, M.D. — Basal Cell Lesions of the Nose and Lip.
Discussed by:
JEROME P. WEBSTER, M.D.
JOHN GERSTER, M.D.

ELDRIDGE L. ELIASON, M.D. — Unusual Kidney Tumor.
Discussed by:
GEORGE CAHILL, M.D.

FRANCIS C. GRANT, M.D. — Experiences with Operative Treatment of Ruptured Intervertebral Discs.
Discussed by:
PHILIP D. WILSON, M.D.
JOHN R. MOORE, M.D.
BRONSON RAY, M.D.

GEORGE P. MULLER, M.D. — Adenoma of Bronchus.
Discussed by:
ALEXANDER E. W. ADA, M.D.

WALTER ESTELL LEE, M.D., and JONATHAN E. RHOADS, M.D. — Relation of the Crush Syndrome to That of Burns and Other Types of Traumatic Wounds of Human Tissues.
Discussed by:
SAMUEL STANDARD, M.D.

THOMAS A. SHALLOW, M.D. — Inflammatory Lesions of the Pancreas and Their Complications.
Discussed by:
R. FRANKLIN CARTER, M.D.

DAMON B. PFEIFFER, M.D. — Surgical Aspects of Certain Blood Dyscrasias.
Discussed by:
WILLIAM DEW. ANDRUS, M.D.

J. STEWART RODMAN, M.D. — Primary Closure Versus Mickulicz Procedure in Resection of Colon.
Discussed by:
FREDERIC W. BANCROFT, M.D.
HENRY CAVE, M.D.
JOHN GERSTER, M.D.
DAMON B. PFEIFFER, M.D.

Meeting of March 6, 1944, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

Papers.

Calvin M. Smyth, Jr., M.D., and T. A. Ranieri, M.D. (By invitation)	The Management of Compound Injuries of the Skull. Presentation of Four Patients.
Irvin E. Deibert, M.D.	Paraplegia Following Spinal Anesthesia.
John Royal Moore, M.D.	Experiences with Delayed Reductions of Fractures.

Meeting of April 3, 1944, Cadwalader Hall, College of Physicians. The Vice-President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

William Erb, M.D.	Atresia of the Intestine in Infants.
S. Dana Weeder, M.D.	Meckel's Diverticulum Complicated by Perforation and Hemorrhage.
David Davis, M.D.	Intubated Ureterotomy.

Meeting of May 1, 1944, Cadwalader Hall, College of Physicians. The President, Dr. Hubley R. Owen, in the Chair.

SCIENTIFIC PROGRAM

J. Walker, Jr., M.D. (By invitation)	A Controllable Method of Reducing Blood Coagulability.
Gilson C. Engel, M.D.	Appendiceal Abscess Treated by Intra-Abdominal Sulfanilamide and Non-Drainage. Report of Two Cases.
George M. Dorrance, M.D., and S. G. Castigliano, M.D. (By invitation)	Selection of Method of Treatment in Epidermoid Carcinoma of the Head and Neck.

Meeting of October 2, 1944, Cadwalader Hall, College of Physicians. The President, Dr. Hubley R. Owen, in the Chair.

SCIENTIFIC PROGRAM

Jonathan E. Rhoads, M.D., and C. Everett Koop, M.D. (By invitation)	The Use of Specially Prepared Gelatin Solutions as a Plasma Substitute.

Hoke Wammock, M.D., (By invitation) and Willard S. Hastings, M.D. (By invitation)	The Borderline Between Cystic Disease and Carcinoma of the Breast.
Harvey B. Stone, M.D. (By invitation)	Intestinal Obstruction.

Meeting of November 6, 1944, Cadwalader Hall, College of Physicians. The President, Dr. Hubley R. Owen, in the Chair.

SCIENTIFIC PROGRAM

Edward T. Crossan, M.D.	Intestinal Obstruction. Report of Three Cases.
Adolph A. Walkling, M.D., and William J. Tourish, M.D. (By invitation)	Thiouracil. Its Use in the Preparation of Thyro-Toxic Patients for Surgery.
Frederick W. Bancroft, M.D (By invitation)	Thrombosis and Embolism with Particular Reference to Proximal Ligation and Veno-Thrombectomy.

Meeting of December 4, 1944, Cadwalader Hall, College of Physicians. The President, Dr. Hubley R. Owen, in the Chair.

SCIENTIFIC PROGRAM

Memoir.

Irvin E. Deibert, M.D.	Memoir of the Late Benjamin Franklin Buzby, M.D.

Case Report.

W. Emory Burnett, M.D., and George P. Rosemond, M.D. (By invitation)	Pyo-pneumo-thorax in Infants of Less than One Month. Report of Two Patients with Operation and Recovery.

Annual Oration.

Calvin M. Smyth, Jr., M.D.	Graduate Education in American Surgery.

REPORT OF THE SECRETARY FOR THE YEAR 1944

During the year 1944, the Philadelphia Academy of Surgery held seven stated meetings and one Conjoint Meeting with the New York Surgical Society. At the seven stated meetings, there was an average attendance of twenty-three Fellows and forty-two guests.

The Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery was held in Mitchell Hall, College of Physicians, Philadelphia, on February 9, 1944. Seven papers were presented. This meeting was attended by thirty-five Fellows of the Philadelphia Academy of Surgery, twenty-seven members of the New York Surgical Society, and sixty-five guests. It was followed by a dinner at the Racquet Club.

During the year, exclusive of the Conjoint Meeting, there were eight case reports and eleven papers presented, of which eight case reports were by Fellows; eight papers were by Fellows and three by guests. There were ninety discussions, seventy-two by Fellows and eighteen by guests.

One memoir was read. (The Late Benjamin F. Buzby, M.D.)

The Annual Oration was pronounced by Doctor Calvin M. Smyth, Jr., on December 4, 1944, on the subject of "Graduate Education in American Surgery."

Two Active Fellows were transferred to the Senior List. (Doctors I. S. Ravdin and Calvin M. Smyth, Jr.)

Two candidates were elected to Active Fellowship. (Doctors John A. Brooke and Sherman A. Eger.)

