TRANS-
ACTIONS
of the
PHILA-
DELPHIA
ACADEMY
OF
SURGERY

1939-1940
1941-1942

VOLUME
XXVII

S.E.N.

TRANSACTIONS

OF THE

PHILADELPHIA

ACADEMY OF SURGERY

VOLUME XXVII

1939-1940-1941-1942

PHILADELPHIA
PRINTED FOR THE ACADEMY
1943

NOTICE

This volume has been prepared according to the plan adopted for previous volumes. The Annual Orations for the years 1939 to 1942 inclusive are presented in full. Memoirs of the Fellows deceased since the last publication which have been presented before the Academy are reproduced. Members in the Armed Forces are designated, but no attempt has been made to denote the rank of each individual.

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ADOLPH A. WALKLING,
Recorder.

PRINTED IN THE UNITED STATES OF AMERICA

CONTENTS

CONSTITUTION AND BY-LAWS

(As Amended to 1942)

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, Non-resident, 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 $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 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, and shall not pay dues.

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 years.

ARTICLE VI

Honorary Fellows, to the number of thirty, 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 years' standing of a reputable Medical School practicing his profession in the City of Philadelphia or within thirty 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 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, regular 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 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 Fellows shall be appointed by the President to serve for five 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 at 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 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. The notice of the meeting succeeding that at which the nominations were made shall contain a list of those nominated for Fellowship, and the date upon which the Council will act upon the same.

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 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 months of his election.

SECTION XVI

INITIATION FEE

Every Fellow shall, on admission, pay an initiation fee of ten dollars.

SECTION XVII

ANNUAL DUES

There shall be an annual assessment of seven dollars, to be paid within three 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.

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 year, being notified of the fact by the Treasurer, in writing, and not paying his dues within two 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 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 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 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

Founded April 21, 1879 *Incorporated December 27, 1879*

* Samuel D. Gross, M.D., LL.D., D.C.L., Oxon.
* D. Hayes Agnew, M.D., LL.D.
* Addinell Hewson, M.D.
* Richard J. Levis, M.D.
* Thomas G. Morton, M.D.
* John H. Packard, M.D.
* John H. Brinton, M.D.
* William H. Pancoast, M.D.
* J. Ewing Mears, M.D.
* Samuel W. Gross, M.D., LL.D.

* Deceased.

LIST OF OFFICERS, 1943

President

DR. ROBERT H. IVY

Vice-Presidents

DR. HUBLEY R. OWEN
DR. JOHN B. FLICK

Secretary

DR. L. KRAEER FERGUSON

Treasurer

DR. HARRY E. KNOX

Recorder

DR. ADOLPH A. WALKLING

Business Committee

DR. CALVIN M. SMYTH, JR., (Chr.)
DR. J. MONTGOMERY DEAVER

With the Recorder

Council

DR. ELDRIDGE L. ELIASON
DR. THOMAS A. SHALLOW

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

Trustees of the Samuel D. Gross Prize

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

PHILADELPHIA ACADEMY OF SURGERY

FOUNDED APRIL 21, 1879
INCORPORATED DEC. 27, 1879

OFFICERS

1879

Temporary Chairman.....ADDINELL HEWSON
Temporary Secretary.....J. EWING MEARS
Temporary Treasurer.....WILLIAM HUNT
Temporary Recorder.....JOHN B. ROBERTS

PRESIDENT

ELECTED
1880 SAMUEL D. GROSS
1884 D. HAYES AGNEW
1891 WILLIAM HUNT
1895 THOMAS G. MORTON
1898 DEFOREST WILLARD
1902 RICHARD H. HARTE
1904 HENRY R. WHARTON
1906 JOHN B. ROBERTS
1908 WILLIAM J. TAYLOR
1910 ROBERT G. LECONTE
1912 GWILYM G. DAVIS
1914 JOHN H. GIBBON
1916 CHARLES H. FRAZIER
1918 EDWARD MARTIN
1920 GEORGE G. ROSS
1922 JOHN H. JOPSON
1924 EDWARD B. HODGE
1926 CHARLES F. MITCHELL
1928 ASTLEY P. C. ASHHURST
1930 GEORGE P. MULLER
1932 JOHN SPEESE
1934 WALTER E. LEE
1936 DAMON B. PFEIFFER
1938 J. STEWART RODMAN
1940 ELDRIDGE L. ELIASON

VICE-PRESIDENTS

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

SECRETARY

ELECTED		ELECTED	
1880	J. Ewing Mears	1915	George P. Muller
1885	J. Henry C. Simes	1920	J. Stewart Rodman
1893	Thomas R. Neilson	1922	Hubley R. Owen
1896	William J. Taylor	1930	DeForest P. Willard
1905	John H. Gibbon	1935	Henry P. Brown, Jr.
1909	Charles F. Mitchell	1940	John B. Flick

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

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

COUNCIL

ELECTED		ELECTED	
1880	John Ashhurst, Jr.	1924	John H. Jopson
1880	John H. Brinton	1924	John Speese
1894	William B. Hopkins	1925	Edward B. Hodge
1895	Henry R. Wharton	1926	Damon B. Pfeiffer
1898	Thomas R. Neilson	1927	Charles F. Mitchell
1900	W. Joseph Hearn	1930	Astley P. C. Ashhurst
1902	Robert G. LeConte	1930	Hubley R. Owen
1906	Thomas R. Neilson	1932	George P. Muller
1910	J. Chalmers DaCosta	1935	DeForest P. Willard
1920	Charles F. Mitchell	1936	Walter Estell Lee
1922	George G. Ross	1936	Robert H. Ivy
1922	James H. Baldwin	1940	J. Stewart Rodman
1923	William J. Taylor		

With President, Vice-President, Secretary and Treasurer

BUSINESS COMMITTEE

ELECTED		ELECTED	
1895	William J. Taylor	1908	Francis T. Stewart
1895	DeForest Willard	1914	John Speese
1896	Richard H. Harte	1916	W. E. Lee
1897	Robert G. LeConte	1916	Morris Booth Miller
1900	G. G. Davis	1917	Damon B. Pfeiffer
1902	John H. Jopson	1917	A. P. C. Ashhurst
1905	George G. Ross	1919	A. Bruce Gill

ELECTED		ELECTED	
1919	J. Stewart Rodman	1930	John B. Flick
1920	Arthur Billings	1931	Henry P. Brown, Jr.
1922	Damon B. Pfeiffer	1932	Edward T. Crossan
1924	DeForest P. Willard	1935	B. Franklin Buzby
1928	Walter E. Lee	1936	John B. Flick
1930	Edward T. Crossan	1938	Lewis K. Ferguson
		1940	J. Montgomery Deaver

With the Recorder

TRUSTEES AND COMMITTEE FOR MAKING AWARD OF THE SAMUEL D. GROSS PRIZE 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.

FELLOWS OF THE PHILADELPHIA ACADEMY OF SURGERY

1932 Alston, Robert S., A.B., M.S., F.A.C.S., 121 W. Walnut Lane, Germantown. Surgeon to the Germantown and Stetson Hospitals.

1928 Bates, William, B.S., M.D., F.A.C.S., 2029 Pine Street. Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Surgeon, Presbyterian, Babies', and Graduate Hospitals.

1922 *Beltran, Basil R., A.M., M.D., F.A.C.S., 2109 Locust Street. Associate Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Surgeon, Misericordia and Fitzgerald-Mercy Hospitals; Associate Surgeon, Graduate Hospital of the University of Pennsylvania; Consulting Surgeon, Nazareth Hospital.

1915 *Billings, Arthur E., M.D., 2020 Spruce Street. Surgeon to the Bryn Mawr Hospital; Clinical Professor of Surgery, Jefferson Medical College.

1934 Birdsall, Joseph C., A.M., M.D., 1900 Spruce Street. Professor of Urology, University of Pennsylvania Graduate School of Medicine; Urologic Surgeon and Director of the Genito-Urinary Department, Presbyterian Hospital; Urologic Surgeon, Graduate Hospital and Babies' Hospital.

1929 †Bothe, Albert E., M.S., M.D., D.Sc., 255 S. 17th Street. Assistant Professor of Urology, University of Pennsylvania Graduate School of Medicine; Urologist, Misericordia, Fitzgerald-Mercy, and Children's Hospitals.

1928 Bothe, Frederick A., M.S., M.D., F.A.C.S., 255 S. 17th Street. Associate in Surgery, University of Pennsylvania Graduate School of Medicine; Surgeon, Presbyterian Hospital; Associate in Surgery, Children's Hospital; Consulting Surgeon, Home for Incurables.

1932 Bower, John O., M.D., F.A.C.S., 2008 Walnut Street. Surgeon, Philadelphia General Hospital; Consulting Surgeon, St. Luke's and Children's Medical Center; Director of Research, Foundation for Clinical and Surgical Research, Temple University Medical School.

1921 *Boykin, Irvine M., M.D., 136 S. 16th Street. Associate in Surgery, University of Pennsylvania School of Medicine; Surgeon, Episcopal Hospital; Visiting Surgeon, Abington Memorial Hospital.

1921 *Brandsfield, John William, M.D., F.A.C.S., 2101 Spruce Street. Visiting Surgeon, St. Vincent's, American Oncologic and Doctors' Hospitals.

* Denotes Senior Fellow.
† Denotes Fellows in Armed Forces.

1919 †*Brown, Henry P., Jr., B.S., M.D., F.A.C.S., 1930 Chestnut Street. Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Associate in Surgery, University of Pennsylvania School of Medicine; Surgeon, Pennsylvania Hospital and Chief of Out-Patient Clinic; Surgeon, Children's and Presbyterian Hospitals.

1938 Burnett, W. Emory, A.B., M.D., F.A.C.S., 3401 N. Broad Street. Professor of Clinical Surgery, Temple University School of Medicine; Associate Surgeon, Temple University Hospital; Surgeon, Philadelphia General Hospital.

1923 Buzby, B. Franklin, A.B., M.D., F.A.C.S., 414 Cooper Street, Camden, N. J. Orthopedist, Cooper Hospital, Camden, N. J., Burlington County Hospital and Germantown Hospital; Consulting Orthopedist, Underwood Hospital.

1919 *Crossan, Edward T., M.D., 5324 Wayne Avenue. Instructor in Surgery, University of Pennsylvania School of Medicine; Chief Surgeon, Episcopal Hospital.

1932 Curtis, Lawrence, A.B., D.D.S., M.D., F.A.C.S., 255 S. 17th Street. Associate Professor of Maxillofacial Surgery, University of Pennsylvania Graduate School of Medicine and School of Dentistry; Associate in Maxillofacial Surgery, Graduate Hospital of the University of Pennsylvania; Associate in Oral and Plastic Surgery, Presbyterian Hospital; Oral and Plastic Surgeon to Bryn Mawr Hospital; Maxillofacial Surgeon to Delaware County Hospital.

1939 Davis, David M., B.S., M.D., 255 S. 17th Street. Professor of Urology, Jefferson Medical College, Attending Urologist, Jefferson Hospital.

1922 *Davis, Warren B., M.D., Sc.D., F.A.C.S., 135 S. 18th Street. Clinical Professor of Oral Surgery, Jefferson Medical College; Maxillofacial Surgeon, Jefferson Medical College Hospital; Consulting Maxillofacial Surgeon, Frankford Hospital and Kensington Hospital for Women.

1934 †Deaver, J. Montgomery, B.S., M.D., F.A.C.S., 1830 Delancey Street. Instructor in Surgery, University of Pennsylvania School of Medicine and Graduate School of Medicine; Assistant Surgeon, Lankenau and Mary J. Drexel Children's Hospitals; Assistant Visiting Surgeon, Abington Memorial Hospital, Abington.

1930 Deibert, Irvin E., M.D., F.A.C.S., 538 Cooper Street, Camden, N. J. Chief Surgical Service B, Cooper Hospital; Attending Surgeon, Camden County Hospital for Tuberculosis, Lakeland, N. J.

1916 *Dorrance, George M., M.D., F.A.C.S., 2101 Spruce Street. Professor of Maxillofacial Surgery, Thomas Evans Institute of the University of Pennsylvania; Consulting Oral Surgeon, University Hospital; Surgeon-in-Chief, American Oncologic Hospital and Doctors Hospital; Consulting Oral Surgeon, Norristown State Hospital and Montgomery Hospital.

1928 †Downs, T. McKean, M.D., 255 S. 17th Street. Assistant Surgeon, Pennsylvania, Germantown and Bryn Mawr Hospitals.

1921 *Eliason, Eldridge Lyon, A.B., M.D., Sc.D., F.A.C.S., 326 S. 19th Street. John Rhea Barton Professor of Surgery, University of Pennsylvania School of Medicine, Surgeon, University of Pennsylvania, Philadelphia General and Presbyterian Hospitals.

1909 *Elmer, Walter G., B.S., M.D., F.A.C.S., 1801 Pine Street. Emeritus Professor of Orthopedics, University of Pennsylvania Graduate School of Medicine; Emeritus Professor of Orthopedic Surgery, Women's Medical College of Pennsylvania; Active Consulting Orthopedic Surgeon, Philadelphia General Hospital; Consulting Orthopedic Surgeon, Jewish Hospital and Elwyn Training School, Elwyn, Pa.

1934 Engel, Gilson Colby, A.B., M.D., F.A.C.S., 1914 Pine Street. Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Chief of Surgical Service B, Lankenau Hospital; Assistant Surgeon, Children's Hospital of Mary J. Drexel Home.

1941 Erb, William H., A.B., M.D., F.A.C.S., 133 S. 36th Street. Associate in Surgery, University of Pennsylvania School of Medicine; Assistant Surgeon, Philadelphia General and University of Pennsylvania Hospitals; Surgeon, Taylor Hospital, Ridley Park, Pa., and Doctors Hospital.

1931 †Ferguson, Lewis Kraeer, A.B., M.D., F.A.C.S., 133 S. 36th Street. Assistant Professor of Surgery, University of Pennsylvania School of Medicine; Assistant Surgeon, Hospital of the University of Pennsylvania; Surgeon, Philadelphia General Hospital.

1931 Fleming, Bruce L., M.D., F.A.C.S., 1930 Chestnut Street. Surgeon, Memorial Hospital, Roxborough; Clinical Assistant in Surgery, Jefferson Medical College Hospital.

1926 †Flick, John B., M.D., Mermont Apts., Bryn Mawr, Pa. Clinical Professor of Surgery, Jefferson Medical College; Chief of Surgical Service A, Pennsylvania Hospital; Attending Surgeon, Bryn Mawr Hospital.

1938 †Freeman, Norman E., A.B., M.D., F.A.C.S., 3400 Spruce Street. Assistant Professor of Research Surgery, University of Pennsylvania School of Medicine; Associate in Surgery, University of Pennsylvania Graduate School of Medicine; Assistant Surgeon, Hospital of the University of Pennsylvania; Surgical Chief, Vascular Clinic, Pennsylvania Hospital.

1942 †Fry, Kenneth E., B.S., M.D., F.A.C.S., 1611 Spruce Street. Associate in Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital.

1942 GAMON, ROBERT S., A.B., M.D., F.A.C.S., 514 Cooper Street, Camden, N. J. Surgeon, Cooper Hospital; Consulting Surgeon, Zurbrugg Memorial Hospital, Riverside, and Camden County Hospital for Tuberculosis, Grenloch.

1939 †GEIST, DONALD C., A.B., M.D., F.A.C.S., 1930 Chestnut Street. Instructor in Surgery, University of Pennsylvania Graduate School of Medicine; Assistant Surgeon, Misericordia Hospital; Associate Surgeon, Fitzgerald-Mercy Hospital.

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

1933 †GIBBON, JOHN H., JR., M.D., 4035 Pine Street. Surgeon, Pennsylvania Hospital; Assistant Surgeon, Bryn Mawr Hospital.

1914 *GILL, A. BRUCE, A.B., M.D., SC.D., 1930 Chestnut Street. Professor of Orthopedic Surgery, University of Pennsylvania School of Medicine and the Graduate School of Medicine; Surgeon, Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases; Consulting Orthopedic Surgeon, Presbyterian Hospital; Chief of Consultant Staff, Children's Seashore Home, Atlantic City, N. J.

1928 GILMOUR, WILLIAM R., M.A., M.D., F.A.C.S., 6616 Woodland Avenue. Associate Surgeon, Methodist Episcopal Hospital; Surgeon, Northeastern Hospital.

1932 GOLDSMITH, N. RALPH, M.D., F.A.C.S., 136 S. 16th Street. Surgeon, Jewish Hospital.

1925 GRANT, FRANCIS CLARK, A.B., M.D., F.A.C.S., 3400 Spruce Street. Professor of Neurosurgery, University of Pennsylvania School of Medicine and Graduate School of Medicine; Neurosurgeon, University of Pennsylvania Hospital; Consulting Neurosurgeon, Children's Hospital; Visiting Neurosurgeon, Abington Memorial and Philadelphia General Hospitals.

1934 GREENE, LLOYD B., M.D., 136 S. 16th Street. Urologist, Pennsylvania and Burlington County Hospitals; Associate Urologist, Bryn Mawr Hospital.

1939 †GROFF, ROBERT A., M.D., F.A.C.S., 255 S. 17th Street. Neurosurgeon, Pennsylvania, Presbyterian, Germantown and Philadelphia General Hospitals; Associate Neurosurgeon, Mt. Sinai and Abington Memorial Hospitals; Consulting Neurosurgeon, Philadelphia State Hospital, Women's Hospital and Misericordia Hospital.

1941 †HARNEY, CHARLES H., A.B., M.D., Mermont Apartments, Bryn Mawr, Pa. Instructor in Surgery, Jefferson Medical College; Assistant Attending Surgeon, Bryn Mawr Hospital; Surgeon in Charge of Anesthesia, Bryn Mawr Hospital; Assistant Attending Surgeon, Pennsylvania Hospital; Assistant Surgeon O. P. D., Jefferson Hospital.

1913 *HEARN, WILLIAM P., B.S., M.D., F.A.C.S., 900 Sansom Street. Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Hospital.

1922 *HERMAN, LEON, B.S., M.D., 136 S. 16th Street. Urologist, Pennsylvania and Bryn Mawr Hospitals.

1925 HINTON, DRURY, M.D., F.A.C.S., 4501 Cedar Lane, Drexel Hill, Pa. Chief, Surgical Service, Delaware County Hospital; Associate Surgeon, Fitzgerald-Mercy Hospital; Consulting Surgeon, Woman's Hospital.

1905 *HODGE, EDWARD B., A.B., M.D., 2019 Spruce Street. Consulting Surgeon, Presbyterian Hospital and Germantown Hospital; Chief Surgeon, Chester County Hospital.

1934 HOWELL, JOHN C., M.D., F.A.C.S., 326 S. 19th Street. Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Visiting Surgeon, Radiological Department, Philadelphia General Hospital and Babies' Hospital; Associate in Surgery, Graduate and Presbyterian Hospitals.

1915 *IVY, ROBERT HENRY, M.D., D.D.S., F.A.C.S., 1930 Chestnut Street. Professor of Maxillofacial Surgery, University of Pennsylvania School of Medicine and Graduate School of Medicine; Chief of Maxillofacial Surgery, University of Pennsylvania and Graduate Hospitals; Oral Surgeon, Presbyterian Hospital; Consulting Plastic Surgeon, Children's and Chestnut Hill Hospitals; Consultant in Maxillofacial Surgery, Walter Reed General Hospital, Washington, D. C.