The Nominating Committee consisting of Doctors Rodman, Muller and Eliason, appointed by the President, recommended the election of the following officers for 1945:

President—Dr. Hubley R. Owen.
First Vice-President—Dr. John B. Flick.
Second Vice-President—Dr. Thomas A. Shallow.
Secretary—Dr. Calvin M. Smyth, Jr.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Eldridge L. Eliason and Dr. Robert H. Ivy.
Business Committee—Dr. Frederick A. Bothe, Chairman, with Dr. W. Emory Burnett and the Recorder.

Respectfully submitted,

CALVIN M. SMYTH, JR., M.D.
Secretary

THE YEAR 1945

Meeting of January 8, 1945, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

Demonstration.

ADOLPH A. WALKLING, M.D., and CHARLES A. HATFIELD, M.D., (By invitation) and ROBERT BUYERS, M.D. (By invitation) — Fluorescein: Its Use in Determining Circulation in Strangulated Intestine.

Case Report.

THOMAS A. SHALLOW, M.D., and FREDERICK WAGNER, M.D. (By invitation) — Coincident Pancreatic and Liver Abscesses. Operation with Recovery.

Paper.

A. BRUCE GILL, M.D. — The Mechanical and Physiological Explanation of Low-Back Strain.

Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery on February 14, 1945, New York Academy of Medicine. The President of the New York Surgical Society, DR. WILLIAM C. WHITE, and the President of the Philadelphia Academy of Surgery, DR. HUBLEY R. OWEN, jointly presided.

SCIENTIFIC PROGRAM

JEROME P. WEBSTER, M.D. — Surgical Treatment of Gynecomastia
Discussed by:
DAVID M. DAVIS, M.D.
WILLIAM C. WHITE, M.D.

EDWARD J. DONOVAN, M.D. — Congenital Diaphragmatic Hernia.
Discussors not known

ALLEN O. WHIPPLE, M.D. — Pancreaticoduodenectomy for Islet Carcinoma with a Five Year Follow Up.
Discussed by:
DAMON B. PFEIFFER, M.D.
WILLIAM C. WHITE, M.D.

OTTO C. PICKHARDT, M.D. — Phrenemphraxis in the Treatment of Hiatus Hernia.
Discussed by:
GEORGE P. MULLER, M.D.
HUBLEY R. OWEN, M.D.

ANDRE COURNAND, M.D. (By invitation) — The State of Circulation in Severe Trauma, Hemorrhage, Abdominal Injury and Burns.
Discussed by:
JONATHAN E. RHOADS, M.D.

COLONEL ROBERT H. KENNEDY — Observations on Fracture Treatment in the Army.
Discussed by:
HUBLEY R. OWEN, M.D.

Meeting of March 5, 1945, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

DAVID M. DAVIS, M.D., and GEORGE P. MULLER, M.D. — A Case of Pyelo-Duodenal Fistula.

W. EMORY BURNETT, M.D. — The Surgery of Bleeding Peptic Ulcer. Report of Cases.

Paper of the Evening.
CAPT. WALTMAN WALTERS, M.C., U.S.N.R., Chief of Surgical Service, U. S. Naval Hospital, Philadelphia, Pa. — The Problem of Peptic Ulcer.

Meeting of April 2, 1945, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

THOMAS J. SUMMEY, M.D., and LAWRENCE PRESSLEY, M.D. (By invitation) — Osteogenic Sarcoma Complicating Paget's Disease.

THOMAS A. SHALLOW, M.D. — Extraperitoneal Closure of Colostomy: A New Technique. Report of Seventy-five Cases.

Paper of the Evening.
HENRY W. CAVE, M.D., Surgeon-in-Chief, Roosevelt Hospital, New York, N. Y. (By invitation) — Surgical Management of Chronic Ulcerative Colitis.

Meeting of May 7, 1945, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

WILLIAM H. ERB, M.D. — Ureteroperitoneal Anastomosis in a Case of Ascites Due to Portal Cirrhosis.

JONATHAN E. RHOADS, M.D., and C. EVERETT KOOP, M.D. (By invitation) — Measurements of the Effectiveness of Parenteral Feedings in Surgical Patients.

Paper of the Evening.
HANS MAY, M.D. — Correction of Cicatricial Contraction of Axilla, Elbow, and Knee.

Meeting of October 1, 1945, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

Memoir.
A. BRUCE GILL, M.D. — Memoir of the Late Edward B. Hodge, M.D.

Case Report.
JOHN C. HOWELL, M.D. — A Case of Elephantiasis.

Paper.
JONATHAN E. RHOADS, M.D. — The Use of Streptomycin in Surgical Patients.

Paper of the Evening.
WILLIAM E. LADD, M.D., Professor of Child Surgery, Harvard University, Boston, Massachusetts — Atresia of the Esophagus.

Meeting of November 5, 1945, Cadwalader Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

Case Report.
GILSON C. ENGEL, M.D., and R. GAYLE SPANN, M.D. (By invitation) — Report of a Case of Jaundice Secondary to Perforation of a Duodenal Ulcer.

Paper.
ALBERT E. BOTHE, M.D. — Symptomless Lesions of the Upper Urinary Tract.

Paper of the Evening.

GEORGE P. MULLER, M.D., and JAMES M. SURVER, M.D. (By invitation)	Five Years' Survival Following Radical Mastectomy for Carcinoma of the Breast.

Meeting of December 3, 1945, Thompson Hall, College of Physicians. The President, DR. HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

THOMAS J. RYAN, M.D.	Total Excision of the Patella for Fracture.
ROBERT H. IVY, M.D.	Repair of Deformity of Face and Upper Extremity Due to Acid Burns.

Annual Oration.

ADOLPH A. WALKLING, M.D.	Inguinal Hernia with Special Reference to Strangulation.

REPORT OF THE SECRETARY FOR THE YEAR 1945

During the year 1945, the Philadelphia Academy of Surgery held seven stated meetings and one Conjoint Meeting with the New York Surgical Society. At the seven stated meetings, there was an average attendance of thirty-one Fellows and sixty-two guests.

The Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery was held at the New York Academy of Medicine on February 14, 1945. Twenty-eight Fellows of the Philadelphia Academy of Surgery attended. Following an excellent program, dinner for the combined societies was held at the Harvard Club.

During the year, there were nine case reports and thirteen papers presented, of which nine case reports and ten papers were by Fellows; three papers were by guests. There were seventy-six discussions by Fellows and nineteen by guests, making a total of ninety-five.

Three candidates were elected to Active Fellowship. (Doctors Herbert R. Hawthorne, Charles A. Steiner, and George P. Rosemond.)

The following Fellows died during the year: Doctors Robert S. Alston, William John Ryan, James A. Kelly, and Edward B. Hodge.

One Memoir was read. (The Late Edward B. Hodge, M.D.)

The Annual Oration was pronounced by Doctor Adolph A. Walkling on December 3, 1945, on the subject, "Inguinal Hernia with Special Reference to Strangulation."

The Samuel D. Gross Prize was awarded to Doctor Robert Elman of St. Louis, Missouri, for his essay on "Parenteral Alimentation in Surgery with Special Reference to Protein and Amino Acids."

On October 29, 1945, the Academy was the guest of the U. S. Naval Hospital, Philadelphia. An interesting and constructive program was presented and was attended by fifty-four Fellows.

The Nominating Committee consisting of Doctors Rodman, Ivy and Pfeiffer, appointed by the President, recommended the election of the following officers for 1946:

President—Dr. John B. Flick.
First Vice-President—Dr. Thomas A. Shallow.
Second Vice-President—Dr. Calvin M. Smyth, Jr.
Secretary—Dr. L. Kraeer Ferguson.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Robert H. Ivy and Dr. Hubley R. Owen.
Business Committee—Dr. Frederick A. Bothe, Chairman, with Dr. J. Montgomery Deaver and Dr. W. Emory Burnett.

Respectfully submitted,

CALVIN M. SMYTH, JR., M.D.
Secretary

THE YEAR 1946

Meeting of January 7, 1946, Thompson Hall, College of Physicians. The President, HUBLEY R. OWEN, in the Chair.

SCIENTIFIC PROGRAM

WILLIAM H. ERB, M.D., and ROBERT D. DRIPPS, JR., M.D. (By invitation)	Cardiac Standstill with Resuscitation, Followed by Pneumonectomy Four Months Later.
ELDRIDGE L. ELIASON, M.D., and ROBERT F. WELTY, M.D. (By invitation)	Report of Three Cases of Spontaneous Rupture of the Esophagus.

Paper of the Evening.

CLAY RAY MURRAY, M.D., Professor of Orthopedic Surgery, College of Physicians and Surgeons, Columbia University, New York City.	An Evaluation of the Present Status of the Treatment of Compound Fractures.

Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery on February 13, 1946, Mitchell Hall, College of Physicians. The President of the Philadelphia Academy of Surgery, DR. JOHN B. FLICK, and the President of the New York Surgical Society, DR. JOHN A MCCREERY, jointly presided.

SCIENTIFIC PROGRAM

PAUL C. COLONNA, M.D.	The Treatment of Bone Defects with Massive Bone Graft.
I. S. RAVDIN, M.D.	Report of Eighteen Cases of Parathyroid Tumor.
CALVIN M. SMYTH, JR., M.D.	Malignant Synovioma. Case Report.
W. EMORY BURNETT, M.D.	Cysts of the Lung.
FRANCIS C. GRANT, M.D.	The Value of Color Photography in Teaching Surgery.
LEON HERMAN, M.D.	Personal Experiences with Injuries to the Ureter.
FREDERICK A. BOTHE, M.D.	Sympathectomy and Hypertension. Two Case Reports.

Meeting of March 4, 1946, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

S. DANA WEEDER, M.D.	A Direction Finder for Internal Fixation of Fractures of the Hip.
L. KRAEER FERGUSON, M.D., and JOSEPH J. JACOBSON, M.D. (By invitation)	Neurogenic Tumor of the Stomach.
Paper of the Evening. HANS MAY, M.D.	Transplantation and Transposition of Tendons in Reparative Surgery of the Hand.

Meeting of April 8, 1946, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

THOMAS J. RYAN, M.D.	Omphalocele. Successful Repair on Infant, Six Hours Old.
THOMAS A. SHALLOW, M.D.	Fibro-sarcoma of the Left Lobe of the Liver.
GEORGE N. J. SOMMER, JR., M.D. (By invitation)	The Treatment of Hemothorax and Empyema Following Thoracic Wounds.

Meeting of May 6, 1946, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

JOHN H. GIBBON, JR., M.D.	Myositis Ossificans Involving Both Triceps Brachii Muscles with Bilateral Ankylosis of Elbow Joints.
JOSEPH W. STAYMAN, JR., M.D. (By invitation)	The Successful Operative Management of a Case of Perforated Carcinoma of the Rectum Complicating Pregnancy.
JOHN ROYAL MOORE, M.D.	The Delayed Bone Graft in Congenital Pseudo-Arthrosis in Children.

Meeting of October 7, 1946, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

J. MONTGOMERY DEAVER, M.D., and A. G. MARTIN, M.D. (By invitation)	Congenital Atresia of the Colon.
HARRY E. KNOX, M.D., and J. G. LOGUE, M.D. (By invitation)	Ruptured Bladder in Premature Five-day-old Infant. Case Report.
Paper of the Evening. I. RIDGEWAY TRIMBLE, M.D., Assistant Professor of Surgery, Johns Hopkins Medical School; Assistant Professor of Surgery, University of Maryland Medical School; Formerly, Surgical Consultant in the Pacific Area Under General MacArthur.	Further Observations on Operations About the Head of the Pancreas.

Meeting of November 4, 1946, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

GEORGE WILLAUER, M.D.	Continuous Spinal Anesthesia for Thoracoplasty.
FRANK F. ALLBRITTEN, M.D. (By invitation)	Simultaneous Repair of Soft Tissue and Nerve Defects in the Forearm.
FRANCIS C. GRANT, M.D.	Operative Results in Meningioma of the Brain.

Meeting of December 2, 1946, Thompson Hall, College of Physicians. The President, Dr. John B. Flick, in the Chair.