1922 *JOHN, RUTHERFORD L., M.D., 256 S. 21st Street. Associate Orthopedic Surgeon, Methodist Episcopal Hospital; Orthopedic Surgeon, Children's Hospital, Fitzgerald-Mercy Hospital and St. Christopher's Hospital.

1942 †JOHNSON, JULIAN, A.B., M.D., D.SC. (MED.), F.A.C.S., 3400 Spruce Street. Associate in Surgery, University of Pennsylvania School of Medicine; Assistant Surgeon, Hospital of the University of Pennsylvania, Philadelphia General, Memorial and Presbyterian Hospitals.

1915 *JONES, JOHN F. X., B.S., A.M., M.D., F.A.C.S., Lincoln Court Apartments, Overbrook, Pa. Surgeon, St. Joseph's, Misericordia and St. Agnes' Hospitals.

1910 *KELLY, JAMES A., A.M., M.D., F.A.C.S., 1815 Spruce Street. Associate Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Visiting Surgeon, St. Mary's Hospital, St. Joseph's Hospital, Misericordia Hospital and Fitzgerald-Mercy Hospital.

1938 †KING, ORVILLE C., A.B., M.D., F.A.C.S., 255 S. 17th Street. Associate in Surgery, University of Pennsylvania School of Medicine

and Graduate School of Medicine; Associate in Surgery, Presbyterian Hospital; Assistant in Surgery, Children's and Pennsylvania Hospitals.

1930 KNOX, HARRY E., M.D., F.A.C.S., 719 66th Avenue, Oak Lane. Associate in Surgery, University of Pennsylvania School of Medicine. Chief Surgeon, Germantown Dispensary and Hospital and St. Christopher's Hospital for Children; Associate Surgeon, Episcopal Hospital.

1914 *LAWS, GEORGE MALCOLM, B.S., M.D., 1907 Spruce Street, Associate in Gynecology, University of Pennsylvania School of Medicine; Gynecologist, Presbyterian Hospital.

1910 *LEE, WALTER ESTELL, M.D., F.A.C.S., 1833 Pine Street. Professor of Surgery, University of Pennsylvania Graduate School of Medicine, Surgeon, Pennsylvania, Germantown, Bryn Mawr, Children's and Burlington County Hospitals and the Graduate Hospital of the University of Pennsylvania.

1938 LEHMAN, JAMES A., M.D., F.A.C.S., 1815 Spruce Street. Associate Professor of Surgery, Woman's Medical College; Surgeon, Hospital of the Woman's Medical College; Attending Surgeon, Roxborough Memorial Hospital and St. Joseph's Hospital; Associate Surgeon, Chestnut Hill and Fitzgerald-Mercy Hospitals.

1932 LEMMON, WILLIAM T., B.S., M.D., 1930 Chestnut Street. Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Hospital and Woman's Medical College Hospital; Attending Surgeon, Philadelphia General Hospital.

1934 LEVERING, J. WALTER, M.D., F.A.C.S., Abington, Pa. Visiting Surgeon to the Abington Memorial Hospital.

1926 LIPSHUTZ, BENJAMIN, A.B., M.D., F.A.C.S., 1007 Spruce Street. Assistant Professor of Neuroanatomy, Jefferson Medical College; Surgeon, Mount Sinai Hospital.

1941 LOCKWOOD, JOHN S., A.B., M.D., D.Sc., 3400 Spruce Street. Associate in Surgery, University of Pennsylvania School of Medicine; Fellow in Surgical Research, University of Pennsylvania; Assistant Surgeon and Director of Tumor Clinic, Hospital of the University of Pennsylvania; Assistant Surgeon, Pennsylvania Hospital.

1933 †MASON, JAMES B., A.B., M.D., M.Sc. (SURG.), F.A.C.S., 1530 Locust Street. Associate in Surgery, University of Pennsylvania Graduate School of Medicine, Assistant Professor of Clinical Surgery, Woman's Medical College; Assistant Surgeon, Presbyterian and Methodist Hospitals.

1940 MAY, HANS, M.D., F.A.C.S., 5501 Greene Street. Instructor in Surgical Pathology, University of Pennsylvania Graduate School of Medicine; Assistant Surgeon, Lankenau, Germantown and Chestnut Hill Hospitals; Consulting Surgeon, Mary Drexel Children's Hospital and Misericordia Hospital.

1929 MCCARTHY, PATRICK A., M.D., F.A.C.S., 1737 Chestnut Street. Instructor in Surgery and Anatomy, Jefferson Medical College; Surgeon, Philadelphia General Hospital; Assistant Surgeon, Out-Patient Department, Jefferson Hospital; Staff, St. Mary's and Clark Hospitals.

1932 MCCLOSKEY, JOHN F., A.B., M.D., F.A.C.S., 8720 Germantown Avenue. Clinical Professor of Surgery, Woman's Medical College; Surgeon, Chestnut Hill Hospital and Skin and Cancer Hospital.

1942 †MCLAUGHLIN, EDWARD F., A.B., M.D., F.A.C.S., 4116 N. Broad St. Assistant Instructor in Surgery, University of Pennsylvania School of Medicine; Chief, Surgical Service, Nazareth Hospital; Assistant Surgeon, Germantown Dispensary and Hospital, Chestnut Hill Hospital and Philadelphia Hospital for Contagious Diseases; Assistant in Surgery, Hospital of the University of Pennsylvania.

1931 †MEADE, RICHARD H., JR., B.S., M.D., F.A.C.S., 2116 Pine Street. Associate in Surgery, University of Pennsylvania School of Medicine; Associate Surgeon, Episcopal Hospital; Surgeon, Philadelphia General Hospital; Consulting Surgeon, Home for Consumptives, Chestnut Hill.

1931 *MECRAY, PAUL M., M.D., F.A.C.S., 405 Cooper Street, Camden, N. J. Chief Surgeon, Cooper Hospital and Zurbrugg Memorial Hospital; Medical Director, Camden County General Hospital; Consulting Surgeon, Salem Memorial Hospital, Salem, N. J.

1917 *MENCKE, J. BERNHARD, A.B., M.D., 1816 Spruce Street. Associate Surgeon, Lankenau Hospital.

1904 *MITCHELL, CHARLES F., M.D., 2003 Pine Street. Surgeon, Germantown and Bryn Mawr Hospitals; Consulting Surgeon, Pennsylvania, Chestnut Hill, and St. Christopher's Hospitals.

1934 MOGAVERO, FRANCESCO, M.D., F.A.C.S., 1930 Chestnut Street. Assistant Surgeon and Director of Out-Patient Department, Misericordia Hospital.

1938 MOORE, JOHN R., A.B., M.D., F.A.C.S., 3701 N. Broad Street. Professor of Orthopedic Surgery, Temple University School of Medicine; Associate Professor of Orthopedic Surgery, University of Pennsylvania Graduate School of Medicine; Chief Orthopedic Surgeon, Shriner's Hospital and Temple University Hospital; Orthopedic Surgeon, Philadelphia General Hospital.

1906 *MULLER, GEORGE P., M.D., F.A.C.S., 1930 Spruce Street. Professor of Surgery, Jefferson Medical College; Surgeon, Jefferson Medical College Hospital and Misericordia Hospital.

1921 *MURPHY, EUGENE C., M.D., F.A.C.S., 1841 S. Broad Street. Surgeon, Doctors Hospital; Consulting Surgeon, Broad Street Hospital; Attending Specialist in Surgery to the United States Public Health Service.

1902 *Mutschler, Louis H., M.D., F.A.C.S., 1625 Spruce Street. Consulting Surgeon, Hospital of the Protestant Episcopal Church and Kensington Hospital for Women.

1938 †Nicholson, Jesse T., B.S., M.D., F.A.C.S., 1726 Spruce Street. Associate Professor of Orthopedics, University of Pennsylvania Graduate School of Medicine; Associate in Orthopedic Surgery, University of Pennsylvania School of Medicine; Orthopedist, Children's Hospital and Children's Seashore Home; Assistant Orthopedic Surgeon and Chief of Orthopedic Out-Patient Clinic, Pennsylvania Hospital; Assistant Orthopedist, Philadelphia General, Graduate and Philadelphia Orthopedic Hospitals; Consultant Orthopedic Surgeon, Children's Hospital of the Mary Drexel Home, Lankenau Hospital and Philadelphia Hospital for Contagious Diseases.

1934 †North, John P., A.B., M.D., F.A.C.S., 322 S. 21st Street. Associate in Surgery, University of Pennsylvania School of Medicine and Graduate School of Medicine; Clinical Professor of Surgery, Woman's Medical College; Surgeon, Philadelphia General and Memorial Hospitals; Associate Surgeon, Presbyterian Hospital.

1938 †Orr, Theodore E., B.S. M.D., 1930 Chestnut Street. Associate in Orthopedic Surgery, University of Pennsylvania Hospital; Chief Orthopedic Surgeon, Presbyterian, Methodist and Chester County Hospitals; Orthopedic Surgeon, Delaware County Hospital.

1915 *Owen, Hubley R., M.D., F.A.C.S., 319 S. 16th Street. Consulting Surgeon, Philadelphia General Hospital.

1939 †Parker, Alan P., A.B., M.D., 255 S. 17th Street. Instructor in Surgery, Jefferson Medical College; Assistant Surgeon, Pennsylvania Hospital; Assistant Attending Surgeon, Bryn Mawr Hospital.

1912 *Pfeiffer, Damon B., A.B., M.D., F.A.C.S., Meadowbrook, Pa. Associate Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Surgeon, Lankenau and Abington Memorial Hospitals; Consulting Surgeon, Woman's Hospital of Philadelphia.

1916 *Randall, Alexander, M.A. M.D., F.A.C.S., 136 S. 16th Street. Professor of Urology, University of Pennsylvania School of Medicine; Urologist, Abington Memorial Hospital, Hospital of the University of Pennsylvania and Chestnut Hill Hospital.

1938 Rankin, Lynn M., A.B., M.D., F.A.C.S., 200 Long Lane, Upper Darby, Pa. Instructor in Surgery, University of Pennsylvania School of Medicine; Demonstrator in Operative Surgery, Jefferson Medical College; Assistant in Surgery, Jefferson Hospital; Associate Surgeon, Presbyterian and Fitzgerald-Mercy Hospitals; Chief, Surgical Service, Delaware County Hospital.

1924 †Ravdin, Isidor S., B.S., M.D., F.A.C.S., 3400 Spruce Street. Harrison Professor of Surgery, University of Pennsylvania School of Medicine; Director of the Harrison Department of Surgical Research, University of Pennsylvania School of Medicine; Surgeon, University of Pennsylvania Hospital.

1941 †Ristine, Edwin R., A.B., M.D., F.A.C.S., 542 Cooper Street, Camden, N. J. Associate Surgeon, Cooper Hospital; Surgeon, Lakeland Hospital; Assistant Surgeon, Zurbrugg Hospital.

1928 †Robbins, Frederick Ross, B.S., M.D., F.A.C.S., 255 S. 17th Street. Instructor in Surgery, University of Pennsylvania School of Medicine and Graduate School of Medicine; Associate Surgeon, Children's Hospital; Assistant Surgeon, Pennsylvania Hospital and Bryn Mawr Hospital; Consulting Surgeon, Naval Hospital, Philadelphia.

1913 *Rodman, John Stewart, M.D., F.A.C.S., 1726 Spruce Street. Professor of Surgery, Woman's Medical College; Surgeon-in-Chief, Woman's Medical College Hospital; Surgeon, Bryn Mawr Hospital; Consultant in Neurological Surgery, Presbyterian Hospital.

1928 Rothschild, Norman S., M.D., 245 S. 16th Street. Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Associate Surgeon, Graduate Hospital; Surgeon, Jewish and Northern Liberties Hospitals.

1930 Ryan, Thomas J., M.D., F.A.C.S., 255 S. 17th Street. Surgeon, Misericordia Hospital and Fitzgerald-Mercy Hospital.

1920 Ryan, William John, A.B., M.D., F.A.C.S., 136 S. 16th Street. Attending Surgeon, St. Mary's Hospital and St. Vincent's Hospital for Women and Children.

1942 Schell, James F., B.S., M.D., F.A.C.S., 2506 S. 20th Street. Assistant Surgeon, Methodist Hospital.

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 Medical College Hospital; Active Consulting Surgeon, Philadelphia General Hospital; Director of Surgery, Delaware County Hospital; Consulting Surgeon to Montgomery County Hospital, Norristown, Sacred Heart Hospital, Norristown, and Grand View Hospital, Sellersville, Pa.

1924 Smyth, Calvin M., Jr., B.S., M.D., F.A.C.S., Methodist Hospital, Broad and Wolf Streets. Assistant Professor of Surgery, University of Pennsylvania Graduate School of Medicine; Surgeon-in-Chief, Methodist Hospital; Surgeon, Abington Memorial Hospital; Director of Surgery, Woman's Hospital of Philadelphia.

1935 Summey, Thomas J., M.D., F.A.C.S., 255 S. 17th Street. Instructor in Surgery, University of Pennsylvania Graduate School of Medi-

cine; Surgeon and Medical Director, Burlington County Hospital; Assistant Surgeon, Pennsylvania and Children's Hospitals.

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

1911 *THOMAS, T. TURNER, M.D., F.A.C.S., 1636 Francis Street. Surgeon-in-Chief, Northeastern Hospital.

1915 *THOMAS, W. HERSHEY, M.D., F.A.C.S., 1505 Race Street. Professor of Urology, Temple University School of Medicine; Urologist, Philadelphia General and Temple University Hospitals.

1928 †WAGONER, GEORGE W., M.D., Haverford, Pa. Professor of Orthopedic Research, University of Pennsylvania Graduate School of Medicine; Assistant Professor of Orthopedic Research, University of Pennsylvania School of Medicine; Associate in Pathology, University of Pennsylvania School of Medicine; Consulting Surgeon, Woman's Hospital; Orthopedist, Bryn Mawr Hospital; Orthopedic Surgeon, Graduate Hospital of the University of Pennsylvania; Clinical Surgeon, Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases.

1928 WALKLING, ADOLPH A., M.D., F.A.C.S., 136 S. 16th Street. Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital and Pennsylvania Hospital; Consulting Surgeon, Frankford Hospital.

1928 WEEDER, S. DANA, M.D., 250 Tulpehocken Street. Chief Surgeon, Germantown Hospital; Associate Surgeon, Chestnut Hill Hospital.

1919 WILLARD, DEFOREST P., M.D., 1726 Spruce Street. Vice-Dean of Orthopedics, University of Pennsylvania Graduate School; Orthopedic Surgeon, Graduate Hospital and Abington Memorial Hospital; Consulting Orthopedic Surgeon, Bryn Mawr, Chestnut Hill, and Pennsylvania Hospitals.

1927 WILLIAMSON, ERNEST G., M.D., F.A.C.S., F.R.C.S. (Edin.), 319 S. 16th Street. Instructor in Surgery, University of Pennsylvania School of Medicine; Associate Surgeon, Children's and Presbyterian Hospitals; Clinical Assistant, Jefferson Medical College Hospital.

1898 *WOOD, ALFRED CONARD, M.D., PH.G., F.A.C.S., 2035 Walnut Street. Honorary Consulting Surgeon, Philadelphia General Hospital; Consulting Surgeon, Montgomery Hospital, State Hospital for Insane, Norristown and Coatesville Hospital, Coatesville, Pa.

NON-RESIDENT FELLOWS

1939 BRADSHAW, HOWARD H., M.D., F.A.C.S., 2611 Renolds Road, Winston-Salem, N. C. Professor of Surgery and Director of the Division of Surgery, Bowman Gray School of Medicine of Wake Forest College; Surgeon, Baptist Hospital, Winston-Salem, N. C.

1900 JOPSON, JOHN H., M.D., F.A.C.S., Pawleys Island, S. C. Emeritus Professor of Surgery, Graduate School of Medicine, University of Pennsylvania.

1925 KEATING, PETER MCCALL, 222 King William Street, San Antonio, Texas. Lt. Col. Medical Reserve Corps, U. S. Army; Chief Orthopedic Section, Station Hospital, Fort Sam Houston.

1908 SWEET, JOSHUA E., M.A., M.D., D.Sc., F.A.C.S., Unadilla, N. Y. Emeritus Professor of Experimental Surgery, Cornell University Medical College.

1934 WEBER, EDGAR H., B.S., M.D., F.A.C.S., 123 S. E. Second Street, Evansville, Ind., Surgical Staff, Protestant Deaconess and St. Mary's Hospitals.

1923 WELLS, J. RALSTON, A.B., M.D., 204½ S. Beach Street, Daytona Beach, Florida. Senior Attending Surgeon, Senior Staff, Halifax District Hospital; Consulting Surgeon, DeLand Memorial Hospital, DeLand.

ARMY AND NAVY MEMBERS

1940 ANDERSON, THOMAS C., A.B., M.D., F.A.C.S., Capt. U. S. Navy, U. S. Naval Hospital, Philadelphia.

1938 CONNER, HASKETT L., M.D., F.A.C.S., Lt. Colonel, Medical Corps, U. S. Army.

1938 CORBY, JOHN F., M.D., Lt. Colonel, Medical Corps, U. S. Army.

1939 HITCHENS, ARTHUR P., M.D., Lt. Colonel, Medical Corps, U. S. Army, retired. George S. Pepper Professor, Preventive Medicine and Public Health, University of Pennsylvania School of Medicine.

1942 SHARR, CAMILLE, M.D., Commander, U. S. Navy, U. S. Naval Hospital, Philadelphia, Pa.

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	William J. Mayo, Rochester, Minn.	July	28, 1939
1906	Dudley P. Allen, Cleveland, Ohio	January	6, 1915
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

ELECTED		DIED
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. de Quervain, 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	
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.	
1929	George W. Crile, Cleveland, Ohio	January 7, 1943
1929	Edward Starr Judd, Rochester, Minn.	November 30, 1935
1929	Dallas B. Phemister, Chicago, Ill.	
1933	John H. Jopson, Mills, N. C.	

FELLOWS DECEASED SINCE LAST PUBLICATION

1939 Thomas R. Neilson

1940 Charles F. Nassau

1940 Louis D. Englerth

1941 James H. Baldwin

1942 James Torrance Rugh

THOMAS RUNDLE NEILSON

Dr. Thomas Rundle Neilson, a skillful and experienced surgeon, a revered and cultured gentleman of the old school, died in the hospital at Bryn Mawr, Pennsylvania, on October 25, 1939. Had he lived four more days he would have been 82 years of age.

Thomas Rundle Neilson was born October 29, 1857, at 325 South Twelfth Street, Philadelphia, which house was occupied by the Neilson family for a period of 70 years.

Doctor Neilson's ancestors were Scotch-Irish. His paternal grandfather, Robert, was born in Strabane, Ireland, and his paternal grandmother was born in Londonderry. Robert Neilson was made Governor of Trinidad, British West Indies, under the English Crown, and Doctor Neilson's father was born on that island during his father's term as Governor. His maternal grandfather was William D. Lewis, one-time Collector of the Port of Philadelphia, and he was also secretary to Henry Clay when that man was Ambassador to Russia. His maternal grandmother was Sarah Claypoole. The doctor was the fourth child of Thomas and Sarah Claypoole Neilson. The family consisted of William, Robert Henry, Sarah, Thomas Rundle, Lewis, Emma Florence, Mary Alice Lewis, and Frederick Brooke. Of these children only three survive—Lewis, a retired vice-president of the Pennsylvania Railroad, Emma Florence, and Mary Alice Lewis. When Doctor Neilson's father was a small boy he was brought to Philadelphia by his parents, where the family have since resided.

Doctor Neilson married Louise Fotterall and they had one son, Thomas Rundle, Jr.

The doctor received his early education at the Episcopal Academy, then located in Juniper street. He was graduated from the University of Pennsylvania in Arts in 1877, after which he entered the medical department and was graduated in 1880.

He started his internship at the Episcopal Hospital in 1880, at which time there were but four interns. In 1886 he was appointed Surgeon of the Staff, which position he held continuously until his resignation in 1924 after 44 years of faithful, active service to the hospital—a period of service that has never been equaled at the hospital. When Doctor Neilson's name was mentioned for appointment as Surgeon to the hospital, one of the managers remarked that he considered him too young for such a responsible position, to which another manager replied that he thought he would outgrow the objection. At the time of his resignation in 1924 he was appointed Surgeon Emeritus, which honor he held until his death.

In 1907 he was elected president of the Medical Board and was still active when taken ill. He rarely missed a meeting, and the last letter he

wrote was one of regret that he did not feel able to attend the October meeting. His judgment and advice were most helpful and constructive.

During his early days of service to the hospital the automobile and electric cars were not in use, and it was his custom to travel to his daily visits to the hospital in the horse cars. He rarely failed to respond to any calls, no matter at what time they might come, as they frequently did on cold, stormy, wintry nights. I can recall seeing the doctor arrive at the hospital bundled in heavy overcoat, muffler, and galoshes, to examine and often to operate on some emergency case. He was always deeply interested in his cases, studying them thoroughly and personally dressing the most critical cases rather than have the interns take the responsibility. He was kind and gentle to his patients, often calling them by their first names and frequently giving them a nickname, as was his custom with his friends and associates.

In the operating room he paid strict attention to asepsis and technic. He was a skillful operator, quick and careful, and his thorough knowledge of anatomy enabled him to meet and cope with any emergency that might arise. Rarely did he speak crossly to his assistants or nurses, and his quiet manner, kindness, and sympathy endeared him to all of those who had the good fortune to work with him. The hospital sustained a great loss through Doctor Neilson's death, and the Board of Managers, in recognition of his long and faithful service, passed a resolution and ordered it spread upon the minutes and a copy sent to his son Thomas, Jr. Following is an abstract of the resolution:

> "Any institution which has had the thought, interest, and services of a man such as Doctor Neilson over such a long period has been fortunate, and we, the present managers of the Episcopal Hospital, wish to spread upon these minutes our sense of appreciation of the part Doctor Neilson took in making the hospital what it is today."

The following is an abstract from the minutes passed at a special meeting of the Medical Board held October 26, 1939:

> "He was a surgeon, safe, skillful and seasoned; a scholar both as a teacher and a student; an associate who had neither rancor nor jealousy; a man with faults as lovable as his virtues were great. We here record that we have lost a keystone. Thomas Rundle Neilson is a luminary in the annals of our history."

The high esteem in which Doctor Neilson was held by his associates is eloquently expressed in the following letter from the dean of the Medical Department of the University of Pennsylvania:

> "Thomas Rundle Neilson graduated in Arts in 1877, Medicine in 1880; two years later in 1882 he became demonstrator of Anatomy; in 1884 he became demonstrator of Surgery and in 1885 Instructor in G. U. In 1901 he was made Clinical Assistant Professor of G. U. diseases, then Clinical Professor, and in 1912 Professor of G. U.

Surgery. In 1923 he retired on account of age. He taught in the Medical school 41 years and was Emeritus Professor for 16 years. He was recognized as an authority in his specialty, an able teacher and clinician, and his retirement from active participation in the affairs of the Medical school and Hospital was a distinct loss to our school.

"Doctor Neilson was a well-beloved member of our faculty. His kindly and genial nature, his sense of humor, and his spirit of comradeship endeared him to all."

Signed, William Pepper, Dean.

For a period of years Doctor Neilson was surgeon to St. Christopher's Hospital. He was a fellow of the College of Physicians, of which he was president from 1922 to 1925, and a fellow of the American College of Surgeons, a fellow of the Philadelphia Academy of Surgery; a member of the Philadelphia County Medical Society; and a member of the Rittenhouse Club and of the Zeta Psi Fraternity.

While in college he showed much interest in cricket, which was at that time quite a popular game. His mother's summer home was at Florence, New Jersey, on the Delaware river, and there he learned to row, becoming quite expert, and he frequently competed in races on the river. Next to surgery his greatest interest was music. During his college days he organized and led a glee club of eight men, a double quartette, which was the nucleus of the present U. of P. Glee Club. Two concerts were held in the college chapel and were managed by him. He studied the pipe organ with David Wood, the blind organist of St. Stephen's Church, and composed the music for one of the Easter hymns which was sung by St. Stephen's choir. He played the organ and piano exceptionally well and for several summers substituted in St. James Church at 22nd and Walnut Streets for the organist, Mr. Dixon, while the latter was on vacation. He was a stockholder of the Academy of Music and attended the opera regularly. He was much interested in the affairs of the Episcopal Church, of which he was a lifelong member. He was a member of the Vestry of the Church of the Ascension, was Rector's Warden, and delegate to the Diocesan convention for that church for several years.

Doctor Neilson is buried in the churchyard of Old St. Davids, Radnor.

LOUIS H. MUTSCHLER.

CHARLES FRANCIS NASSAU

DR. CHARLES FRANCIS NASSAU died on August 11, 1940, a great loss the city of Philadelphia, the medical profession, and to Jefferson Medical College.

The son of a Presbyterian medical missionary, Doctor Nassau was born in Benita, French Congo, West Africa, on November 12, 1868. When Charles was two years old, his parents returned to this country, establishing residence in Philadelphia, where he received his preliminary education in the Hancock Grammar School, Friends' School, and Eastburn Select School. Ill health in his childhood was responsible for these changes. Later, he attended Pennsylvania Military College, taking a two-year course in civil engineering. At the end of his second year, with an excellent scholastic standing, he decided to study medicine. Taking the first step in this direction, he entered the School of Biology at the University of Pennsylvania for a two-year course. While studying this subject, Doctor Nassau served as prosector and official photographer on the Brooks' Expedition to the Bahama Islands in 1887. This expedition was under the leadership of Professor Brooks of Johns Hopkins University. In the summer of 1889 he was employed by the Smithsonian Institution in Washington preparing the skeletons of small mammals and birds.

In 1888 he matriculated at the Medical School of the University of Pennsylvania, being graduated in 1891. His internship was served at Presbyterian Hospital, Philadelphia, and subsequently he took postgraduate work in pathology and surgery at the Old German Hospital. Later, he went abroad, studying in Berlin, Strassburg, and the University of Vienna. While in Europe, Doctor Nassau became a student of von Bergmann, von Olshausen and von Recklinghausen. He worked under Professor Rubner in the University of Berlin, taking a course in bacteriology, and took special courses in operative surgery under Professor vonSonnenburg. At the University of Vienna he studied general pathology under Koliske and worked with Krause in internal medicine. When time permitted, special work was taken in the use of the ophthalmoscope and special diagnostic courses with Doctor Regnier, Chrobak's first assistant, in diagnosis of diseases of women. Nassau was also associated with Budinger in Billroth's Klinik in Surgical Diagnosis.

In the spring of 1894 he went to Birmingham, England, having been appointed Clinical Assistant to Mr. Lawson Tait. This appointment was to have lasted for six months, but he was asked to return to the United States to work on the Surgical Service of Professor William S. Halsted at Johns Hopkins Hospital. He therefore left England and reached Baltimore in September, 1894, working at Johns Hopkins as a Resident in Surgery for a period of 13 months.

In 1895 Doctor Nassau came to Philadelphia and began the practice of medicine, working in the Outpatient Department of the Old German Hospital and at St. Joseph's Hospital. In the latter institution, he met Dr. J. Chalmers DaCosta and a true friendship lasting a lifetime sprang up between them. DaCosta was impressed with Nassau's ability and persuaded him to attend Jefferson Medical College. He entered the Senior Class, receiving a degree from the Institution in 1906.

Doctor Nassau then began to climb the ladder of promotion in the Surgical Department at Jefferson, beginning as Demonstrator of Anatomy, under the late George McClellan, and mounting to the rank of Professor of Clinical Surgery, the position he held until his death.

In addition to his work at Jefferson, Doctor Nassau was Surgeon-in-Chief to Frankford Hospital; Surgeon to St. Joseph's Hospital; Surgeon to Mt. Sinai Hospital, and Consulting Surgeon to many hospitals throughout the state.

He served as president of the Philadelphia County Medical Society and as director of the Department of Public Health. He received the degree of LL.D. from Villanova College and of Sc.D. from St. Joseph's College. He was a member of numerous medical and surgical societies in the United States.

Not only was Doctor Nassau a distinguished surgeon, but he was a sportsman in the truest sense of the word. His interests were varied, comprising camping, hunting, fishing, and photography. He was a member of the Easton Anglers' Association, the Izaak Walton League of America, the Camp and Trail Club, the Quaker City Gun Club, and many other societies devoted to sports and civic interests.

In World War I, Doctor Nassau served as Chief Surgeon, Base Hospital No. 38, having the rank of major. He was the recipient of a World War medal, Meuse-Argonne, Defensive Sector. He was honorably discharged from active service, remaining in the Medical Reserve Corps with the rank of lieutenant colonel.

He was a brilliant student, a skilled surgeon, a great man.

THOMAS A. SHALLOW.

LOUIS DANIEL ENGLERTH

DR. LOUIS D. ENGLERTH was, during his entire professional career, a student of surgery. This is best illustrated by the fact that upon completion of his notable service with the American Expeditionary Forces, he attended the University of Edinburgh, in Edinburgh, Scotland, and in 1919 received his degree of F.R.C.S.

He had been a member of U. S. Hospital No. 38; was detailed with an operating team at Evereux, France; with Evacuation Hospital No. 6 at Souilly, France; and with Mobile Hospital No. 1 at Esnes, France.

He endeared himself to those who were associated with him in U. S. Hospital No. 38, as he had with his many associates in Philadelphia.

A native of Dayton, Ohio, and having received his preliminary education in that city, he received his degree of Doctor of Medicine from Jefferson Medical College in 1914.

After having served as Intern and Chief Resident at the Frankford Hospital, and for many years as Assistant to Dr. Charles F. Nassau, he was appointed as Surgeon to the Frankford Hospital, in the neighborhood where he lived, and where he enjoyed an enviable reputation as a friend and surgeon to his many patients.

He was Surgeon to St. Joseph's Hospital; the Philadelphia General Hospital; the Northeastern Hospital, and to Girard College, and Consulting Surgeon to Grandview Hospital, Sellersville, Pa.; Friends Hospital, Philadelphia, and Baptist Home, Philadelphia.

In addition to these many hospital duties, he imparted his knowledge and experience to the students of Jefferson Medical College, where he was Demonstrator in Surgery.

His membership in medical and surgical societies and clubs was legion.

Doctor Englerth, who was known as "Louie" to his many friends, was always admired for the sense of earnest responsibility of his profession; for his modesty; his quiet sense of humor and for the strict interpretation of medical ethics, to which interpretation he adhered so drastically.

It could be truly said that Doctor Englerth had no enemy. He was admired and beloved by all the members of the medical profession, and by his patients.

Doctor Englerth died in the Jefferson Hospital on August 16, 1939, at the age of fifty—far too prematurely. He has been and will be greatly missed.

We share with his widow, his sister, and his brother the deepest sympathy of his loss.

HUBLEY R. OWEN.

JAMES HARVEY BALDWIN

Dr. James Harvey Baldwin was born in East Liberty, Ohio, in 1871. The son of a country doctor, he decided early in life to study medicine. His early education was received in local schools, and upon graduation from high school he entered Wooster College, receiving his A.B. from that institution in 1897. The following Fall he matriculated in the Medical Department of the University of Pennsylvania and was granted the M.D. degree in 1897. On the day of his graduation from Medical School he was married to Clarabel Taylor, also of East Liberty. Two children, James Todd and Mary Nicholson, were born of this union. His widow and his two children survive him.

Following an internship in the Mercy Hospital, Pittsburgh, he returned to Philadelphia where he soon developed a large general practice in the South Philadelphia district. In 1902 he became associated with the Methodist Hospital as assistant to Dr. James Hutchinson, but continued his general practice for some years afterward. During the First World War he served as a Captain in the Army, and following his discharge in 1919 was appointed surgeon to the Methodist Hospital, succeeding to the position vacated by his former chief.

Doctor Baldwin was elected to Fellowship in the Academy of Surgery in 1917, serving for two years as a member of the Council. In addition he was a Fellow of the Philadelphia College of Physicians and the American College of Surgeons.

In 1932, because of failing health, he retired from his active service at the Methodist, a step which he took with the greatest reluctance since it was in that institution he had spent practically his entire professional life. From that time he was never quite the same and continuing poor health and increasing financial worries eventuated in a general nervous breakdown, necessitating his admission to the Veterans' Hospital at Coatesville, where he died April 18, 1941, and was buried in the National Military Cemetery in Baltimore.

Although a surgeon for thirty years, Doctor Baldwin was at heart a "family doctor." He brought to his patients a keen personal interest and their regard for him was only slightly short of worship. He spent much time in the Children's Ward, and often would take an hour or more to do a dressing if he thought that by so doing he could save a patient pain. He sometimes allowed his great natural sympathy to surmount his best judgment in these matters. He was conscientious almost to a fault, and because of his consideration for the feelings of others he was often taken advantage of. These characteristics prevented him from ever acquiring a large surgical

practice. His last years were marked by many disappointments and disillusionment, but he at no time expressed any bitterness. A sound surgeon, a great gentleman, a loyal friend, and a good companion, he was truly a gentle soul who deserved far better of the world than it gave him.

Calvin M. Smyth, Jr.

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 of 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.

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

ANNUAL ADDRESS FOR 1939

LOBECTOMY FOR CHRONIC PULMONARY SUPPURATION

JOHN B. FLICK, M.D.

BRYN MAWR, PA.

THE treatment of chronic pulmonary suppuration, until recently, has provided a perplexing problem in therapeusis. It is only within the last decade that progress in thoracic surgery has made possible, with any degree of safety and success, the surgical treatment of those who suffer from this loathsome, disabling, and ofttimes fatal disease.

Until modern diagnostic methods became available, it was difficult or impossible to differentiate, except at autopsy, between chronic pulmonary abscess, bronchiectasis, and cystic disease of the lung, nor was it possible to determine with any exactness the extent of the pathology present.

The work of Chevalier Jackson in diagnostic bronchoscopy and of Jackson[1] and Lynah and Stewart,[2] in mapping out the bronchial tree by means of injecting substances opaque to the roentgen-rays, and the introduction of iodized oil for this purpose by Sicard and Forestier,[3] has been invaluable in determining the nature, location, and extent of lesions of the bronchi and lungs, and has thereby furthered their treatment.

The determination of the extent of the pathology present is of even greater importance than the differentiation between the various lesions which give rise to chronic pulmonary suppuration, with the exception of pulmonary neoplasm. All of these lesions must be dealt with radically, and no diseased tissue permitted to remain if satisfactory results are to be obtained. Paget,[4] writing in 1896, said, "We see that simple abscess and bronchiectasis, though they are of different origins and run different courses, are not sharply defined from each other surgically. But if we take, on the one hand, cases of large single simple abscess of the lung, and on the other, cases of advanced bronchiectasis, where a great part of the lung is riddled with small irregular tortuous cavities, we see how different the chances of successful operation must be in these two diseases." Unfortunately, too often, acute pulmonary abscess leads to bronchiectasis when simple drainage can no longer effect a cure of the disease.

The high mortality of lobectomy during the first quarter of this century discouraged its employment, except by a few men who were convinced that radical surgery offered the only hope for cure in bronchiectasis and who had the vision and the courage to persist in their efforts to develop it. In 1925 Lilienthal[5] reported 32 cases of lobectomy for chronic pulmonary suppuration,

* The Annual Address before the Philadelphia Academy of Surgery, October 7, 1940. Reprinted from BULLETIN OF THE AYER CLINICAL LABORATORY, 3, 375–394, No. 17, December, 1941.

with 19 deaths, a mortality rate of approximately 60 per cent.* Although other surgeons reported isolated cases and small groups were reported, Lilienthal's series is fairly representative of the mortality rate of that particular period.

As late as 1929 there was still an average death rate from lobectomy for bronchiectasis of nearly 60 per cent.[6] In 1929 Brunn[7] published an article on "Surgical Principles Underlying One Stage Lobectomy," in which he reported six operations with four cures, one improved, and one death. He demonstrated the possibility of removing a lobe of the lung in a free pleural cavity with relative safety, and laid the foundations for the modern one-stage lobectomy. Brunn's paper attracted widespread attention and revived the interest of many surgeons in the problems of this operation. In 1932 Shenstone and Janes[8] added to Brunn's technic the use of an ingenious tourniquet for controlling the hilum during lobectomy. Since the appearance of Brunn's paper in 1929, there has been a steady reduction in the mortality rate. In 1937 Churchill[9] reported a series of 40 patients in whom lobectomy for bronchiectasis and cystic disease of the lung had been completed with a mortality rate of 5 per cent. This series included a death following a first-stage operation. He had a mortality rate of 2.6 per cent for 38 patients subjected to lobectomy by methods "now recommended," and no mortality in the last 30 successive cases. Churchill employed a one- or two-stage procedure, according to the problem presented by the individual patient. When he employed a two-stage procedure, he delayed the second stage, the actual removal of the lobe, for at least six weeks, and in most cases, considerably longer, and noted improvement during the interval in almost all of his patients.

Edwards[10] in 1939 reported 166 individual operations for lobectomy with a mortality of 12 per cent. Six of these patients had removal of the lower lobe, followed by removal of the upper lobe at intervals of four weeks to three years, thus the upper-lobe lobectomy meant completion of a total pneumonectomy in two stages. Only two deaths occurred in the last 54 cases of lobectomy—a mortality rate of 3.7 per cent—and both of these patients had advanced and extensive disease. Edwards' table of mortality rate according to age showed that in the group aged from 4 to 16, comprising 38 children, there were no deaths. This table is of further interest in that it "confirms a view generally held that the older the patient the greater the risk of this operation." Edwards has consistently employed a one-stage operation, but in his last eight lobectomies in adults, he has practiced "pleural poudrage" as a preliminary measure to promote adhesions. The modern two-stage lobectomy is a composite of technical features taken from the operations devised by a number of men and assembled as experience accumulated. At the first stage, adhesions about the diseased lobe are separated, the pulmonary ligament is divided, and an attempt is made to create adhesions over the healthy lobes.