SCIENTIFIC PROGRAM

Rudolph Jaeger, M.D.	Treatment of Malignant Exophthalmus by Intracranial Decompression of the Orbit.
Frederick R. Robbins, M.D.	Report of Two Thousand Four Hundred Consecutive Cases of Appendicitis in a U. S. Naval Hospital with Emphasis on Fatal Cases.
Annual Oration.	
John H. Gibbon, Jr., M.D.	The Pathogenesis and Treatment of Pulmonary Edema in Relation to Surgery.

REPORT OF THE SECRETARY FOR THE YEAR 1946

During the year 1946, the Philadelphia Academy of Surgery held seven stated meetings and one Conjoint Meeting with the New York Surgical Society. At the seven stated meetings there was an average attendance of thirty-three Fellows and forty-eight guests. During the year, the Academy voted to move to Thompson Hall in the College of Physicians since previous quarters had been so crowded.

Papers were presented by twelve Fellows and case reports by three Fellows. Guests of the Academy presented papers on seven occasions and case reports were presented by guests on two occasions. Discussions of papers were given from the floor by Fellows of the Academy in sixty-one instances and by five guests.

On February 13, 1946, the New York Surgical Society was the guest of the Philadelphia Academy of Surgery. The scientific portion of the program was held in Mitchell Hall of the College of Physicians and sixty-eight members of the New York Surgical Society were our guests. A very interesting program was presented which was discussed by members of the New York Surgical Society, after which a dinner was enjoyed at the Racquet Club.

The Annual Oration was given on December 2, 1946, by Doctor John H. Gibbon, Jr., on the subject "The Pathogenesis and Treatment of Pulmonary Edema in Relation to Surgery."

During the year the following men became Senior Fellows of the Academy: Doctors John O. Bower, Patrick A. McCarthy, Joseph C. Birdsall, and John F. McCloskey.

The following men were transferred to Non-Resident Fellowship: Doctors John S. Lockwood, James B. Mason, John Paul North, Norman Freeman, and Bruce L. Fleming.

Those elected to Fellowship in the Academy were: Doctors Lewis C. Manges and Rudolph Jaeger.

This year marked the return of many of our Fellows from the service and on numerous occasions the Chair has welcomed these returning veterans.

The Nominating Committee consisting of Doctors Eldridge L. Eliason, Robert H. Ivy, and Hubley R. Owen presented the following nominations for the year 1947:

President—Dr. John B. Flick.
First Vice-President—Dr. Thomas A. Shallow.
Second Vice-President—Dr. Calvin M. Smyth, Jr.
Secretary—Dr. L. Kraeer Ferguson.
Treasurer—Dr. S. Dana Weeder.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Robert H. Ivy and Dr. Charles Mitchell.
Business Committee—Dr. Frederick A. Bothe, Chairman, with Dr. J. Montgomery Deaver and Dr. W. Emory Burnett.

Respectfully submitted,

L. Kraeer Ferguson, M.D.
Secretary

THE YEAR 1947

Meeting of January 6, 1947, Thompson Hall, College of Physicians. The First Vice-President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

Case Report.	
Edward F. McLaughlin, M.D. and V. R. Manning, Jr., M.D. (By invitation)	Persistent Vitelline Artery and Gangrenous Meckel's Diverticulum.
Papers.	
Robert H. Ivy, M.D., and P. C. Iverson, M.D. (By invitation)	Surgical Treatment of Traumatic Tattoos.
W. Emory Burnett, M.D.	Modern and Effective Treatment of Empyema.

Meeting of February 3, 1947, Thompson Hall, College of Physicians. The President, Dr. John B. Flick, in the Chair.

SCIENTIFIC PROGRAM

THOMAS A. SHALLOW, M.D., and F. B. WAGNER, JR., M.D. (By invitation)	Etiology of Primary Varicose Veins.
HERBERT R. HAWTHORNE, M.D.	Transthoracic Esophagogastrostomy. (With Motion Picture Demonstration.)
W. EMORY BURNETT, M.D., and H. T. CASWELL, M.D. (By invitation)	A Method of Treatment for Late Strangulated Hernia.

Conjoint Meeting of the Philadelphia Academy of Surgery and the New York Surgical Society on March 12, 1947, New York Academy of Medicine. The President of the New York Surgical Society, DR. JOHN A. MCCREERY, and the President of the Philadelphia Academy of Surgery, DR. JOHN B. FLICK, jointly presided.

SCIENTIFIC PROGRAM

FRANK J. MCGOWAN, M.D. *Discussed by:* FREDERICK A. BOTHE, M.D.	Leiomyoma of the Stomach.
THOMAS I. HOEN, M.D. *Discussed by:* ROBERT A. GROFF, M.D.	A Successful Method of Closing Large Gaps in Peripheral Nerves.
SAMUEL WILSON MOORE, M.D. *Discussed by:* JOHN R. MOORE, M.D. JESSE T. NICHOLSON, M.D. THEODORE E. ORR, M.D.	Excision of the Os Calcis Following Severe Comminuted Fracture.
RUSSEL H. PATTERSON, M.D. *Discussed by:* GEORGE P. ROSEMOND, M.D.	Clinical Application of Venograms.
WILLIAM L. WATSON, M.D. *Discussed by:* JULIAN JOHNSON, M.D. JOHN H. GIBBON, JR., M.D.	Carcinoma of the Trachea.
WILLIAM BARCLAY PARSONS, M.D. *Discussed by:* ADOLPH A. WALKLING, M.D.	Radical Resection of the Head of the Pancreas for Calcification.

RALPH COLP, M.D. *Discussed by:* JOHN H. GIBBON, JR., M.D. PAUL SAUER, M.D., of New York	Complications Following Supra-Diaphragmatic Vagotomy in Duodenal Ulcer without Obstruction.
R. FRANKLIN CARTER, M.D. *Discussed by:* CALVIN M. SMYTH, M.D.	Reconstruction of the Common Duct.