* Dr. Lilienthal gives a table of 34 cases. Some of these had had more than a lobe removed. Two cases are classified as total pneumonectomies, and the latter are excluded in arriving at the mortality rate.

TABLE I

ONE STAGE LOBECTOMY

Case	Age, Sex	Duration of Symptoms	Etiology and History of Previous Treatment	Sinus Disease	Amount of Sputum	Hemoptysis	Lobes Involved	Operation and Date	Residual Empyema	Remarks	"Follow-up"	Result
1 E. W.	30 M.	22 yrs.	Pneumonia. Bronchoscopic treat. Phrenic exeresis 4–19–28. Partial thoracoplasty preliminary to lobectomy 5–29–29	No	Small	Severe pulmonary hemorrhages	R. L.	Removal of R.L.L. 6–5–29	Small	Bronchocutaneous fistula. Closed by operation 12–27–29	7–19–40 (11 yrs.) No cough or expectoration. Wound healed. Normal activity. No hemoptysis since lobectomy	Cured
2 H. A.	26 M.	9 yrs.	Pneumonia. Bronchoscopic treat. Phrenic exeresis 5–24–32	No	90 to 120 cc. per day	No	L.L.	Removal of L.L.L. 3–3–33	Small. Treated by irrigation through tube for closed drainage. No evidence of open bronchus	Dschg. from hosp. 4–22–33. Readm. 7–25–34 & residual emp. drained. Osteomyelitis of rib end. Reoperated upon for similar condition in April, 1935	7–19–40 (7 yrs.). No cough or expectoration. Wound healed. Normal activity	Cured
3 A. S.	27 M.	3 yrs.	Pneumonia. Bronchoscopic treat. Pneumothorax unsuccessful. Phrenic exeresis 3–17–33	No	540 to 720 cc. per day	No	L.L.	Removal of L.L.L. 4–28–33	Small. Treated by irrigation through tube for closed drainage	5–8–33. Hemorrhage due to erosion of pulmonary vessel in wall of residual empyema cavity		Death (operative)
4 S. W.	32 M.	9 mos.	Pulm. abs. following appendectomy. Bronchoscopic treat. Pneumothorax unsuccessful. Phrenic nerve crushed 2–24–33	No	Large	Yes	L.L.	Removal of L.L.L. 5–12–33		Within few hours developed mediastinal and subcutaneous emphysema. Tension pneumothorax not present. Died 5–13–33		Death (operative)
5 J. R.	16 M.	10 yrs.	Pertussis 1924. Pneumonia and empyema left side in 1927. Thoracotomy done. Bronchoscopic treat. Phrenic exeresis 6–23–31	No	Small	Frequent blood spitting	L.L.	Removal of L.L.L. 12–14–34	Small. Open drainage established on 10th day. Evidence of open bronchus		7–19–40 (5 yrs., 5 mos.). No cough or expectoration. Wound healed. Normal activity. No hemoptysis since lobectomy	Cured

TABLE I—*Continued*

Case	Age, Sex	Duration of Symptoms	Etiology and History of Previous Treatment	Sinus Disease	Amount of Sputum	Hemoptysis	Lobes Involved	Operation and Date	Residual Empyema	Remarks	"Follow-up"	Result
6 C.B.	28 M.	1 yr., 5 mos.	Pulm. infection following tonsillectomy. Bronchoscopic treat. Pneumothorax unsuccessful	Yes	Small	Frequent severe pulm. hemorrhages	R.L.	Removal of R.L.L. 11–15–35	Small. Open drainage established on 7th day. Evidence of open bronchus on 10th day	Bronchocutaneous fistula closed by operation 1–13–37	7–19–40 (4 yrs., 8 mos.). No cough or expectoration. Wound healed. Normal activity. No hemoptysis since lobectomy	Cured
7 A. Y.	16 M.	3 yrs.	Pulm. abs. L.U.L. following tonsillectomy Aug. '32. Bronchoscopic treat. Pneumothorax unsuccessful. Ext. drainage of pulm. abs. Oct. and Dec. '34. Bronchocutaneous fistula	Yes	Small	Pulmonary hemorrhages	L.U. and L.L.	Removal of L.L.L. 11–29–35. Removal of L.U.L. 5–25–36. (Carr "automatic hilar ligature" used)	Small. Open drainage established on 32nd day. Evidence of open bronchus. Small. Open drainage established on 14th day. Evidence of open bronchus		7–27–40 (4 yrs.). No cough or expectoration. Wound healed. Deformity of chest and scoliosis, marked. Normal activity. No hemoptysis since last operation	Cured
8 R. P.	18 F.	2 yrs., 4 mos.	Pulm. abs. L.U.L. following tonsillectomy, Aug. 1934. External drainage April and May 1935. Bronchocutaneous fistula	No	Small	No	L.U.	Removal of L.U.L. 12–2–36. (Carr "automatic hilar ligature" used)	Small. Open drainage established on 9th day. Evidence of open bronchus		7–18–38 (1 yr., 7 mos.). No cough or expectoration. Wound healed. Normal activity	Cured
9 D. B.	12 M.	4 yrs.	Pneumonia and empyema, right side, 1934	No	400 to 500 cc. per day	No	R.M. and R.L.	Removal of R.M. and R.L.L. 1–10–38	Small. Open drainage established on 7th day. Evidence of open bronchus		7–30–40 (2 yrs., 6 mos.). No cough or expectoration. Wound healed. Normal activity	Cured
10 L. S.	15 F.	3 yrs. +	Pneumonia in 1936, 1937, and 1938	Yes	30 to 60 cc. per day	Yes	L.L.	Removal of L.L.L. 1–25–39	None. Tube for closed drainage removed on 9th day	Sulfanilamide used before and after operation	7–26–40 (1 yr., 6 mos.). No cough or expectoration. Wound healed. Normal activity	Cured

TABLE I—*Continued*

Case	Age, Sex	Duration of Symptoms	Etiology and History of Previous Treatment	Sinus Disease	Amount of Sputum	Hemoptysis	Lobes Involved	Operation and Date	Residual Empyema	Remarks	"Follow-up"	Result
11 E. W.	29 F.	5 yrs.	Chronic nasal accessory sinus disease	Yes	60 to 120 cc. per day	Yes	R.L. (saccular) and L.L. (cylindrical)	Removal of R.L.L. 6–21–39	Tube for closed drainage removed on 12th day. Small emp. drained on 50th day. Bronchus open	Sulfanilamide used before and after operation	7–11–40 (1 yr.). Improved. Still cough and expect. To return for removal of L.L.L.	Improved
12 E. K.	18 F.	15 yrs.	Measles and chickenpox at 3 yrs. of age. Chr. sinus disease. Chr. otitis media	Yes	60 to 90 cc. per day	No	L.L. (saccular) R.L. (few cylin. dilatations)	Removal of L.L.L. 9–29–39	None. Tube for closed drainage removed on 10th day	Sulfanilamide used before and after operation. Individual ligation of hilar structures	7–23–40 (10 mos.). Slight cough but no expect. Normal activity	Greatly improved
13 J. P.	24 F.	6 yrs.	Pulm. abs., R.L.L., following tonsillectomy	No	60 to 90 cc. per day	No	R.L.	Removal of R.L.L. 1–31–40	None. Tube for closed drainage removed on 12th day	Sulfanilamide used before and after operation	7–9–40 (6 mos.). No cough or expectoration. Wound healed. Normal activity	Cured

TABLE II

TWO STAGE LOBECTOMY

Case	Age, Sex	Duration of Symptoms	Etiology and History of Previous Treatment	Sinus Disease	Amount of Sputum	Hemoptysis	Lobes Involved	Operations and Dates	Residual Empyema after Lobectomy	Remarks	"Follow-up"	Result
1 J. O'D.	33 M.	20 yrs.	Cough since childhood. Tonsillectomy in 1930, followed by acute symptoms. Bronchoscopic treatment. Phrenic exeresis 3–6–31	No	120 to 180 cc. per day	No	R.L.	1st stage 11–7–31. Removal of R.L.L. 12–14–31	Small. Open drainage established on 9th day. Evidence of open bronchus	Symptoms of cerebral abscess on 19th day. Death on 25th day		Death (operative)
2 S. M.	22 F.	Since infancy	Chronic sinus disease (antra and ethmoid). Bronchoscopic treat. Pneumothorax unsuccessful. Phrenic exeresis 1–20–32	Yes	120 to 150 cc. per day	No	R.L.	1st stage 5–18–34. Removal of R.L.L. and lingula of R.U.L. 8–14–34 (complete transposition of viscera)	Small. Open drainage established on 10th day. No evidence of open bronchus		Feb. '38 (3 yrs., 6 mos.) Slight cough and expect. at times. Wound healed. Being treated for sinus disease. Normal activity	Greatly improved
3 B. T.	17 F.	3 yrs.	Pneumonia. Bronchoscopic treatment	No	30 to 60 cc. per day	No	L.L.	1st stage 1–25–35		Bronchopneumonia. Empyema. Fibrinous pericarditis. Hemolytic strept. Death on 11th day. (Autopsy)		Death (operative)
4 L. H.	24 F.	Since infancy	Measles at 9 mos. followed by pertussis. Operation for ethmoid disease at 6 yrs. Bronchoscopic treat.	Pansinusitis. Chr. ear disease	30 to 60 cc. per day	Yes	R.M. (saccular) R.L. (a few cylindrical dilations)	1st stage 2–20–35. Removal of M.L. 4–22–35	Small. Open drainage established on 39th day. Probably bronchus open		8–14–40 (5 yrs., 4 mos.) Greatly improved. Still slight cough and expectoration. Still suffering from chronic disease of sinuses. Wound healed. Normal activity	Greatly improved
5 J. B.	12 M.	11 yrs.	Pneumonia. Bronchoscopic treatment. Phrenicectomy 4–11–32	No	30 to 60 cc. per day	No	R.L.	1st stage 1–30–35. Removal of R.L.L. 6–28–35	Small. Open drainage established on 10th day	Developed scarlet fever after 1st stage	7–25–40 (5 yrs.). No cough or expectoration. Wound healed. Normal activity	Cured

TABLE II—(*Continued*)

TWO STAGE LOBECTOMY

Case	Age, Sex	Duration of Symptoms	Etiology and History of Previous Treatment	Sinus Disease	Amount of Sputum	Hemoptysis	Lobes Involved	Operations and Dates	Residual Empyema after Lobectomy	Remarks	"Follow-up"	Result
6 R. McD.	24 F.	2 yrs.	Acute respiratory infection	Yes	30 to 60 cc. per day	Pulmonary hemorrhages	L.L.	1st stage 3–22–35. Removal of L.L.L. 7–5–35	Small. Open drainage established on 14th day. Probably bronchus open		7–26–40 (5 yrs.). No cough or expectoration. No hemoptysis since lobectomy. Wound healed. Normal activity	Cured
7 V. D.	20 F.	16 yrs.	Pertussis at 3 yrs. Bronchoscopic treat.	Pansinusitis	45 to 90 cc. per day	No	L.L.	1st stage 6–21–35. Removal of L.L.L. 10–11–35	Small. Open drainage established on 17th day		Never completely free from cough and expect. Bronchoscopy showed pus coming from L.U.L. Bronchopneumonia 3 yrs. after lobectomy, from which she died	Death (3 yrs. after lobectomy)
8 J. R.	15 F.	2 yrs., 6 mos.	Influenza	Yes	Small	Frequent	L.L.	1st stage 7–12–35. Removal of L.L.L. 12–13–35		Developed anuria following blood transfusion. Died on 9th day. (Autopsy)		Death (operative)
9 E. C.	21 F.	4 yrs.	Pulmonary abscess following tonsillectomy in July, 1933. Bronchoscopic treat. Pneumothorax unsuccessful. Three unsuccessful attempts to mobilize R.L.L.	No	Small	Frequent	R.L.	Partial mobilization of R.L.L. 1–18–35. Removal of R.L.L. 6–16–37	Small. Open drainage established on 14th day. No evidence of open bronchus	Marked improve- after mobilization of R.L.L. 1–18–35	6–26–40 (3 yrs.). No cough or expectoration. Wound healed. Normal activity	Cured
10 F. S.	29 M.	4 yrs.	Measles followed by acute mastoiditis. Mastoidectomy. Bronchoscopic treatment	Yes	30 to 60 cc. per day	Yes	L.L.	1st stage 12–10–37. Removal of L.L.L. 2–7–38	None	Tube for closed drainage removed on 11th postoperative day	7–19–40 (2 yrs., 5 mos.) No cough or expect. Wound healed. Normal activity	Cured
11 L. G.	17 F.	8 yrs.	Pneumonia in 1930 (right). Pneumonia and empyema in 1936 (R). Maxillary and frontal sinuses operated upon in 1934	Pansinusitis	60 to 120 cc. per day	Yes	R.L. and L.L. (involvement more extensive on left side)	1st stage 10–12–38. Removal of L.L.L. 9–27–39	Small. Open drainage established on 11th postoperative day. Evidence of open bronchus	Sulfanilamide given before and after lobectomy. Individual ligation of hilar structures	7–29–40 (10 mos.). Improved. Still has cough and expectoration. To return for removal of R.L.L.	Improved

At the second stage, the diseased lobe is removed, using the Brunn-Shenstone-Janes technic or the Carr "automatic hilar ligature"[11] or individual ligation of hilar structures, and closed drainage is instituted.

Most surgeons believe that the presence of adhesions between the healthy part of the lung and the chest wall reduces the dangers of lobectomy because it reduces the amount of respiratory disturbance during and immediately after operation and prevents the collapse of the remaining lobe, thus limiting the extent of empyema resulting should infection of the pleural cavity occur. Usually, evén though no adhesions are present, the remaining lobe, if healthy, expands and rapidly becomes adherent to the chest wall. Edwards suggests that the occurrence of a total empyema in certain cases where adhesions do not exist between the healthy lobes and the chest wall is secondary to the inhalation of thick mucopus into the bronchi of the remaining lobes, which thereby fail to expand and which gradually become the site of multiple abscesses. He states that a lobe partially held out by adhesions will probably retain enough air within the bronchi either to prevent the ingress of pus expressed from the affected lobe during its operative removal or to enable its expulsion by the act of coughing after the chest has been closed.

Various methods have been used to produce pleural adhesions as a preliminary step to lobectomy. Most of these have required a major first-stage operation and some of them have been quite complicated. Not all of them, by any means, have been effectual. Robinson[12] used iodoform gauze laid against the collapsed healthy lobe, Lilienthal depended upon roughing the healthy visceral and parietal pleura with gauze and then painting the surfaces with tincture of iodine. Sauerbruch[13] introduced a mass of paraffin wax ("plombe") on the parietal pleura. Alexander used "gentle stroking" of the visceral and parietal pleura and Churchill "scrubbed briskly" the parietal pleura with dry gauze. In 1935 Bethune,[14] after a series of animal experiments, recommended the use of "iodized talc powder, blown in with a special blower under thoracoscopic guidance, in a closed pneumothorax." The air introduced before the "pleural poudrage" was immediately aspirated. He said that "It would appear that a technique has been found which can produce, at will, selective pleural symphysis, without fluid formation, as a preliminary to intrathoracic surgical operations such as lobectomy." He had used it four times and ten months after its first clinical use found "no reason to believe that ill effects are to be expected from the presence of this insoluble powder between the two pleural surfaces." Edwards used this method in eight lobectomies for adults and all developed adhesions sufficient to retain the larger part of the residual lobe against the chest wall. Pleural poudrage is, of course, a minor operation in comparison to the first stage of the ordinary two-stage lobectomy when adhesions are created by mechanical irritation of the pleura, but apparently it has not, so far, become popular.

There is still a difference of opinion regarding the performance of lobectomy in one or two stages and in certain other technical points which, no doubt, will be settled with the accumulation of experience. The author has preferred

to complete the lobectomy in a single stage where adhesions held out the healthy lobe or lobes, with certain exceptions. Churchill gives the following indications for choice of operation, with which the author is in accord:

One-stage: "Obliterated pleural space or strong adhesions binding the upper lobe to the chest wall: Children: Middle lobe resections: Severe Hemoptysis; Lobar atelectasis, particularly if upper lobe does not show emphysema; Draining thoracotomy sinus or bronchocutaneous fistula: Cutting into infected lung when freeing adhesions; Bronchial stenosis." Two-stage: "Adults with free pleural space; Febrile patients with active pneumonitis; Patients treated for a prolonged period with artificial pneumothorax and not having an adherent upper lobe: Bilateral cases with free pleural space."

With the accomplishment of reduction in the mortality rate of lobectomy for bronchiectasis to that comparable with other operations for equally serious conditions, attention has been directed to achieving more satisfactory results in other respects and to refinements in technic. The chief cause for unsatisfactory operative results is failure to determine the full extent of the pathology present before operation, and most often, it is involvement of the left upper lobe lingula which is not diagnosed. In a series of 86 cases of bronchiectasis operated upon by Churchill, the disease was limited to a single lobe in only 20 per cent. In 55 of the 86 cases the lower lobe was removed as the major focus of the disease and in 44 of these 55 cases, or 80 per cent, the lingula of the left upper lobe also was resected because of demonstrable bronchiectasis. Belsey[15] found that in 108 cases of bronchiectasis of the left lower lobe operated upon at the Brompton Chest Hospital, London, the lingula was involved in 81, or 75 per cent. Thus it appears that only complete preoperative study will avoid disappointment in operative results.

The anatomy of the lung has been restudied from the standpoint of surgical procedure by Nelson,[16] Churchill and Belsey,[17] Blades and Kent,[18] and others. Nelson suggests that each lung be considered as having four lobes: the upper, middle, dorsal, and lower. The dorsal lobe represents the apical portion of the lower lobe. The lingula of the left upper lobe corresponds in position to the right middle lobe and by some is considered the homologue of the latter. In support of Nelson's suggestion is the demonstration of four major bronchovascular segments in each lung and the not infrequent occurrence of rudimentary fissures subdividing the lung into four lobes. "Each lobe possesses an independent bronchus and blood supply and is separate from the adjacent lobes by a complete or partial fissure, or by an avascular plane of cleavage across which no vascular communications are encountered until the hilum is approached" (Churchill and Belsey). Churchill and Belsey studied the anatomy of the lingula segment of the left upper lobe and describe its bronchus as arising from the inferior aspect of the left upper lobe bronchus 1 to 2 cm. from its origin and running downward and forward to terminate by division into an anterolateral and posteromedial branch. They observed that commonly, when the lingula is involved in bronchiectasis, the posteromedial branch alone is diseased and only rarely are both branches involved. The

lingula, usually, is resected at the time of removal of the lower lobe and through the same incision. If the lingula alone is involved, which rarely occurs, it can be removed through an anterolateral approach similar to that which is used for middle lobe resections. When both lingula and left lower lobes are involved, Churchill and Belsey resect the lingula after the lower lobe has been removed and its stump dealt with. They remove the lingula by individual ligation of its hilar structures. The lingula bronchus or branch bronchus is temporarily occluded, other parts of the lung are inflated, and the delineated lingula is removed by a segmental lobectomy.