Meeting of April 7, 1947, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

L. KRAEER FERGUSON, M.D.	A Case of Mesenteric Thrombosis with Resection and Anastomosis.
PAUL C. COLONNA, M.D.	The Problem of the Ununited Hip Fracture with Neck Absorption.
FRANCIS C. GRANT, M.D. *Read by:* DR. SHENKIN	Treatment of Extra-dural Hemorrhage.

Meeting of May 5, 1947, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

S. DANA WEEDER, M.D., and JOSEPH W. STAYMAN, JR., M.D. (By invitation)	Neurogenic Tumor of the Stomach.
ALAN P. PARKER, M.D., and FRANCIS X. BAUER, M.D. (By invitation)	The Spontaneous Rupture of the Transverse Colon.

Paper.

DAMON B. PFEIFFER, M.D.	The Principles of Surgery for Carcinoma of the Rectum.

Meeting of October 6, 1947, Thompson Hall, College of Physicians. The President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

JOHN H. GIBBON, JR., M.D., and RICHARD J. CHODOFF, M.D. (By invitation)	Multiple Pancreatic Pseudo Cysts. Report of a Case Treated by Cystogastrostomy.

J. Montgomery Deaver, M.D., and David Dunn, M.D. (By invitation)	Two Cases of Carcinoma of Head of Pancreas Treated Surgically.
Motion Picture Presentation. W. M. Firor, M.D. (By invitation)	Studies of Malignant Cells by Phase Difference Microscopy.

Meeting of November 3, 1947, Thompson Hall, College of Physicians. The President, Dr. John B. Flick, in the Chair.

SCIENTIFIC PROGRAM

Memoir. Damon B. Pfeiffer, M.D.	Memoir of the Late George P. Muller, M.D.
Case Reports. Ralph Goldsmith, M.D., and J. Zaslow, M.D. (By invitation)	Intestinal Lipodystrophy.
I. S. Ravdin, M.D., and Harold A. Zintel, M.D. (By invitation)	A Case of Portal Hypertension with Severe Hematemesis Treated by Porto Caval Anastomosis.
Paper. W. Emory Burnett, M.D.	The Technique and Improved Results in Lobectomy for Bronchiectasis.

Meeting of December 1, 1947, Thompson Hall, College of Physicians. The President, Dr. John B. Flick, in the Chair.

SCIENTIFIC PROGRAM

Case Report. Julian Johnson, M.D.	Surgical Treatment of Infantile Type of Coarctation of the Aorta.
Frederick A. Bothe, M.D.	Two Cases of Postoperative Infectious Epiphysitis.
Annual Oration. L. Kraeer Ferguson, M.D.	Considerations Concerning Carcinoma of the Large Gut.

REPORT OF THE SECRETARY FOR THE YEAR 1947

The Academy held six stated meetings at Thompson Hall, College of Physicians, during the year and met in March of 1947 with the New York Surgical Society. The meetings were extremely well attended, averaging about 150 at each meeting.

During the year, the Academy voted to revive the custom of giving new Fellows certificates of membership. A certificate has been drawn up and it was decided that the signatures on the certificate should be those of the Officers in office at the time that the member was admitted to the Academy. The preparation of these certificates is now in progress.

The Doctors John B. Flick, Francis C. Grant, Benjamin Lipshutz, and David M. Davis were elevated to Senior Membership in the Academy during the year.

Doctors James F. Schell and Henry P. Brown were transferred from the Active to Non-resident Membership.

Doctors Louis Kaplan, William Parker, and Edwin Shearburn were elected to Fellowship in the Academy during the year. The Academy now has fifty-six Active Fellows, fourteen Non-resident Fellows, and forty-five Senior Fellows.

During the year 1948, eight Fellows now on the Active List will become eligible for Senior Fellowship.

The Nominating Committee presented the following nominations for the year 1948:

President—Dr. Thomas A. Shallow.
First Vice-President—Dr. Calvin M. Smyth, Jr.
Second Vice-President—Dr. L. Kraeer Ferguson.
Secretary—Dr. J. Montgomery Deaver.
Treasurer—Dr. S. Dana Weeder.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Francis Grant and Dr. John B. Flick.
Business Committee—Dr. Frederick A. Bothe, Chairman, with Dr. W. Emory Burnett and the Recorder.

The Annual Oration was pronounced by Doctor L. Kraeer Ferguson on the subject of "Considerations Concerning Carcinoma of the Large Gut."

Respectfully submitted,

L. Kraeer Ferguson, M.D.
Secretary

THE YEAR 1948

Meeting of January 5, 1948, Thompson Hall, College of Physicians. The First Vice-President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

Memoir.

THOMAS A. SHALLOW, M.D. — Memoir of the Late Warren B. Davis, M.D.

Case Reports.

CHARLES A. STEINER, M.D. — Angiosarcoma of the Colon.

ROBERT H. IVY, M.D.
HERBERT R. HAWTHORNE, M.D., and
JOSEPH A. RITTER, M.D.
(By invitation) — Construction of Skin-Tube Esophagus, Following Surgical Treatment of Tracheo-esophageal Fistula.

Paper.

HANS MAY, M.D. — Repair of Contractures of the Hand.

Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery on February 11, 1948, Mitchell Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

LAWRENCE CURTIS, M.D. — Cystic Teratoma of the Head.
Discussed by:
JEFFERSON BROWDER, M.D.

JOHN ROYAL MOORE, M.D. — An Additional Aid in the Valuation of Joint Injuries.
Discussed by:
ROBERT KENNEDY, M.D.

RUDOLPH JAEGER, M.D. — Aneurysms of the Posterior Communicating Artery.
Discussed by:
BRONSON RAY, M.D.

CALVIN M. SMYTH, JR., M.D. — Congenital Absence of the Common Duct.
Discussed by:
EDWARD J. DONOVAN, M.D.

JONATHAN E. RHOADS, M.D. — Simultaneous Abdominal and Perineal Approach for Carcinoma of the Rectum and Rectosigmoid.
Discussed by:
JOHN MULHOLLAND, M.D.
WILLIAM DEW. ANDRUS, M.D.

JOHN H. GIBBON, JR., M.D. — Surgical Treatment of Bronchogenic Carcinoma.
Discussed by:
FRANK BERRY, M.D.

Meeting of March 1, 1948, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

THOMAS A. SHALLOW, M.D., and
KENNETH E. FRY, M.D. — Case Report of a Father and Daughter Who Had Parathyroid Tumors.

Papers.

GEORGE P. ROSEMOND, M.D., and
F. N. COOKE, M.D.
(By invitation) — Comparison of Open and Closed Anastomosis in Colon Resections.

CHARLES A. HATFIELD, M.D.
(By invitation) — Surgical Treatment of Chronic Ulcers of Lower Leg by Excision and Skin Graft.

Meeting of April 5, 1948, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

EDWIN W. SHEARBURN, M.D. — Hemangioma of the Liver Treated by Partial Hepatectomy.

CALVIN M. SMYTH, M.D., and
T. A. RANIERI, M.D.
(By invitation) — Carcinoma of the Gallbladder with Fragmentation Simulating Common Duct Stone.

Paper.

ANTHONY F. DEPALMA, M.D.
(By invitation)
GEORGE BENNETT, M.D.,
(By invitation) and
GERALD CALLERY, M.D.
(By invitation) — Variational Anatomy of the Shoulder Joint and Its Clinical Significance.

Meeting of May 3, 1948, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

EDWARD F. MCLAUGHLIN, M.D.
VALENTINE R. MANNING, JR., M.D., and
(By invitation)
EMMETT F. CICCONE, M.D.
(By invitation) — Carcinoid Tumor of the Small Intestine.

L. KRAEER FERGUSON, M.D., and
ROBERT F. WELTY, M.D.
(By invitation) — Experience with the Use of the Crosby Button in Cases of Cirrhosis with Ascites.

Paper.

DAVID M. DAVIS, M.D.	Carcinoma of the Prostate.

Meeting of October 4, 1948, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

JOHN W. BRANSFIELD, M.D., and SIDNEY BECK, M.D. (By invitation)	Interscapulothoracic Amputation Plus Radical Mastectomy for Advanced Carcinoma of the Breast.
BENJAMIN LIPSHUTZ, M.D., and RICHARD J. CHODOFF, M.D. (By invitation)	Massive Resection of Small Intestine, Leaving Only Six Inches.

Paper.

JULIAN JOHNSON, M.D., and CHARLES K. KIRBY, M.D. (By invitation)	Aneurysm of the Common Carotid Artery.

Meeting of November 1, 1948, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

GEORGE P. ROSEMOND, M.D.	Unusual Case of Gastrointestinal Hemorrhage.
HANS MAY, M.D.	Reconstruction of an Artificial Vagina.

Paper.

HERBERT R. HAWTHORNE, M.D. and A. FROBESE, M.D. (By invitation)	Chronic Stenosing Regional Enteritis: Pathology and Surgical Treatment.

Meeting of December 6, 1948, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

J. WALTER LEVERING, M.D., and D. B. MILLER, M.D. (By invitation)	Meckel's Diverticulum Causing Recurrent Intussusception.

JULIAN JOHNSON, M.D., and CHARLES K. KIRBY, M.D. (By invitation)	The Use of Vein Grafts to Replace Large Arteries.

Annual Oration.

JONATHAN E. RHOADS, M.D.	Studies on Intraperitoneal Adhesions.

REPORT OF THE SECRETARY FOR THE YEAR 1948

During the year of 1948, the Academy held seven stated meetings and one Conjoint Meeting with the New York Surgical Society.

The Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery was held in Philadelphia at the College of Physicians on February 11, 1948. Following the meeting, dinner was held for the combined Societies at the Racquet Club.

During the year there were thirteen case reports and eight papers presented.

One memoir was read. (The late Warren B. Davis, M.D.)

Two candidates were elected to Active Fellowship. (Doctors Preston C. Iverson and Lloyd W. Stevens.)

Two Fellows died during the year. (Doctors J. Bernhard Mencke and T. Turner Thomas.)

The Annual Oration was pronounced by Jonathan E. Rhoads, M.D., on the subject of "Studies on Intraperitoneal Adhesions," on December 6, 1948.

Respectfully submitted,

J. MONTGOMERY DEAVER, M.D.
Secretary

THE YEAR 1949

Meeting of January 3, 1949, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

FREDERICK A. BOTHE, M.D.	Congenital Absence of the Gallbladder. Report of Two Cases.
ROBERT H. IVY, M.D., and JOHN D. REESE, M.D. (By invitation)	Massive Third Degree Burns of Legs: A New Technique in the Management of Split Skin Grafts.

Paper.
JOHN H. GIBBON, JR., M.D., and FRANK F. ALLBRITTEN, JR., M.D. (By invitation) — Treatment of Carcinoma of the Thoracic Esophagus Above the Aortic Arch by Radical Resection and Cervical Esophagogastric Anastomosis.

Meeting of February 7, 1949, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Report.
JAMES A. LEHMAN, M.D. — Syphilis of the Stomach.

Paper.
S. DANA WEEDER, M.D., and JAMES S. C. HARRIS, M.D. (By invitation) — The Treatment of Perforated Peptic Ulcer Before and After the Use of Antibiotics and Sulfonamides.

Paper.
JOHN H. GIBBON, JR., M.D. — Experimental Reconstruction of Cardiac Valves by Venous and Pericardial Grafts.

Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery on March 9, 1949, New York Academy of Medicine. The President of the New York Surgical Society, DR. EDWARD J. DONOVAN, and the President of the Philadelphia Academy of Surgery, DR. THOMAS A. SHALLOW, jointly presided.

SCIENTIFIC PROGRAM

PRESTON A. WADE, M.D. *Discussed by:* I. S. RAVDIN, M.D. — Carcinoma of Parathyroid Gland: Hyperparathyroidism. End Result Seven Years Postoperative.

ROBERT H. WYLIE, M.D., and EDGAR L. FRAZELL, M.D. *Discussed by:* JOHN H. GIBBON, JR., M.D. — Carcinoma of Upper Oesophagus: Resection: Oesophagogastrostomy.

BEVERLY C. SMITH, M.D. *Discussed by:* FREDERICK A. BOTHE, M.D. — The Use of Radioactive Sodium in the Evaluation of Operability in Essential Hypertension.