Churchill and Belsey point out that "Bronchiectasis is frequently limited to one or more bronchopulmonary segments within a lobe, the remainder of the lobe being normal," and that "it also tends to be primarily multilobar in its distribution." To further quote these authors, "This characteristic of the disease provides a rational basis for proposing the resection of diseased bronchopulmonary segments from several lobes, if necessary, with the conservation of normal lung segments, rather than continuing with the removal of entire lobes as unit structures. This principle finds particular application in early cases of bronchiectasis and those with a bilateral distribution."

Blades and Kent have studied the anatomy of the hilar region of the lungs from the standpoint of individual ligation of the hilar structures of a lobe. They have pointed out the pitfalls, particularly in connection with removal of the right lower lobe where, without sufficient knowledge of the anatomy and identification of structures, the middle lobe bronchus can inadvertently be occluded. They very aptly suggest that preoperative bronchoscopy will give the surgeon some idea of the point of origin of the dorsal lobe bronchus (superior division of lower lobe) and will in a measure afford advance information as to what may be expected of the anatomic characteristics of the bronchus to be attacked.

The first successful lobectomy for bronchiectasis in England was done by A. Tudor Edwards, in April, 1929, by separate ligature of the vessels and the lobar bronchus at the hilum in a boy of 16 years. Clarence Crafoord,[19] of Stockholm, while in this country in the spring of 1939, spoke of having successfully completed four lobectomies by this technic. Recently Blades and Kent reported before the American Association for Thoracic Surgery ten cases in which they employed individual ligation technic for lower-lobe lobectomy with no deaths and an average stay in the hospital of 30.7 days. No particular attempt was made to cover the divided root structures with mediastinal pleura. Six patients developed a postoperative empyema and five of these demonstrable bronchial fistulas. The results of Blades and Kent are excellent, but the author believes that closure of the mediastinal pleura would further reduce the incidence of bronchial fistulas and empyemata. This technic probably will be limited in application to the cases in which there is little if any inflammatory reaction in the tissues of the hilar region but, with early diagnosis, a better understanding of prognosis in bronchiectasis

and the already accomplished lower mortality for lobectomy, more cases suitable for this technic will be referred to the surgeon.

This report is based upon experience gained in operating upon 24 patients with bronchiectasis or bronchiectasis associated with chronic pulmonary abscess covering a period of 11 years beginning in 1929. Eighteen of these cases have been reported elsewhere[20] and all except two have been followed to a recent date. Therapeutic pneumothorax had been attempted or carried out for varying periods of time without success in six patients in this group. No beneficial effect was observed, but it is conceivable that in certain cases it might bring about temporary amelioration of symptoms. Obviously, it is not without danger, for the accidental wounding of the lung infected with the putrid organisms found in bronchiectasis may cause a virulent and highly dangerous form of empyema. Phrenic paralysis had been induced as a separate procedure or preliminary to lobectomy in eight patients. It is doubtful whether it was of value in any of these cases. In a few cases where the movement of the diaphragm was troublesome during lobectomy, the phrenic nerve was temporarily paralyzed by injecting it with novocaine from within the pleural cavity as suggested by Churchill. Hemoptysis, varying in amount from slight blood spitting to large hemorrhage requiring transfusion, was present in 13 patients. Phrenic paralysis was tried in three of these without demonstrable benefit. No patient had a recurrence of bleeding following lobectomy. Chronic disease of the nasal accessory sinuses was present in 12 patients, but in only six of these did it merit serious consideration. Of these patients having extensive chronic nasal accessory sinus disease, five had demonstrable bronchiectasis of more than one lobe, and in the sixth patient, involvement of the opposite lung ultimately was demonstrated. This patient was readmitted to the hospital with bronchopneumonia from which she died three years after removal of a left lower lobe. A partial thoracoplasty was performed as a preliminary step to lobectomy in one case. This patient, who had had severe pulmonary hemorrhages, was operated upon in 1929 and it was thought at that time that a partial collapse of the lower half of the chest might lessen the hazard of removal of a lobe. After amputation of the lower lobe the ligated stump was brought through a piece of rubber dam with a hole in the center and the wound in the chest wall closed about it. This patient had a stormy convalescence but eventually made a good recovery. Subsequently it was necessary to close a bronchocutaneous fistula with a muscle graft.

Bronchoscopic and postural drainage and treatment of nasal accessory sinus disease, when it existed, were carried out in preparation for operation in all patients. Many of them were given small blood transfusions to combat anemia and to build up resistance to infection. Attention was paid to nutrition and to possible avitaminosis. A reduction in the amount of sputum, a loss of its foul odor and a disappearance of fever frequently were the reward for persistence in these measures.

Patients were urged, while in bed, to remain for long periods in that

position in which they would be placed upon the operating table. The patients usually were operated upon in the middle of the day after the morning period of coughing, and in those cases where the amount of sputum was large, bronchoscopic aspiration was done a few hours before operation. Various anesthetic agents, including spinal anesthesia, were tried. For the last 19 cases, avertin supplemented with nitrous oxide oxygen and sometimes a little ether, was used for anesthesia. Usually this was given through an intratracheal tube and with a machine which permitted the use of positive pressure. During operation, the air passages were kept free by aspiration through the intratracheal tube. In order that shock could be combated promptly, should it arise during operation, a cannula was put in a vein after the patient was placed in position on the operating table and an infusion of 5 per cent glucose in normal saline solution was given slowly. An electric heating pad was wrapped about the hands in an effort to prevent a drop in body temperature. Unnecessary exposure of body surface was avoided. These measures seemed to be indicated since there is a loss of heat from the pleural surfaces while the thoracic cavity is open. At the end of operation, or earlier if shock developed, blood plasma or blood previously obtained was introduced through the cannula already in place. Because of the danger of transfusion reaction, in recent years plasma was given in preference to blood, unless considerable hemorrhage occurred during operation. The patient was placed in an oxygen tent upon reaching the ward and was encouraged to cough as soon as consciousness returned. Subsequent infusions of plasma or blood transfusions were given to those patients who lost a large amount of serum through drainage.

If a one-stage procedure was contemplated for removal of a lower lobe, the seventh rib was subperiosteally resected through a posterolateral incision. In some of the earlier cases an intercostal incision was used, a rib being divided above or below, if necessary, but resection of almost the entire length of one rib has proven, in the author's experience, to be more satisfactory.

In this series of 24 completed lobectomies in 23 patients, 14 were done in one stage. The left upper lobe alone was removed in a single stage, in two cases. One patient, a female, had a chronic pulmonary abscess with a bronchocutaneous fistula as well as bronchiectasis of the left upper lobe. The incision was made lateral to the left breast and below it and the breast, together with the underlying pectoral muscle, was reflected medially. The fourth rib, resected 18 months previously, had not regenerated, and the defect in the bony structures of the chest wall had been bridged by a dense membrane. This was incised and adequate exposure obtained without further resection of rib. In the other case, the left upper lobe was removed subsequent to removal of the left lower lobe, thus completing removal of the lung. This patient had a chronic pulmonary abscess in the left uper lobe with a bronchocutaneous fistula and bronchiectasis of the left lower lobe. The abscess had been drained anteriorly a year previously, when segments of the third and fourth ribs were removed from in front. The lower lobe was removed

through a posterolateral incision with resection of the seventh rib. Six months later, the upper lobe was removed through a posterolateral incision with resection of the sixth, fifth, and fourth ribs. An unusual amount of rib was excised at the operation for removal of the upper lobe, partly to secure more exposure and partly to facilitate obliteration of the space which would result after removal of an entire lung. The right lower and the right middle lobes were removed in one patient.

In a planned two-stage procedure for removal of a lower lobe, the eighth rib was subperiosteally resected through a posterolateral incision at the first and the seventh rib at the second operation. In two cases, an anterolateral incision was made for the first stage, but the exposure was poor. In addition to good exposure with a posterolateral approach, there is the advantage that the musculature of the back favors a solid closure and there is less danger of an open pneumothorax should infection of the wound occur. At the first operation in the two-stage procedures an attempt was made to create adhesions over the healthy lobe or lobes by traumatizing the pleural surfaces with dry gauze, adhesions between the diseased lobe and contiguous structures were severed, and the pulmonary ligament was divided. Drainage was not used but occasionally it was necessary to aspirate the serum which accumulated during the first few days. At second stage, the diseased lobe was removed. Ten lobectomies were completed in two stages. One of these, in a previous report, was classified as a one-stage operation. This patient had had three attempts made to mobilize the right lower lobe, and on each occasion, the operation was terminated either because of poor condition of the patient on the operating table or penetration of the diaphragm. Lobectomy finally was successfully completed more than two years after the last previous operation. In only one case was the middle lobe alone removed. The approach was through an anterolateral incision with resection of the sixth rib at the first and of the fifth rib at the second stage. In one patient, who had a complete transposition of viscera, the right lower and the lingula of the right upper lobe were removed.

Tourniquets were used in most cases, the cords constricting the hilum during amputation of the lobe and while the stump was being secured with sutures. In two cases the Carr "automatic hilar ligature" was used, leaving the ligature cord and the small metal box in the thorax until later when the residual cavity was drained. In both of these a chronic pulmonary abscess with a bronchocutaneous fistula was present as well as bronchiectasis. The adhesions in these cases were dense and their separation time consuming. After the lobe was freed, the condition of the patient was critical and the need for speed urgent. Both patients made complete recoveries. In two cases, both left lower lobe lobectomies and both operated upon in September, 1939, individual ligation of hilar structures was successfully accomplished. In the first of these, lobectomy had been attempted almost a year previously with termination of the operation after division of adhesions because of the development of shock. At the subsequent operation, the adhesions were not nearly

so dense. The veins were ligated first, then the artery, and finally, two bronchi. The bronchi probably were the dorsal lobe bronchus and the main trunk of the lower lobe. In this case, it was not feasible to cover the bronchial stumps and vessels with pleura. An empyema developed, but was not putrid. It was drained eleven days after lobectomy. Similar technic was used for the second patient, except that a ligature was placed proximal to the dorsal lobe bronchus and that it was possible to close the mediastinal pleura over the stumps of the vessels and the bronchus. This patient did not develop an empyema. In both cases, chromic catgut was used for ligation of the vessels and the bronchi were occluded with single ligatures of silk.

The successful use of sulfanilamide to prevent and combat infections in traumatic wounds and in surgical wounds following operations upon infected viscera suggested that its use might be of value in thoracic surgery. Accordingly, it was used in five cases of the group reported in this paper.

In all cases in this series, except the first, drainage was provided after lobectomy by means of catheter brought out through a stab wound and the operative incision was completely closed. A negative pressure of about 4 cm. of water was maintained. If the bronchus opened or there was evidence of a putrid empyema, open drainage of the residual space was promptly established and the cavity packed with gauze moistened with 1–1000 solution of neutral acriflavine. Healing occurred without the development of an empyema in four patients. Three of these had been given sulfanilamide by mouth, beginning the day preceding operation and, in addition, sulfanilamide crystals were placed in the pleural cavity before the wound was closed. In one of these, in addition to using sulfanilamide, the lobectomy was done by individual ligation technic. Sulfanilamide was employed in a total of five patients, two of which developed small, non-putrid empyema. In one of these, the empyema was not evident until late and open drainage was done on the fiftieth day.

There were four operative deaths in 24 completed lobectomies on 23 patients. Three of these occurred in the first five patients subjected to operation. One death occurred on the twenty-fifth postoperative day from cerebral abscess and meningitis; one eight hours after operation from mediastinal emphysema; one on the tenth day from hemorrhage into the residual space left by the lobectomy; and one from uremia following a blood-transfusion reaction. The patient dying from mediastinal emphysema did not have a tension pneumothorax. The site of operation was carefully explored after death but permission for a complete autopsy could not be obtained. The cause and source of the leakage of air into the mediastinal tissues was not determined. Anesthesia had been given through an intratracheal tube and positive pressure had been used from time to time to inflate the lung. Postmortem examination through the wound in the patient dying from hemorrhage showed a small residual empyema cavity with an erosion of a pulmonary vessel in its wall. There had been a virulent infection and open drainage should have been instituted. In the patient dying from uremia, the condition of the thorax at autopsy was satisfactory. This was a typical case of posttransfusion anuria. In addition

to the above deaths, one patient, not included under completed lobectomies, died following the first stage of a planned two-stage procedure. The lobe was not removed. This patient developed bronchopneumonia, hemolytic streptococcus empyema, and pericarditis. There has been but one death, that of the patient dying from uremia following blood transfusion reaction, in the last 19 completed lobectomies on 18 patients. This group includes a left lower and a left upper lobe lobectomy in the same patient, an interval of six months elapsing between the two operations. Of the patients surviving operation, 13 are classified as cured, three as greatly improved, two as improved, and one as dead. This death occurred three years after lobectomy from bronchopneumonia. Two of those classified as greatly improved are normally active but have slight cylindrical dilations of a few bronchi in another lobe and still have a slight cough and some expectoration. One classified as greatly improved has nasal accessory sinus disease, a slight cough, and some expectoration at times, but no demonstrable bronchiectasis and is normally active. Two classified as improved are cases of bilateral lower-lobe bronchiectasis with nasal accessory sinus disease who have had one lower lobe removed and are awaiting removal of the other.

In brief, it may be stated that the conservative treatment of bronchiectasis is not curative and that lobectomy in selected cases gives excellent results. Experience has shown that the risk of this operation in the earlier stages of the disease is far less than in the late stages and that young individuals tolerate this type of surgery better than those who are older. The involvement of more than one lobe is not necessarily a contraindication to radical surgery but satisfactory surgical results can be achieved only by removal of all the diseased segments of pulmonary tissue. The application of the principles of segmental pneumonectomy, particularly with reference to the lower lobes, suggested by Churchill, will, if necessary, permit the resection of diseased bronchopulmonary segments from several lobes with the conservation of normal pulmonary tissue. The mortality of lobectomy has been lowered to the extent that it now conforms with the mortality of other operations for equally serious conditions. Refinements in technic have been developed which have reduced the occurrence of postoperative bronchial fistulas and empyemas and have shortened the period of hospitalization. The use of sulfanilamide or a similar chemical agent offers some promise of further reducing the incidence of postoperative pleural infection.

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A REVIEW OF THE RESULTS IN THE TREATMENT OF 100 CONSECUTIVE CASES OF BRAIN ABSCESS*

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THE three problems to be solved in an attack upon a brain abscess are when, where, and how to operate. Increased clinical experience with this lesion plus the use of ventriculography and other technical aids have made diagnosis and localization relatively simple. But determination of the proper time to attack an abscess and selection of the appropriate procedures for drainage require the nicest surgical judgment. Only too often the initial feeling of satisfaction in the surgeon's mind over an apparently successful outcome is abruptly shattered by the appearance of some unexpected and possibly disastrous complication.

A study of the records of 100 consecutive cases of subcortical abscess of the brain seen in the Neurosurgical Clinic in the University Hospital form the basis of this report. Little stress will be laid upon etiology, types of organism, or methods of diagnosis and localization unless these factors bear directly upon the surgical results. These cases were seen between January 1, 1926, and January 1, 1940. An adequate follow-up over a number of years is, therefore, available in many of the survivors.

Of these 100 cases, 47 recovered and 53 died. Ten patients died before any operative procedure could be instituted, seven with 12 hours of admission to the hospital, three within 48 hours. These last three cases were all seen prior to 1930, when the value of ventriculography in the localization of the lesion was less clearly recognized. Nine patients had meningitis, as indicated by lumbar-puncture findings prior to operative intervention. Surgical attack upon the abscess was undertaken as a last resort. In six evacuation was successful and in three the lesion was found at autopsy. All of these fatalities occurred prior to 1937. The use of the appropriate sulfa-derivatives, had they been available, might well have saved half of this group. In nine patients multiple abscesses were present. One or more of these lesions were adequately drained in seven; in two the evacuation of pus was unsatisfactory. In seven of these patients the abscesses were so widely separated, parietal and cerebellar, front and occipital, right and left frontal, frontal and parietal (3), right and left cerebellar, that formation of a second adjacent abscess following bad drainage of the original cavity seemed impossible. In two cases, one of multiple frontal and another of multiple cerebellar abscess, it is conceivable that inadequate drainage of the primary abscess may have resulted in a

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spread of infection and the formation of a second cavity. The 25 remaining fatal cases are grouped together under the general heading of "bad operative technic" as the cause for the mortality. It is from an analysis of this group that we hope to find the errors in the operative methods. In all these cases the surgeon had every opportunity to save the patient, in none was any great emergency present, and the operative procedure was planned and carried out deliberately in the manner he considered to be the best adapted to the problem at hand.

What are the particular problems connected with the surgery of brain abscess? The brain lies within the close confines of the skull; therefore, an increase in intracranial pressure may appear before or after operation and must be promptly and adequately relieved. The entire brain is surrounded by the subarachnoid space containing the cerebrospinal fluid. Furthermore, those areas of the brain overlying the abscess must be traversed by the drainage tract. Again, the abscess may tend to burrow down toward and rupture into the ventricle. Prevention of infection either before or after drainage, is, therefore, a problem. Lastly, since certain cortical areas of the brain overlying many an abscess have important functions, evacuation of the cavity with a minimum of destruction of cortex is essential if recovery without motor or sensory loss is to be accomplished.

All neurosurgeons agree that if possible an abscess should not be attacked until encapsulation has occurred. Three to four weeks from the onset of symptoms should, therefore, be permitted to elapse before the cavity is evacuated, although that certain abscesses will never become encapsulated is frankly conceded. The use of the sulfa-derivatives has largely overcome the dangers of infection. The proper way to handle the problem of increased intracranial pressure and the amount of destruction of adjacent brain tissue and overlying cortical areas justifiable to secure adequate drainage are the outstanding points at issue. If intracranial pressure is to be relieved and adequate open drainage of the abscess under direct vision assured, a large opening must be made in the bone and overlying cortical areas and brain tissue adjacent to the abscess destroyed. This may well result in impairment of function and the large scarred area in the brain may subsequently cause convulsive attacks. But if only a small trephine opening is made, the abscess simply tapped and drained to minimize injury to the surrounding brain, the drainage may be inadequate or the intracranial pressure persist with fatal results.

Curiously enough in this series, 48 cases were handled by a closed drainage with 32 recoveries and 16 deaths, and a 33 per cent mortality, while 18 cases had a larger opening made for open drainage with 12 recoveries and 6 deaths, a 33 per cent mortality. The advocates of open drainage may well claim that had more of the 48 patients treated by closed drainage been widely opened the mortality might have been lower. In this group of 16 fatal cases, in six following tap and difficulty in insertion of the drainage tube, autopsy showed a marked cerebritis about the abscess. In every instance

the abscess was deep-seated, small, and encapsulated. Five of these lesions were cerebral, one right frontal, one right temporal, one left frontal, two in the left temporal region, and one in the cerebellum. Admittedly these small deep-seated encapsulated lesions are hard to handle by any method. The left frontal and temporal areas are too important physiologically to justify free excision. A cerebellar abscess is difficult to drain widely because of the heavy muscles attached to the occipital bone. The two lesions in the right cerebral hemisphere might have been saved by section of the cortex. In seven patients with relatively superficial lesions apparently adequately drained by simple trephine, death was attributed to increased intracranial pressure, on clinical evidence in four instances and at autopsy in three. Two of the three autopsied cases had cerebral, one a cerebellar abscess. No meningitis was noted nor was there an unusual amount of cerebritis present about the lesion. The striking feature in each case was a pressure cone about the cerebellar tonsils. Admittedly these seven cases might have been saved by a wider opening in the bone with cortical section and more ample drainage or by the addition of a contralateral decompression to the operative procedure for the purpose of reducing intracranial pressure. In three instances postoperative meningitis was the cause of death, due in one case to rupture of the abscess into the adjacent ventricle, and in two to infection presumably of the subarachnoid spaces about the drainage tract.