HERBERT CONWAY, M.D. *Discussed by:* ROBERT H. IVY, M.D. — Reconstructive Surgery Following Radical Operation for Malignancy of the Head and Neck.

HENRY F. GRAHAM, M.D. *Discussed by:* JONATHAN E. RHOADS, M.D. — Chronic Mesenteric Ileus: Duodenojejunostomy.

JOHN LEO MADDEN, M.D. *Discussed by:* GEORGE P. ROSEMOND, M.D. — Resection of the Left Auricular Appendix: A Prophylaxis in the Treatment of Recurrent Arterial Emboli.

HOWARD A. PATTERSON, M.D. *Discussed by:* L. KRAEER FERGUSON, M.D. — The Incidence of Carcinoma in Association with Chronic Ulcerative Colitis: Seven Cases.

Meeting of April 4, 1949, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Report.
L. KRAEER FERGUSON, M.D., and JAMES HOLT, M.D. (By invitation) — Complete Severance of the Brachial Artery with Primary Suture.

Papers.
J. MONTGOMERY DEAVER, M.D., and JOSEPH F. PATTERSON, JR., M.D. (By invitation) — Carcinoma of the Cecum. Review of Twenty-nine Cases.

HARRY E. KNOX, M.D., and ROBERT GLOVER, M.D. — Aortography. Its Importance in Surgery of the Aortic Arch.

Meeting of May 2, 1949, Thompson Hall, College of Physicians. The President, DR. THOMAS A. SHALLOW, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.
JOHN B. FLICK, M.D., and WOODROW W. LINDENMUTH, M.D. (By invitation) — Fecal Fistula Following Escape of Mercury from a Miller-Abbott Tube.

W. EMORY BURNETT, M.D., and H. TAYLOR CASWELL, M.D. (By invitation) — A Case of Mediastinal Reduplication of Gastrointestinal Tract Communicating with Small Intestine. (Colored Moving Pictures.)

Paper.
FRANCIS C. GRANT, M.D. — Significant Factors Affecting End Results Following Suture of Peripheral Nerves.

Meeting of October 3, 1949, Thompson Hall, College of Physicians. The President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Irvin E. Deibert, M.D., and Wesley M. Jack, M.D. (By invitation) — Plasmacytoma of the Thyroid.

Charles H. Harney, M.D., and George E. Murphy, M.D. (By invitation) — Spontaneous Rupture of the Kidney Parenchyma with Massive Retroperitoneal Hemorrhage.

Paper.

Donald C. Geist, M.D., and Paul Grotzinger, M.D. (By invitation) — Multiple Primary Malignancies.

Meeting of November 7, 1949, Thompson Hall, College of Physicians. The President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Jonathan E. Rhoads, M.D., and Albert Behrend, M.D. (By invitation) — Cholecysto-choledochal Fistulae.

Damon B. Pfeiffer, M.D., and David B. Miller, M.D. (By invitation) — Islet Cell Adenoma of the Pancreas.

Paper.

I. S. Ravdin, M.D., and Charles K. Kirby, M.D. (By invitation) — A New Method for Detection of Gall Stones in the Bile Duct.

Meeting of December 5, 1949, Thompson Hall, College of Physicians. The President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

Hans May, M.D. — Avulsion of Scrotum and Skin of Penis: Plastic Repair.

Case Report.

Herbert R. Hawthorne, M.D., and James O'Neill, M.D. (By invitation) — Duplication of the Cecum as a Cause of Intussusception in an Infant.

Paper.

I. S. Ravdin, M.D., and Charles Bailey, M.D., and (By invitation) Robert Glover, M.D. (By invitation) — Mitral Stenosis. Its Surgical Treatment by Commissurotomy.

REPORT OF THE SECRETARY FOR 1949

During the year of 1949, the Academy held seven stated meetings and one Conjoint Meeting with the New York Surgical Society.

The Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery was held in New York at the New York Academy of Medicine on March 9, 1949. Following the meeting, dinner was held for the combined Societies at the Manhattan Club.

During the year, there were twelve case reports and nine papers presented.

Five candidates were elected to Active Fellowship. (Doctors Frank F. Allbritten, Jr., Richard J. Chodoff, Anthony F. DePalma, John D. Reese and Harold A. Zintel.)

Two Fellows died during the year. (Doctors Basil E. Beltran and George M. Dorrance.)

The Annual Oration was pronounced by Francis C. Grant, M.D., on the subject of "Treatment of Intractable Pain from Inoperable Cancer" on January 9, 1950.

The Nominating Committee presented the following nominations for the year 1950:

President—Dr. Calvin M. Smyth.
First Vice-President—Dr. I. S. Ravdin.
Second Vice-President—Dr. L. Kraeer Ferguson.
Secretary—Dr. J. Montgomery Deaver.
Treasurer—Dr. S. Dana Weeder.
Recorder—Dr. Jonathan E. Rhoads.
Council—Dr. Francis Grant and Dr. Thomas A. Shallow.
Business Committee—Dr. John H. Gibbon, Jr., Chairman, and Dr. W. Emory Burnett and the Recorder.

Respectfully submitted,

J. Montgomery Deaver, M.D.
Secretary

THE YEAR 1950

Meeting of January 9, 1950, Thompson Hall, College of Physicians. The President, Dr. Thomas A. Shallow, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

Ralph Goldsmith, M.D., and Jerry Zaslow, M.D. (By invitation) — Two Cases of Obstructive Jaundice of Uncommon Origin.

Paper.

Julian Johnson, M.D., and Charles K. Kirby, M.D. (By invitation) — Clinical and Experimental Considerations of Cardiac Resuscitation.

Annual Oration.

Francis C. Grant, M.D. — Treatment of Intractable Pain from Inoperable Cancer.

Meeting of February 6, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

John H. Gibbon, Jr., M.D., and Joseph W. Stayman, Jr., M.D., (By invitation) and Theodore P. Eberhard, M.D. (By invitation) — Papillary Adenocarcinoma of the Thyroid with Metastasis: Treated by Total Thyroidectomy and Radioactive Iodine.

Papers.

Hans May, M.D. — The Surgical Treatment of Intractable Plantar Warts.

S. Dana Weeder, M.D., and J. S. C. Harris, M.D. (By invitation) — Cholecystectomy with Closure of the Abdomen without Drainage.

Joint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery on March 8, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Ralph Jones, M.D. and Cecelia Riegel, Ph.D. (By invitation of Dr. I. S. Ravdin) — An Evaluation of Certain Tests for Cancer.

J. Montgomery Deaver, M.D. — Report of a Case: Concomitant Pyloric-Esophageal Obstruction Due to Peptic Ulcer—Treated Surgically.

Harold A. Zintel, M.D.
James Dull, M.D. (By invitation)
Miss Helen Ellis (By invitation)
Miss Anna Nichols (By invitation) — Evaluation of Phisoderm G-11, a Liquid Soap with G-11 and a Bland Cake Soap When Used as a Pre-Operative Scrub.

W. Emory Burnett, M.D., and George P. Rosemond, M.D., and Robert M. Bucher, M.D. (By invitation) — Studies on the Reconstruction of the Trachea.

Francis C. Grant, M.D. — Two Cases of Large Spina Bifida, with Surgical Repair.

Julian Johnson, M.D., and Charles K. Kirby, M.D. (By invitation) — A New Incision for Cardiac Decortication: Report of a Case.

Frank F. Allbritten, Jr., M.D., and John Y. Templeton, III, M.D. (By invitation) — Treatment of Giant Cysts of Lung. (Moving Picture Demonstration.)

Meeting of April 3, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

Colonel Harold A. Conrad and Frederick R. Robbins, M.D. — Congenital Atresia of the Intestine in the Newborn. Report of a Successful Surgical Management of a Case.

Papers.

George Willauer, M.D., and John DeTuerk, M.D. (By invitation) — Continuous Spinal Anesthesia for Thoracoplasty.

L. Kraeer Ferguson, M.D., and Mark W. Wolcott, M.D. (By invitation) — The Treatment of Idiopathic Ulcerative Colitis.

Joint Meeting of the U. S. Naval Hospital Staff and the Philadelphia Academy of Surgery on April 10, 1950, U. S. Naval Hospital, Philadelphia.

SCIENTIFIC PROGRAM

Case Report.

Lieutenant (j.g.) G. T. Van Petten (MC), U.S.N.	Reduplication of the Appendix.
Captain Luther G. Bell (MC), U.S.N.	Acute Pneumocholecystitis. Report of Two Cases.
Commander Robert B. Brown (MC), U.S.N.	Congenital Anomalies of Intestinal Rotation and Mesenteric Fixation as a Cause of Intestinal Obstruction in the Adult.

Meeting of May 1, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Memoir.

L. Kraeer Ferguson, M.D.	Memoir of the Late Eldridge L. Eliason, M.D.

Papers.

Lt. Col. Edward G. Sion (By invitation)	Massive Echinococcus Cyst of the Liver Handled by Surgical Resection.
James D. Hardy, M.D. (By invitation) (Introduced by Dr. Jonathan E. Rhoads)	Factors Influencing the Postoperative Retention of Salt and Water.
Edward F. McLaughlin, M.D.	Femoral Hernia. Present Aspects.

Meeting of October 2, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Papers.

Fred G. Medinger, M.D. (By invitation) Introduced by Dr. Calvin M. Smyth	Evaluation of End Results of Carcinoma of the Breast at Abington Hospital.
Charles Rogers, M.D. (By invitation) Introduced by Dr. I. S. Ravdin	Method of Caring for Extensive Strictures of the Common Duct.

Case Report.

S. Dana Weeder, M.D., and John Y. Templeton, III, M.D. (By invitation)	Lipoma of the Sigmoid Colon with Intussusception.

Meeting of November 6, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

William H. Whitely, M.D. (By invitation) Introduced by Dr. Rudolph Jaeger	Avulsion of the Brachial Plexus: Report of Two Cases.
Harold A. Zintel, M.D.	Partial Hepatectomy for Pylephlebitis.

Paper.

Frank F. Allbritten, Jr., M.D., and Col. Robert W. DuPriest (MC) (By invitation)	The Association of the Aberrant Lung Lobe, Bronchial Cysts, and Anomalous Bronchial Arteries.

Meeting of December 4, 1950, Thompson Hall, College of Physicians. The President, Dr. Calvin M. Smyth, in the Chair.

SCIENTIFIC PROGRAM

Annual Oration.

W. Emory Burnett, M.D.	Experience with Sixty-five Cases of Surgical Lesions of the Mediastinum.

Case Reports.

James B. Carty, M.D. (By invitation) Introduced by Dr. Orville C. King	Desmoid Tumor of the Scapular Area.
Fletcher D. Sain, M.D. (By invitation) Introduced by Dr. Calvin M. Smyth	Two Cases of Congenital Absence of Gallbladder.

REPORT OF THE SECRETARY FOR THE YEAR 1950

During the year 1950, the Philadelphia Academy of Surgery held eight regular stated meetings. In addition, on March 8, 1950, sixty-five members of the New York Surgical Society met with fifty-seven Fellows of the Phila-

delphia Academy of Surgery in Philadelphia. Following the scientific program, dinner for the combined Societies was held at the Racquet Club. A joint meeting of the U. S. Naval Hospital Staff and the Philadelphia Academy of Surgery was held at the U. S. Naval Hospital at 8:30 P.M. on Monday, April 10, this meeting also being in addition to the above stated meetings.

Due to a fire on June 16, 1950, in the office of the Secretary, the following records were destroyed: Report of Minutes of meetings for the years 1948, 1949 and for the first five months of 1950, plus the Minutes of Council for the year 1949 and the first five months of 1950. All other records for the Academy are intact. Due to this happening, it was decided by the Academy when it reconvened on October 2, 1950, after its summer recess, to send duplicate copies of minutes of the meetings to the Recorder, Doctor Jonathan E. Rhoads. In addition, a photostatic copy was procured of the Constitution and By-Laws; this copy has been placed in the hands of Doctor Rhoads for safekeeping.

Respectfully submitted,

J. MONTGOMERY DEAVER, M.D.
Secretary

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