The six deaths following open drainage with a wide craniectomy and cortical incision, all in cerebral lesions, were caused in two instances by the development of uncontrollable brain fungus, meningitis in three patients, and inanition in a single case after a three-months struggle.

The three fatalities consequent upon an attempt at complete enucleation of the abscess were due to meningitis consequent upon its rupture during removal in two instances. In the third, a large, chronic, encapsulated abscess of the left middle fossa, the middle cerebral vessel was adherent to the lesion and was torn during its removal. The wound healed well and no meningitis could be demonstrated, but the patient died about two months later as a result of the vascular injury.

In the 47 cases that recovered, 24 were tapped and a drain inserted; eight were tapped without permanent drainage. In 12 wide drainage by craniectomy and cortical section was instituted. In three, complete enucleation of the abscess was successfully accomplished.

This brief review of the surgical methods employed suggests definitely that the tendency in this clinic has been toward conservatism with regard to the destruction of brain areas overlying or adjacent to the abscess in establishing drainage.

In support of this conservatism the follow-up statistics furnish important information. Forty-one cases have been followed, four are lost, and two are known to be dead from outside causes. Among 29 cases treated conservatively by tap or tap and drain, 22 have completely recovered and, as far as our information goes, have returned to their original economic

status. Seven patients have neurologic sequelae, weakness, or convulsive attacks, which have seriously crippled their wage-earning power. In 12 patients in whom deliberately or through necessity a craniectomy was performed with cortical destruction for more adequate drainage, but three have made complete recoveries, and nine have sequelae of such a nature that they are seriously handicapped economically.

As a result of this study the conclusion seems unavoidable that conservative treatment, tap, or tap and drainage through a small trephine in the bone produces the better result in the long run in the treatment of brain abscess. The use of wide open drainage has not, in this series at least, lowered the operative mortality in comparison with more conservative methods. A careful follow-up in this group of cases indicates that a much higher proportion of patients are returned to their former occupation following the use of simpler procedures for instituting drainage than when a more radical technic involving wide destruction of brain tissue is employed.

We feel, therefore, that the initial attack upon a brain abscess should be carried out through a small trephine opening with the insertion of a drainage tube. This will result in a cure in many instances. If this technic does not produce satisfactory results a resort must be had to more radical methods involving the patient in a greater hazard from serious neurologic sequelae.

TABLE I

100 CASES BRAIN ABSCESS VERIFIED

53 Died	Causes	47 Recovered
Percentage	Mortality	53 Cases
10 cases died unoperated		19%
9 cases died meningitis preoperatively		17%
9 cases died multiple abscesses		17%
25 cases died "bad surgical management"		47%

TABLE II

OPERATIVE STATISTICS

	Died	Recovered	Percentage
Closed drainage	16	32	66
Open drainage	6	12	66
Enucleation	3	3	50
Totals	25	47	

TABLE III

OPERATIVE TECHNIC

(47 RECOVERIES)

Tap and drain	24
Tap only	8
Wide drainage	12
Enucleated	3
Total	47

TABLE IV

CAUSES OF DEATH AFTER CLOSED DRAINAGE

Increased pressure	7
Cerebritis	6
Meningitis	3
Total	16

TABLE V

CAUSES OF DEATH AFTER OPEN DRAINAGE

Meningitis	5
Cerebritis	3
Vascular injury	1
Total	9

TABLE VI

CAUSES OF DEATH FOLLOWING "BAD" OPERATIVE TECHNIC

(25 CASES)

Cerebritis	9
Meningitis	8
Increased pressure	7
Vascular injury	1

TABLE VII

FOLLOW-UP STATISTICS (COMPARISON OF RESULTS IN "CLOSED" AND "OPEN" TECHNIC)

41 Cases Followed		2 Dead	4 Lost	
"Closed" Technic	Tapped, or tapped and drained			29
	Complete recoveries			22
	Complications			7
"Open" Technic	Wide drainage			12
	Complete recoveries			3
	Complications			9

ANNUAL ADDRESS FOR 1941

THE CONTROL OF SOMATIC PAIN*

WILLIAM BATES, M.D.

PHILADELPHIA, PA.

THE relief of pain has always been one of the chief functions of a doctor. Pain of visceral origin has been the reason for the existence of the surgeon. However, the differentiation between visceral and somatic pain has not always been an easy one to make.

The types of somatic pain to which we have devoted most of our attention are those neuralgias which are segmental in distribution, and which are associated with tenderness. Pain due to segmental neuralgias, by virtue of its variation in location, character, and intensity, may simulate the pain of almost any form of visceral disease.

Evaluation of the pain in these patients has been made more definite by various factors: (1) The finding of tender points at the emergence of the posterior, lateral, or anterior cutaneous branches of the intercostal nerves. (2) The establishment of tenderness in segments corresponding to definite dermatomes which are associated with the pain. (3) The fact that segmental tenderness is rarely initiated by the pain of visceral stimuli, unless a widespread peritonitis is present. (4) The finding of reduced skin-surface temperature which is often associated with the pain and tenderness. (5) Following nerve block, pain and associated tenderness should disappear simultaneously.

In many series of operations, the failures reported are chiefly in patients whose cardinal complaint was pain. This I believe is not due to incomplete or incompetent surgery, but failure in diagnosing the source of the pain.

In 1926, when a former member of this Academy, the late Dr. J. Berton Carnett, first wrote about intercostal neuralgia of the abdominal wall, he was pointing toward this same fact. His work met with great differences of opinion, but his basic reasoning has been substantiated many times. It is very true that to follow his teachings of that time might have led to some serious mistakes. On the other hand, it would have prevented many useless operations.

In his teachings, he advocated differentiating visceral from somatic pain. He described his tests to accomplish this. He gave a series of causes for the existence of somatic, or, as he called it, intercostal pain, and lastly, he attempted to outline methods of treatment for this condition. This latter fact was unfortunate, and was forced upon him and his staff rather prematurely. Having brought the picture of surface pain to the attention of practitioners, they demanded some method of treatment.

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Among the recommendations for treatment was, of course, the classic one of "eliminate the cause." This we tried to do, but in many cases we found that the pain persisted after the apparent cause had been removed. Something more was needed in those patients whose toxic foci were eliminated and postural defects corrected, with no relief of their pain. At this stage of development of our clinical problem an attempt was made in cases of local pain and tenderness to inject with novocain. As one might expect, most of the cases got temporary relief. However, quite a few got prolonged relief, encouraging us to repeat the injections. Where prolonged relief was obtained by such a simple procedure, we felt that the basic cause or causes were probably corrected in conjunction with the infiltration.

About this time—in 1931 to be exact—one of the men on Doctor Carnett's service at the Graduate Hospital, Dr. B. D. Judovich, did some work on the pitcher plant—Sarracenia purpurea. He prepared an aqueous solution derived from this plant, and observed that it was of value in relieving pain of neuralgic origin. It was believed at that time that its properties were due to the presence of amines. It was noted that the preparation had an action upon sensory nerves, relieving neuralgic pain without producing changes in skin sensation, and having no effect upon motor nerves.

Doctor Carnett suggested that Doctor Judovich be allowed to work in what we called the Intercostal Neuralgia Clinic. Heretofore, we had been trying to determine the cause of the pain, and treat by means of eliminating focal infections, correction of postural defects, and giving Goldthwaite exercises and the occasional injection of novocain solution in some of the more persistent localized pain and tenderness cases. From then on, even though we did not know the actual active principle of the solution we called Sarapin, it was administered to numerous patients suffering with pain of somatic origin.

Controls of novocain, saline, and water were used, and the results recorded. The key numbers of these various ampules were changed several times, and on analysis in each series it was found that Sarapin produced prolonged relief in contrast to fleeting or negative results with the other solutions. In a number of instances, patients who had been injected with novocain with only a short period of relief of pain obtained prolonged relief by a subsequent injection of the pitcher-plant distillate. Toxicity tests revealed that it was harmless. It caused no tissue coagulation or sclerosis. This action was unusual and difficult to substantiate. We were treating a subjective symptom, and laboratory animals gave no actual data except in reference to toxicity. Routine pharmacologic tests in three different laboratories cast no light upon the problem.

Following injection of peripheral nerves, there were no cases of motor weakness or loss of touch, pressure, pinprick, or temperature sensibility. In some cases, one infiltration of the distillate was sufficient to provide permanent relief of pain, even though of long duration. The psychic factor of relief in this type of pain largely has been ruled out by establishing the presence of tenderness in the dermatomes of the painful segments.

In intercostal neuralgia, paravertebral injection of the nerve trunks involved results in an immediate intensification of the pain, followed during the next 30 minutes by a gradual contraction of the hyperalgesic area in the dermatome supplied by the nerve. In a large number of cases it is followed by complete relief of neuralgia.

Because of the segmental overlap, the effect of the distillate upon sensations other than neuralgia cannot be evaluated. If, in a case of sciatic neuralgia, the region of the sacro-sciatic foramen is infiltrated with the pitcher-plant distillate, there is relief of the neuralgic pain as well as of tenderness along the nerve trunk. This is similar to results from infiltration of novocain, but in contrast to the action of novocain there is no numbness, no loss of sensibility, and no motor weakness. Reflexes remain unchanged.

In 1939 investigation was made by Drs. Winifred Stewart and Joseph Hughes, using the cathode-ray oscillograph, to determine the action of the pitcher-plant distillate upon the nerve impulse. Their studies indicated that the C-wave impulse, which is supposedly transmitted by way of the small unmyelinated fibers, was depressed in each instance. A report of these findings was made in 1940 by Stewart, Hughes, and Judovich in which they stated that "The ability of the pitcher-plant distillate to abolish neuralgic pain which, like fascial pain, is aching, poorly localized, and frequently associated with nausea and sweating, and at the same time, to leave pinprick as well as other forms of sensibility unaffected, led us to inquire further into its physiological action, and to attempt to determine its active principle.

"The effect of pitcher-plant distillate on the action potentials of the saphenous nerve of the cat was observed. The nerve was mounted in a nerve chamber in a gas mixture of 5 per cent carbon dioxide and 95 per cent oxygen. The temperature was maintained at 37.5 to 38° C. The nerve was so mounted that it could be bathed in the solution to be studied. The pitcher-plant distillate was adjusted to a pH of 7.4. The action potentials were recorded on a cathode-ray oscillograph.

"After five minutes' immersion in pitcher-plant distillate, the maximal A spike was somewhat reduced while the C fiber potentials were obliterated."

For the first time it appeared that we had some definite objective proof that the solution we had been using should relieve pain. It became vitally important for us to know the chemical constituents of this solution. The first report we received on the analysis showed that the crystalline compound which was isolated was ammonium chloride. All three gave the same result. Clinically, injection of Sarapin and of solution of ammonium produced the same prolonged relief.

On checking the chemical process by which the distillate was obtained, Judovich found that the end-result should have been a sulfate and not a chloride. Accordingly, further experiments were carried out with a solution of known ammonium sulfate. Both clinically and experimentally the results were parallel to those found with the solution of ammonium chloride.

From this it was readily deducted that as the ammonium radical was the only constant, it was the active principle responsible for our results.

These observations on actual patients led us to believe that neuralgic pain and pinprick are mediated by separate fiber groups. Clinically, however, we have been unable to relieve pain of visceral or sympathetic origin by the injection of ammonium salts. In two instances the first and second lumbar sympathetic ganglia were injected in a patient with vascular occlusion of the lower extremity. There was no change in the skin-surface temperature of the leg following infiltration of these points with ammonium salts. The needles were left in place and at the end of 20 minutes 2 per cent novocain solution was injected. Within two minutes definite rise in skin temperature was obtained. All cases benefited by these injections were cases of pain of somatic origin associated with tenderness.

On occasion, the infiltration of the pitcher-plant distillate and the ammonium salts have been of value in differential diagnosis, neither of these substances having any effect upon vascular pain or pain of visceral origin. Just what the difference is, in the unmyelinated C fiber of peripheral distribution which responds to these injections, and the unmyelinated C fibers of the sympathetic nerves and their ganglia which do not respond to these injections, we do not know. Experiments are being conducted by Doctors Stewart and Hughes upon sympathetic fibers to determine the effect of ammonium salts upon the nerve impulse in this type of tissue.

This has no doubt been a prolonged explanation of a simple procedure, but the clinical results have been so satisfactory to some of us that the confirmation of our ten years of clinical observations by laboratory proof made it seem worth while to render this report.

Our work in attempting to control pain is by no means complete, as the same solutions are now being tested intraspinally for intractable pain. To date the results in certain cases have been most promising. I am not recommending regional infiltration as a cure-all for aches and pains, but without it, we would have been unable to relieve many patients of their persistent pain.

ANNUAL ADDRESS FOR 1942

CHOLECYSTITIS-CHOLELITHIASIS: A REVIEW*

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There have been so many papers and so much discussion about war wounds, it seemed to me a discussion of a totally unrelated subject might be a relief. I also feel there is still a war on at the home front which should not be neglected, and upon those of us who remain at home there falls the duty of pressing on relentlessly against our old enemies.

There are many problems, diversity of opinions, and confusion in the minds of some of us about certain features of cholecystitis and cholelithiasis. As this subject has not been discussed recently by members of the Academy, I have felt it might be of interest to review it.

Today there is a growing tendency to view the gastro-intestinal tract including the liver and bile passages and the pancreas with its ducts as a whole. To think of it as being made up of many unrelated parts and pigeon holing each section in a separate compartment is short-sighted and does not give a proper perspective of gastro-intestinal diseases.

From the stomach to the anal canal there are five sphincters, all of which are dependent on each other for the normal functioning of the gastro-intestinal tract. These sphincters are the cardiac, pyloric, sphincter of Oddi, ileocecal, and anal. It would indeed be surprising if they worked independently of each other. We are amassing evidence constantly to demonstrate an interrelationship. The reflex connection between distention of the fasting stomach and the desire to defecate is a common experience needing no particular elaboration. Much work has been done on the gastrocolic and "feeding reflex." This reflex is demonstrated by putting food in the empty stomach and observing increased contraction of the colon. The action is reversible. Irritation of the ileocecal region or distention of the colon will bring about spasm of the pylorus and delay in emptying the stomach, and sometimes, if continued, vomiting. Smith and Miller[1] produced increased pyloric tonicity and hyperperistalsis of the stomach by irritating the cecum or appendix with croton oil.

Hedblom and Cannon[2] and Pearcy and Van Viere[3] have also demonstrated the slowing of gastric discharge when the colon was irritated. Goldman and Ivy[4] have demonstrated in dogs an inhibition of bile flow following distention of the colon. This effect has been proven to be of reflex origin.

The relationship between duodenal or gastric ulcer and pylorospasm is well recognized. Although there is no definite experimental evidence to support the view that there is a relationship between the functioning of the

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pylorus and the sphincter of Oddi, I think it would be more difficult to disbelieve, particularly in the light of the fact that in many cases of gallbladder disease the first symptoms and in some the only symptoms are "indigestion," and symptoms of pylorospasm and gastric dysfunction. The relationship between the sphincter of Oddi and the ileocecal sphincter might be difficult to demonstrate experimentally. I do not know of its having been done. However, as will be cited later, the relationship between disease of the appendix and gallbladder disease is, I believe, unequivocal. Whether it be reflex or infectious or both has not been proven. It is logical to believe these reflexes along the gastro-intestinal tract work in much the same way as the block signals on a railroad. When the various segments are ready to receive the contents for their particular action, the go signal is given. The reverse must also be true that when a segment is not ready to function the stop signal will be sent back. These signals certainly must control the pouring out of such large quantities of important digestive juices as bile and pancreatic juice. The stop signal when there is dysfunction anywhere along the line to a patient is "indigestion." Deaver in his inimitable way called the stomach the "loud speaker" of the gastro-intestinal tract.

The sphincter of Oddi is a very finely adjusted mechanism. Stimulation of the vagus produces both spasm of the sphincter of Oddi and gallbladder contractions. Sympathetic splanchnic stimulation produces relaxation of the gallbladder wall and contraction of the sphincter of Oddi. In anaphylactic shock the gallbladder and sphincter of Oddi both contract. It is also interesting to note that when bile is discharged normally it is discharged in spurts from the common duct, but when the gallbladder has been removed the bile passages dilate and the bile is discharged from the common duct in a continuous flow.

There are those who might argue that all these reflexes are hormonal in origin, but workers in this field have declared that some of these reactions are so rapid it would be hard to explain them on any other basis than nervous.

The contractions of the gallbladder are brought about by a hormone cholecystokinin which is manufactured by the mucous membrane of the upper intestinal tract. The manufacture of this hormone is stimulated by the presence of fat, and, to a lesser degree, protein in the duodenum. It is well known pituitrin will also stimulate contraction of the gallbladder. Atropin inhibits emptying of the gallbladder but is ineffectual against cholecystokinin. Inflammation of the gallbladder definitely delays its emptying and inhibits its other normal function, that of concentration of the bile.

The nerve supply to the gallbladder and bile passages comes through the plexus on the cystic artery which is derived from the hepatic plexus which in turn comes from branches of the solar plexus and left and right pneumogastric nerves.

These few points on anatomy and physiology have been brought to your attention because of the direct bearing they have on questions which will arise later in our discussion.

Etiology. No discussion of the etiology of gallbladder disease and gallstones could be opened in a more simulating way than to describe the dramatic and unique manner in which the late John B. Deaver drove home his concept to his students. After dipping his gallbladder spoon into the gallbladder, he would hold it aloft over a white enamel basin held by his assistant. Cautioning all to listen attentively he would drop a stone into the basin and ask his assistant, "What was that?" Always there was the expected reply, "That, sir, was a monument erected to the memory of the bacteria which lie within." Sir Berkley Moynihan, holding the same view, described it in his picturesque way when he declared a stone to be the sarcophagus of the bacteria within.

Unfortunately, space does not permit a full discussion of the work that has been done on this very interesting subject. Judd,[5] Branch,[6] Nelson and Rehfuss,[7] and many others starting with John Hunter and including such names as Gilbert, Girode, Cushing, Italia, Rosenow, Blalock, Brown, and Burden have attempted to prove the infectious origin of gallbladder disease. Elkeles and Mirizzi[20] made cultures of the gallbladder wall and bile, common duct bile, and duodenal contents at the time of operation on human subjects and drew some interesting conclusions. Today I think no one believes infection plays the whole rôle and few that it plays the primary rôle in the etiology.

Metabolic changes, particularly altered cholesterol metabolism, are believed by some to play a large part in stone formation. Interesting work along this line has been done by Potter[8] and Aronsohn and Andrews.[9] Wolfer,[10] Popper,[11] Schiller,[12] and Bisgard and Baker[13] have studied the problem from the standpoint of pancreatic juice reflux. Obstruction with stasis of the bile passages as a contributing cause has been shown by Cole, Novak and Hughes[14] and Phemister, Aronsohn and Pepinsky.[15] The relationship of pregnancy has been studied by Gerdes and Boyden,[16] Courvoisier,[17] Kalk and Schoendube,[18] and Potter.[19]

When we analyze all the clinical and experimental data at hand, we must admit a confusion and a great uncertainty. Obviously there is no one etiologic factor that is responsible only in the production of all cases of cholecystitis and cholelithiasis. Again because of the uncommon occurrence of cholecystitis without stone, we ask the question—Are cholecystitis and cholelithiasis unrelated diseases? If they are related, which one comes first, and how is each dependent upon the other?

It seems that infection in the absence of damage to the gallbladder wall, normal concentration of bile salts and bile cholesterol content, and absence of stasis is not and cannot be the etiologic factor in the genesis of either cholecystitis or cholelithiasis.

It may be further concluded that in the presence of stasis or obstruction alteration in the composition of the bile is practically certain to follow, this in turn damaging the gallbladder wall or causing the precipitation of stones, or both.

It may be here, then, that infection plays its rôle in the further destruction of the gallbladder wall and the bacteria acting as a nucleus around which precipitation of bile constituents occurs or such precipitation may form lamina around already existing stones. When obstruction to the cystic duct becomes complete, then calcium carbonate stones form or calcium carbonate lamina surround preexisting stones of other chemical nature.

The manner in which stones occur is still debatable. Alterations in the operation of the very complicated mechanism of the sphincter of Oddi undoubtedly plays a leading rôle. Whether this alteration be nervous, hormonal, congenital, or dependent upon alterations in the normal functioning of other sphincters of the alimentary canal cannot be definitely stated. Probably all play a part in some manner and under certain fortuitous circumstances yet to be described. Changes in the tonicity of the sphincter of Oddi in combination with anatomic peculiarities in which the biliary and pancreatic systems are communicating systems must be considered because of changes resultant upon reflux of pancreatic juice.

The alterations in the chemical composition of bile must be further studied relating changes in composition of the bile as a result of differences in diet, metabolism, hepatic function, etc., which alter the chemistry of the bile before it reaches the gallbladder and is subject to the changes in it that may occur there. In this connection emphasis may be placed on the frequency of stones found in hemolytic icterus and pernicious anemia.

Finally, the manner in which bacteria reach the gallbladder and bile is still an unsolved problem upon which much depends in properly understanding these conditions and bearing a direct relationship to prophylaxis and treatment. John Deaver used to say the treatment of gallbladder disease was only half finished with measures directed to the gallbladder and bile passages only. It was completed by appendectomy. He felt with many others that the appendix was the primary offender. This concept has been supported and strengthened by many who have observed the frequency with which evidence of disease of the appendix is associated with gallbladder disease. McCarty[21] of the Mayo Clinic found evidence of disease in the appendix in 69 per cent of patients with cholecystitis who had had cholecystectomy. The way in which chronic appendicitis may be a factor in the production of gallbladder disease seems at the present time to be dependent upon reflex irritation that disturbs the normal mechanism of the stomach, duodenum, and the mechanism of the gallbladder and bile passages. Bacteria may conceivably reach the gallbladder and bile passages by ascending the common duct from the duodenum, by way of the portal vein to the liver, and thence to the bile and gallbladder or through the hepatic lymph channels to the gallbladder wall, or through the adjacent lymphatics and the venous blood supply of the gallbladder. Lastly by way of the arterial blood supply.

The undamaged sphincter of Oddi offers a very strong barrier to ascending infection from the duodenum; as the duodenum itself is usually sterile under normal conditions this method of invasion must be very uncommon. However,

if there be duodenitis or ulcer present in the duodenum or stomach the biliary mechanism may be so altered that bacteria may ascend the common duct.

Infection through the portal vein and hepatic lymphatics to the wall of the gallbladder is a very likely route. Bartlett, Crile, and Graham[22] have shown lymphatic connections between the liver and gallbladder wall. The liver is a great filter of bacteria derived mostly from the intestine. It is not hard to understand how bacteria may reach the gallbladder wall by this route and such a possibility has been supported further by the experimental evidence of Peterman, Priest and Graham.[23]

Infection by way of the arterial blood supply has been dealt with in the discussion of the infectious origin of cholecystitis and the experimental work done by Rosenau and Nelson and Rehfuss and others.

Here is a fertile field, then, for further work so that our understanding of this important subject may be improved.

Diagnosis and Differential Diagnosis. There is nothing to be gained by reviewing diagnosis and differential diagnosis with this audience excepting to bring before you the differential diagnosis between certain heart conditions and gallbladder disease and the relationship between the two.

Layne and Bergh[24] studied 30 patients who had previously had choledochotomy and intubation of the common bile duct. They connected the tube in the choledochus with an infusion flask. A Murphy drip bulb and glass manometer were included in the system so that the rate of flow and pressure could be observed. Physiologic salt solution was used to distend the common duct under pressures ranging from 50 to 100 centimeters of water for from 30 to 300 seconds. In several patients a balloon was placed in the duodenum so that intraduodenal pressures could be recorded at the same time.

Sudden distention of the common duct caused deep epigastric or right upper quadrant pain in all but one patient. Gradual distention did not induce pain. In 18 of the 29 the pain remained localized. In the remaining 11 the pain also radiated to the interscapular or right subscapular region. The pain subsided after the pressure was reduced unless the sphincter of Oddi was in spasm. The pain of spasm was similar to, but more intense, than the pain of distention. These patients declared the pain to be similar to their preoperative pain. Associated with the pain there was marked rigidity of the abdominal wall more marked in the right upper quadrant. Vomiting occurred in two patients; a third vomited after the experiment was over. Five patients complained of nausea, and many described a feeling of fullness of the epigastrium. Six patients belched.

It is fairly well established that visceral pain does exist. The stimuli that are known definitely to elicit visceral pain are produced by spasm or distention, the pain of spasm being greater than that induced by distention. Nervous impulses from the region of the biliary tract travel by way of the great splanchnics to the central nervous system through the spinal ganglia

of the sixth to ninth thoracic segments of the spinal cord. If the diaphragm be irritated, then the pain will pass through the dorsal ganglion to the spinal cord of the fourth cervical segment.

Ogilvie[25] distended the gallbladder in patients who had cholecystostomy and found it produced pain vaguely localized in the region of the gallbladder, once to the xiphoid process of the sternum and once to the left hypochondrium spreading around the costal margin to the left scapular region.

Ravdin, Royster, and Sanders[26] state, "the occurrence of gall stone disease with cardiac syndromes is now so well recognized that it would seem that further evidence of this relationship is hardly necessary." They quote Fitzhugh and Wolferth who pointed out, "a patient with gall stone disease may present such a convincing array of cardiac symptoms that the internist may wrongly condemn the sufferer to a 'life sentence' of cardiac servitude. In addition to this problem of mimicry there is a growing conviction among internists and surgeons alike that chronic disease of the gallbladder may either irritate or aggravate actual heart disease, especially so-called coronary heart disease. It seems clear from the evidence, however, that not only may gallbladder disease injure the myocardium but that the process at least to a certain extent, is reversible."

Ravdin, *et al.*, report two cases with serious heart disease greatly improved after gallbladder surgery. They further report two cases with angina pectoris in whom cholecystectomy and drainage of the common duct was done. Pressure studies on the common duct were made on these two cases on the eighth postoperative day. In both instances the preoperative anginal type of pain was caused to recur by distending the common bile duct. These pains were precordial, radiating down the ulnar side of the left arm, or into the left shoulder. The pains were relieved when the pressure was reduced. Both patients were cured of their anginal attacks after operation.

Belet and Meade in their laboratory were unable to produce changes in the electrocardiogram of normal dogs by distending the gallbladder or common duct, but when even a small coronary vessel has been ligated then distention of the common duct brought about abnormalities in rhythm and conduction.

Gilbert[27] and his associates have shown a decrease in coronary flow following distention of the gallbladder or irritation of the ducts.

There is a large and growing list of careful observers who have added their observations bearing out this relationship between gallbladder disease and certain cardiac syndromes. They also have given their support to surgical intervention in the hope of relieving these patients of at least part of their symptoms.

Cholecystectomy vs. Cholecystostomy. To many this will seem an unnecessary discussion. Recently, when a surgeon was asked, "Did you remove the gallbladder?"—I heard this answer given: "No, there were so many stones in the gallbladder I thought I had better drain it."

Glenn,[28] in an article as late as October, 1939, said he believes cholecystectomy to be the operation of choice, but should not be done (1) in the

presence of peritonitis, (2) in conditions which make it difficult to identify important structures in the biliary fossa, (3) in the presence of severe jaundice caused by obstruction to the common duct, (4) in patients whose general condition is so grave that a general anesthetic and prolonged operative procedure are not justified. Other even less well-defined criteria have been offered for cholecystostomy.

There is nothing in the literature either on a statistical basis of mortality or morbidity, the clinical observation of surgeons with a wide experience, or the theoretical concept of biliary disease that will support the procedure of cholecystostomy. It is conceivable that fibrotic changes resulting in great distortion and atrophy of the gallbladder or the most extraordinary changes in the anatomy of the parts would necessitate cholecystostomy in rare instances.

Fallis and McClure[29] in a recent article said, "Cholecystostomy is a rare operation at Henry Ford Hospital."

Allen O. Whipple, in discussing a paper of Pennoyer's[30] on acute cholecystitis, made the point that "he had not performed a cholecystostomy in the last two years for acute cholecystitis."

Foss,[31] in discussing secondary operations on the biliary system, points out that of 140 patients requiring secondary operations 74.2 per cent had had cholecystostomy performed.

Heyd,[32] in analyzing 4000 operations on the external biliary system, found cholecystostomy had been done in 2 per cent of the cases and there was a mortality in chronic cholecystitis of 33.3 per cent when this operation was done. Of those who survived 68 required subsequent operation with a mortality of 7.4 per cent.

Cholecystostomy in acute cholecystitis was followed by a mortality of 34.4 per cent which almost equaled the mortality of acute cholecystitis with perforation and peritonitis. Of these patients who recovered it was necessary to do further surgery on them resulting in a mortality of 20 per cent.

In the follow-up clinic Heyd found 54 per cent of the patients had symptoms referable to the biliary tract after cholecystostomy.

It should be further pointed out that Heyd, Foss, and many others as far back as Deaver have believed cholecystic disease to be a progressive one beginning as a mild localized condition in the gallbladder extending into the cystic duct with obstruction, further involvement of the common duct, hepatic ducts, pancreatic ducts and finally pancreatitis and liver disease.

It is easily curable in the early stages but prone to produce serious or fatal complications if surgical treatment be too little or too late.

Cholecystostomy, then, is the answer of the defeatist whose surgical stature is that not of a man but of a mouse.

Early vs. Late Cholecystectomy in Acute Cholecystitis. This argument has been going on for some years, but has become particularly heated since most of us have been convinced and settled in our minds about the cholecystostomy versus cholecystectomy argument. There are several reasons why there has been confusion. The most important reason, I believe, is that

of definition. Definition not only of the terms "emergency," "early," and "late" operation, but also definition of the pathologic condition under discussion. There has been little unanimity among writers on this subject in their reports. For example, Fallis and McClure[29] divided 320 cases into three groups: the clinical, the surgical, and the pathologic. The report was based on a study of those diagnosed pathologically as acute cholecystitis. Others have used the leukocyte count or temperature readings as a basis. The majority do not define acute cholecystitis. Some writers base the time element before operation on the length of time the patient had been in the hospital before operation—others, more accurately, from the beginning of the onset of symptoms of the acute attack necessitating operation. Also there has not been a clear understanding of the reasons behind the argument for or against early or late operation. Of course mortality and morbidity are the reasons in the last analysis, but starting from these the analysis as a rule ends with statistical data relating the interval between the onset of attack or admission to the hospital and operation.

Great stress has been laid on the possibility of gangrenous changes in the wall of the gallbladder with empyema, perforation, and peritonitis. To avoid these complications is the reason given by those who urge early operation. Some have argued as you remove an acute appendix early so you should remove an acute gallbladder early, presupposing the pathologic conditions to be alike, which is not the case. But in studying the causes of death from acute cholecystitis, empyema, gangrene, and perforation are not impressive as the causes of mortality. All agree in many of the cases of empyema that the bile is sterile and exceedingly few gallbladders that have perforated produce a general peritonitis. The perforation or abscess or both are well walled off.

Pennoyer,[30] in the study of a group of 300 consecutive cases of clinically acute cholecystitis, found the higher mortality to be in those adults who were subjected to emergency operation. Half of those so operated were operated upon under an erroneous diagnosis, and half of the mortality for the entire series was in this group. There were 59 such cases with a mortality of 25 per cent. This gives an erroneous impression, however, as none of them had the benefit of any preoperative preparation and should not be used as evidence against early operation, as they do not properly fall into the group used to support early operation.

In an effort to clarify this situation Cave[33] grouped his cases under the terms "immediate," "early," and "delayed." His basis for the diagnosis of acute cholecystitis, although not stated, must have been clinical. The "immediate" group were those operated upon within the first 24 hours after admission to the hospital. The "early" group were those operated upon between one day and five days after admission to the hospital, and the "delayed" group those who were operated upon weeks or months after admission.

This grouping does not take into consideration the length of time between

the onset of symptoms and admission to the hospital, so that the figures are not entirely clear and accurate deductions may not be drawn. He found, however, that the highest mortality occurred in those cases operated upon "immediately." He quotes Branch and Zollinger, who found 14.4 per cent of 235 cases were operated upon immediately because of definite signs of peritonitis or threatened perforation with a mortality of 20.3 per cent. The rest of the patients were treated on an average of four to seven days before operation with a mortality of 8.7 per cent.

Referring to the report of Judd and Phillips, he found only 14 of 508 cases had been operated upon "immediately," and believes, therefore, these surgeons should be classified as those who favor "early" operation. In their report of 508 cases there were 61 cases of perforation out of 68 cases in which the wall of the gallbladder was gangrenous, and of those that perforated only three were not walled off but had ruptured into the free peritoneal cavity followed by general peritonitis.

Cave reviewed the study of Graham, of Toronto, who had a 16 per cent mortality in those patients operated upon "immediately," and a 4.8 per cent mortality in those operated upon "early."

Russell Best,[34] writing on the subject, says, "It has been only within the last four years that my personal ideas have come to agree with this latter viewpoint (serious surgical consideration in every case of acute cholecystitis). I can truthfully say that this change has come about through some rather unhappy observations and experiences as well as through a close study of reported series of cases." He then analyzes 44 cases of acute cholecystitis which he divided into two groups of 22 patients each. The first group represented a period in which he did not operate until "certain signs, symptoms, or laboratory findings were reasonably definite, although in several instances the decision to operate was based entirely on that inexact factor called 'clinical impression.'" The second group is comprised of an equal number during a later period in which the policy was to operate more readily unless there was an abatement of signs, symptoms, and improvement in the patient's general condition. The mortality in the first group was reduced from 13.6 to 4.5 per cent in the second, by favoring early operation.

Fallis and McClure,[29] in their report of 320 cases of acute cholecystitis, used for analytical purposes only the cases that were diagnosed as acute cholecystitis by the pathologist. They grouped their cases according to the time interval between the onset of symptoms of the acute attack and the time of operation. This appears to me as the proper way to classify them. The "emergency" group were operated upon within 24 hours, the "early" group those operated upon between 24 and 72 hours, and the "delayed" group after three or more days. The mortality figures for the three groups were 8.4 per cent, 7.3 per cent, and 3.4 per cent, respectively.

It would be difficult to correlate these findings with those of Cave's because it is likely Cave's cases had been ill at home at least 24 hours and possibly 48 or 72 hours before admission to the hospital. If this be true, most

of Fallis and McClure's cases would fall into Cave's second group. Some of each author's cases would be in the first group. Unfortunately, Fallis and McClure do not state the outside limit of time in their third group, but it is assumed to be days and not weeks which would, therefore, bring these cases also into the second group as classified by Cave. If these deductions be true, both authors are in agreement as to the favorable time of operation on a statistical basis.

The analysis of a large group reported by Heyd[32] shows some interesting figures. Of 574 patients in this group of acute cholecystitis, 128 were operated upon within six hours of admission to the hospital with a mortality of 15.6 per cent. Two hundred seventy-nine were prepared from six to 24 hours for operation. Among these there was a mortality of 7.4 per cent. Fifty-six patients operated upon from 24 to 48 hours after admission had a mortality of 10.35 per cent and 93 patients operated upon from 48 hours to 24 days after admission resulted in a mortality of 17.6 per cent. It is unfortunate the time between onset of the acute attack and operation was not given and that the cases operated upon between two days and 24 days were not further broken down to show the mortality in those operated upon between two and seven days and seven and 24 days. The mortality for those operated upon within the first 24 hours after admission of 9.8 per cent is approximately that of Fallis and McClure's emergency group of 8.4 per cent.

Heyd's study shows that of those operated upon within the first six hours 10 per cent were perforated. The delay of 18 hours increased the number of perforations by only 3.1 per cent but caused a reduction in mortality from 15.6 per cent to 7.4 per cent. A very interesting figure is that of a 17.6 per cent mortality in the group operated upon between the second and twenty-fourth day. From the figures given by others in their groups operated on between the second and seventh days of about 8 per cent, it would seem fair to assume that the mortality rate steadily rose up to the twenty-fourth day, reaching a mortality of 17.6 per cent.

This assumption is borne out by the figures of Graham and Hoefle[35] who report a mortality of 22.72 per cent in patients operated upon five days or more after the onset of symptoms. They report a mortality of 3.92 per cent in patients operated upon within 48 hours of the onset of symptoms and 7.4 per cent in those operated upon between two and five days of the onset of symptoms. In their cases there were three in which perforation had occurred in the group operated on within 48 hours of onset of symptoms and no cases of perforation in those operated on between two and five days, and one in the group operated upon five days or more after onset of symptoms.

Although the conditions upon which statistical data have been given have not been the same from the standpoint of time of operation and the criteria for diagnosis of acute cholecystitis have not been uniform, nevertheless deductions may be drawn which point rather definitely and convincingly to the favorable time for operation which is between the second and seventh days after the onset of an acute attack.

Secondly, although variations in technical skill of different operators should never be a determining factor in establishing sound practice, it should be pointed out that removal of an acutely inflamed gallbladder in the early stages (two to seven days) is very much easier, as the fibrinous exudate which attaches the duodenum, hepatic flexure of the colon, great omentum, and gastrohepatic omentum to the gallbladder and cystic duct, separates like wet paper when the proper line of cleavage is established. This is not true later. Then the exudate is more likely to be fibrous, firmly fixing these structures to the gallbladder making more likely damage to the common duct, duodenum, or colon.

Operation then should not be too early and certainly not too late. The majority of surgeons obviously favor "early" operations.

It is evident too from these studies that the reasons given by most writers on the subject, the fear of gangrene, perforation followed by abscess, or peritonitis should not be the determining reason for early operation. All agree that perforation without walling off into the free peritoneal cavity is a rare occurrence. It happened in three cases out of 508 reported by Judd and Phillips, 53 of 574 reported by Heyd. It is a fact also, however, that the mortality is higher in patients with gangrene, perforation, or walled-off abscess. Although the incidence of these complications was increased by 3.1 per cent by delaying operation six hours after admission, the mortality for the whole group of acute cholecystitis was reduced by 8 per cent.

Further study of the mortality of both acute and chronic cholecystitis reveals the fact that when the causes of death common to most operative procedures on the abdomen or pelvis (peritonitis and pneumonia) have been subtracted, the important and really significant cause of death in gallbladder disease is liver damage, and secondary damage to the kidney with which liver function is so intimately related. This, then, is the real determining factor in gallbladder mortality. The determination to operate early (after nature's immediate defensive mechanism has been established) is to prevent further damage to the liver and indirectly to kidney function. This is the principal reason, then, for early operation.

In Heyd's[32] report of 4,000 operations upon the external biliary system he states the death-producing conditions, in order of frequency, were peritonitis, pulmonary complications, and varying states of hepatic insufficiency.

Fallis and McClure,[29] in analyzing 17 deaths in 320 patients with acute cholecystitis who had had cholecystectomy found four died of pneumonia, two of myocardial failure, two of peritonitis, two of pulmonary embolism, two from surgical accidents. The remaining five died with temperatures above 101°. In four cases the temperature rose to 107°, and two were listed as peritonitis, and two as liver deaths. Of the fatal cases 82.3 per cent had occurred by the fifth postoperative day. The authors add, "This finding suggests that liver insufficiency may be a factor in many of the fatal cases as well as those definitely attributed to liver death."

Two other conditions adding greatly to mortality are jaundice and common-duct stones. These will be discussed separately later.

The term liver death was used by Heyd[36] to describe what has also been called the hepatorenal syndrome. He divided them into three types: (1) Those having chronic cholecystitis with jaundice who never regain consciousness after operation, with a rapidly rising temperature and pulse rate, and who die within 36 hours. (2) Those with obstructive jaundice having had choledochostomy with drainage. They progress favorably for a few days, then become delirious, lapse into coma, and the bile becomes less in amount and watery in appearance. (3) A rare form found in patients with a diseased pancreas and bile ducts without jaundice who after choledochostomy with drainage appear to be progressing favorably, when suddenly the pulse becomes markedly accelerated, there is a fall in blood pressure, suppression of urine, and collapse.

The similarity of this condition to thyroid crisis has been noted, and many believe them to be identical.

Briefly stated, the functions of the liver are very complex, but we know something of some of its functions. The liver manufactures prothrombin which is activated by vitamin K. It is essential for the normal coagulation of blood. Fibrinogen is manufactured in the liver. Heparin is stored there to some extent. The interaction between blood coagulation and anticoagulation is a nicely balanced function of the liver.

The detoxifying action of the liver is one of its most important functions.

Carbohydrates are stored in the form of glycogen, the conversion of dextrose from proteins and fats and the maintenance of the blood sugar level is brought about by the liver.

The liver manufactures most of the albumin fraction and some of the globulins.

The storage of a certain amount of fat in the liver is normal, but above that level it is abnormal and renders the liver more susceptible to damage.

The manufacture of bile acids from choleic acid and the amino acids glycine and taurine is a very important function.

Certain tests have been devised based on normal liver function to evaluate liver damage. The more important are the estimation of the prothrombin level and the response after activation by vitamin K.

The bromsulphalein test tests the detoxifying capacity of the liver.

The Quick[37] hippuric-acid test is another test based on the ability of the liver to conjugate benzoic acid with glycine to form hippuric acid which the urine excretes. This test obviously depends upon normal kidney function, which is so often altered in the hepatorenal syndrome.

The function of glycogen storage is tested by the galactose tolerance test and the ability of the liver to store proteins may be tested by serum albumin and globulin determination. There are other liver-function tests but none has merited full confidence.

Andrus, Lord, and Lake[38] believe the comparison of prothrombin levels

before and after the response to the intramuscular administration of two methyl one-four naphthoquinone is the most accurate test. In order of reliability the galactose tolerance test, hippuric acid and bromsulphalein follow according to their opinion.

It has been definitely shown that anoxemia, trauma, anesthesia, drop in intra-abdominal temperature, changes in hepatic and biliary pressure, kidney damage, and jaundice impair liver function.

Orr and Helwig[39] reported five cases of trauma to the liver with hepatorenal syndrome. Two of the five died. There are those who believe the syndrome is the result of some toxin created following failure of liver function which puts the detoxifying burden upon the kidneys in addition to their normal duties. The kidneys in succession fail in their duty and death ensues with uremic manifestations.

Boyce[40] suggests the reaction may be on a basis of anaphylactoid reaction.

The removal of those agents causing liver damage, the refraining from producing further liver damage, and the use of those measures that fortify the liver against further damage and restore at least to a degree normal liver function, are the desirable objectives in the treatment of diseases of the biliary passages.

Certainly the "early" removal of the gallbladder removes the principal offending agent. This should be done with the least possible trauma just as soon as the measures that fortify the liver against further damage and at least partial restitution of normal function have been instituted.

The preoperative and postoperative treatment are directed, therefore, toward the restoration of normal liver function. This may be accomplished by certain definite measures, of most importance being the reestablishing of a high glycogen content in the liver. It has been definitely shown that the glycogen content of the liver is reduced in proportion to the lipid content and that this lipid content may hold toxins causing degeneration and necrosis of liver cells. It has been further shown that glycogen will displace the fat within the liver cell, and that the glycogen content can be increased by certain dietary measures.

Many investigators, including Opie, Alford, Ravdin and his co-workers, Whipple, Bollman and Mann, have demonstrated that a high caloric diet rich in carbohydrate and protein and low in fat content will increase the glycogen content and reduce the lipid content of the liver, and that these measures protect the liver from damage or help to restore liver function after damage.

Others have demonstrated the value of vitamin B complex and certain vitamin-like substances in protecting the liver from damage.

Under ordinary circumstances these measures are made available to the patient by the use of a diet of high caloric value with a high protein (preferably milk as a source of amino acids), a high carbohydrate and low fat content.

If the patient be unable to take these foods by mouth, the carbohydrates may be administered by intravenous infusion of glucose and the protein as amino acids which may also be given intravenously. The supplementing by intravenous administration of mouth feeding is indicated in the majority of patients preoperatively and for a few days postoperatively.

The rôle of anoxemia in producing liver damage has been shown by Rich, and by Goldschmidt, Ravdin and Lucke.[41] This bears directly on the choice of anesthetic agent at the time of operation. The anoxemia accompanying pulmonary complications, anemia, and the demand in those patients whose oxygen requirements are high may lead to serious impairment of liver function.

Jaundice. Jaundice, because of its great damage to liver function and because of the bleeding tendency produced by it, is a complication fraught with great danger to the patient and presents additional problems to the surgeon. The discussion of jaundice in this paper of course deals with common-duct stone and inflammatory stricture of the common duct as the causes of the jaundice.

Heyd[32] gives a mortality in his group of cases with jaundice as 13 per cent. Coller and Farris,[42] in reporting operations upon 4,000 jaundiced patients, found postoperative hemorrhage to be the cause of death in 16 per cent.

The bleeding tendency has been found to be due to the low prothrombin level. Prothrombin is a serum protein manufactured by the liver. It is synthesized in the liver by an intrinsic factor manufactured in the liver and an extrinsic factor, the fat-soluble vitamin K requiring emulsification by bile and absorbed from the intestine. The discovering of vitamin K by Dam of Copenhagen is one of the great contributions to medicine.

A low prothrombin level may, therefore, result from impaired liver function or an inadequate supply of vitamin K reaching the liver.

The response of the liver as determined by the prothrombin level before and after the administration of vitamin K is as before stated an excellent test of liver function.

It is imperative to administer to the jaundiced patient vitamin K or one of its related compounds before and after operation. When the prothrombin level reaches normal, the patient may be operated upon without the fear of postoperative hemorrhage.

It goes without saying that these jaundiced patients need the other fortifying measures to restore liver function given to the nonjaundiced patients before enumerated.

The test for prothrombin Quick's,[43] or for the presumptive presence of prothrombin, the "Ivy[44] bleeding time" and serum volume index of Boyce and McFetridge[45] are the tests usually done. The latter two are very simple tests, and can be done with a minimum of equipment.

Common-duct Stones. This brings us inevitably to the serious consideration of common-duct stones.

Stones were present in the common duct in 86 per cent of 254 patients with jaundice; 17 per cent of patients with acute cholecystitis; 6.9 per cent

of patients with chronic cholecystitis, and 80 per cent of all patients operated upon secondarily after cholecystectomy in Heyd's[36] group.

Lahey[46] writes that he explores the common duct in from 40 to 50 per cent of patients operated upon for gallstones and finds stones in one out of six patients (16 to 20 per cent). He points out a very interesting group of 4 per cent in whom stones were found in the common duct, but not in the gallbladder.

Parsons[47] explored the common duct in 16 per cent and found stones in 2.4 per cent.

Fallis and McClure[29] found stones in the common duct alone in 1.2 per cent and in the gallbladder and common duct in 6.2 per cent.

In the report of Foss and Lillie,[31] 140 patients out of 2,485 were subjected to a second operation following either cholecystostomy or cholecystectomy. It was necessary to open the common duct in 34 per cent. Ninety-five of the group had had cholecystostomy. Of those, stones were found in the common duct in 24. Of the 33 having had cholecystectomy stones were found in 11.

Cutler and Zollinger[48] explore the common duct by opening it in 40 per cent of their cases, and have found stones in 22.8 per cent. They have set forth the following criteria for exploration of the common duct:

1. History or presence of jaundice
2. Cholangitis associated with cholelithiasis
3. Recurrent symptoms after cholecystectomy
4. Frequent attacks of gallstone colic
5. Pronounced involuntary vomiting
6. Suggestion of stone by palpation
7. Dilated or thickened duct
8. Contracted, thickened gallbladder
9. Dilated cystic duct
10. Thickening of the head of the pancreas
11. Many small stones in cystic duct and gallbladder.

Foss and Lillie gave as their indications for common-duct exploration "a dilated common duct, if stones are palpated within it if the clinical history and laboratory findings in any way suggest the presence of common-duct calculi."

Lahey[46] has added to these puncturing the common duct with a hypodermic needle and withdrawing into a glass syringe common-duct bile. When the bile is transparent, golden yellow and clear, rarely are stones found in the common duct.

These statistics present a very strong argument for frequent exploration of the common duct. In spite of its apparent advisability many authors find at autopsy and subsequent operations that stones have either been missed or others formed after previous thorough exploration. Best[34] summarizes the views of numerous surgeons who believe many stones are left in the common duct in spite of palpation, scoops, probing, and irrigation.

In necropsy material, Jurg[49] found stones were left after operation in 16.4 per cent; Bernhard[50] of the Giessen Clinic, 5 per cent; and W. J. Mayo[51] in nearly one-third of those whose common ducts had been opened.

There seems to be agreement among most writers that the mortality is not affected following exploration of the common duct. Lahey says, "The mortality in the face of these explorations has gone progressively downward and not upward."

Heyd found, in analyzing his group of cases, that the mortality in cholecystectomy for chronic cholecystitis was 3.61 per cent, and that with the addition of choledochostomy the risk was three times as great resulting in a mortality of 11.34 per cent when stones were present in the duct. However, without stones the mortality was not raised, being 3.8 per cent.

However, of 39 patients who had choledochostomy following a previous cholecystectomy, 32 had recurrent or overlooked stones and seven stenosis of the common duct. The mortality in this group was nearly 40 per cent.

Fallis and McClure found opening the common duct, which they did only in the presence of stone in the common duct, did not alter the mortality.

In this connection it is interesting to note that Heyd's study revealed that in those patients who had symptoms less than two years, only 2 per cent had common-duct stones; of those whose symptoms lasted between two and ten years, 9 per cent had common-duct stones; and in those with symptoms over ten years, 16 per cent had common-duct stones.

The high percentage of stones found after exploration of the common duct and the exceedingly high mortality following choledochostomy in cholecystectomized patients causes us to wonder whether the emphasis has been placed in the right direction in our approach to this subject.

I cannot pass this opportunity to speak of instrumental dilatation of the sphincter of Oddi, recommended by several leaders in surgical practice. This procedure seems to me to be unsound in every part except the hope of securing better drainage for common-duct bile or the passage of stones that might be overlooked or reform. There is no example of mechanical dilatation of any other sphincter in the body in which the sphincter remains dilated for any appreciable time after such mechanical dilatation. Witness, for example, the dilating of the uterine cervix. It is the experience of all that when normal conditions have returned and irritating reflexes have been discontinued the sphincters of the body as a rule return to normal function. Other objections have been raised—the possibility and danger of infection, duodenal reflux, and the splitting of the wall of the duct or duodenum. Such accidents have been reported by Lahey and others.

In addition Zollinger, Branch and Bailey[52] have shown by experimental evidence on both the human and dogs that damage may be done to the papilla of Vater by dilatation with hemorrhage and inflammation in the acute stages followed by scarring later which resulted in a smaller opening than that which followed cholecystectomy alone. They also demonstrated that perfusion pressures were greater, indicating a spasm of the sphincter of

Oddi which persisted for approximately four days after operation. Allen and Wallace[53] disagree on the basis of clinical comparative statistical data. They believe the conditions found in the dog's biliary apparatus should not be used as reliable comparative data with that found in the human, and their clinical studies they believed discredited those findings.

Cholangiography would seem in a small measure to answer some of this problem. It is a simple test, and will usually reveal the presence of a stone that has been overlooked or show abnormal functioning of the sphincter of Oddi following common-duct exploration. If a stone is demonstrated to be present, fair success in fragmenting it and washing out the fragments by irrigation after the modified method of Pribham may be expected. Two or three cubic centimeters of a mixture of two-thirds ethyl ether and one-third ethyl alcohol are used to fragment the stone, after which sterile normal salt solution may be used to wash out the fragments. Cholangiography may then be used to check the success of the effort. This of course, requires that a tube be placed securely within the common duct, which all agree should be done if the common duct be explored.

Surgical Technic. There are a few points of technic I believe worthy of bringing to your attention. Recently Lahey wrote a paper on this subject and recommended a trick which we have used in the dissecting room for many years to facilitate exposure of the bile passages. Packs are placed over the stomach and duodenum and fixed with retractors. A clamp is then placed on the ampulla of the gallbladder and held firmly. This puts the gastrohepatic omentum on tension. By blunt dissection with Mayo scissors the ducts may be exposed with great ease. In teaching residents and assistants I have carried this farther and suggest that by blunt dissection both the outer as well as the inner angle formed by the junction of the cystic duct with the common duct be exposed. Two clamps may then be applied to the cystic duct and the duct cut between without danger of injuring the common duct. This permits the gallbladder end of the cystic duct to be brought forward and a clamp can be applied with ease to secure the cystic artery, the points of the clamp directed away from the direction of the common duct. If the gallbladder is greatly distended and interferes with the exposure of the ducts, the withdrawing of its contents by aspiration with a trochar and cannula will greatly facilitate the removal of the gallbladder.

These seem to be simple points, but they are anatomically sound and give one a greater feeling of security than when the clamps are applied blindly to the gastrohepatic omentum.

The choice of the anesthetic agent is of course a particularly important one because of the liver damage incident to both gallbladder disease and anesthetic agents. This also applies to hypnotics and sedatives used preoperatively, for 24 or 48 hours. They are all detoxified by the liver and therefore throw a decided extra burden on the liver which may be the determining factor in breaking down an already badly damaged liver.

Coleman[54] studied the effect of local anesthesia, nitrous oxide and oxygen, nitrous oxide and oxygen and ether vapor, open ether, avertin, and spinal anesthesia on liver function using the bromsulphalein test in 100 cases. Avertin produced the greatest damage which lasted the longest time. He found the following to be the order of toxicity: local, nitrous oxide and oxygen, spinal, nitrous oxide and oxygen and ether vapor, open ether, and most toxic, avertin.

Frequently one is confronted with the problem of multiple operations. In the light of the great suspicion placed upon the appendix in the etiology of gallbladder disease the appendix should be removed. It is wise to remove the appendix first before the region of the gallbladder is disturbed.

In Heyd's report there was a mortality of 13.85 per cent when gastro-enterostomy was done in addition to cholecystectomy, 31 per cent when gastric resection was added, and 11.8 per cent when hysterectomy was done in addition to cholecystectomy.

Wound drainage is imperative because of the possibility of leakage of bile as a result of accessory ducts that have not been ligated, discharge of bile from the gallbladder bed, or slipping off of the ligature from the cystic duct or leakage around a tube in the common duct when that has been used. The mortality from bile peritonitis as reported by McLaughlin[55] was 62 per cent. Allen and Wallace[53] report a mortality of 22 per cent. Drainage should also be instituted because of the possibility of a collection of blood in the subhepatic space and some organisms lurking about the stump of the cystic duct or in the tissues which would result in a localized infection or possibly spread to a generalized peritonitis. Not to drain is taking an unnecessary and dangerous chance.

Allen and Wallace recommend placing the drainage tubes through a stab wound in the right flank. They find by so doing a reduction in major wound infections from 14.6 per cent to 3.4 per cent, wound dehiscence from 1.7 per cent to 0 per cent, and hernia in the scar from 7.1 per cent to 0.85 per cent.

Results. Finally, what can be expected from surgery in these diseases of the extrahepatic passages? Great improvement in mortality and morbidity figures will follow if the profession at large, particularly the general practitioner and gastro-enterologist, will be convinced that gallbladder disease is a progressive disease, easily cured in the early stages, but going on to acute attacks of cholecystitis, disease of the common duct, jaundice, cholangitis, hepatitis, and pancreatitis if the patient be denied the benefit of early and complete surgery. Also that these complications increase the mortality and add to the difficulty of curing the patient of his symptoms.

In Heyd's group of cases the mortality following cholecystectomy with and without appendectomy in patients who had symptoms under two years was only 1.35 per cent. In those who had a previous attack within two years, the mortality rose to 7.1 per cent. Each acute attack increased the mortality by 2 per cent. Jaundice in any degree raised the mortality to 13 per cent; in chronic cases and those who had no jaundice at time of operation

but a history of jaundice the mortality was 15.8 per cent, and jaundice in the acute cases resulted in a mortality of 20.6 per cent.

Pancreatitis was associated with common-duct stones in 78 per cent. In Foss and Lillie's cases 10 per cent of all those requiring secondary operations had marked evidence of chronic pancreatitis. They quote Weir and Snell of the Mayo Clinic who attribute, among other reasons, persistence of symptoms following cholecystectomy to residues of cholecystic disease such as cholangitis, hepatitis, and pancreatitis. Secondly, if we are to gain the highest percentage of cures with the lowest mortality, the surgeons must be convinced not only of early operation but complete operation with the removal of all diseased parts so that there cannot be further spread of the disease nor the necessity for secondary operations.

Writing on the subject of failure to cure patients with gallbladder disease Eliason and North[56] found 6 per cent were unrelieved of all symptoms. They divided them into four groups: the first were those who had a mistaken diagnosis, in the second group the gallbladder disease was only partly responsible for the symptoms. In the third group relief was delayed six to eight months following operation. The fourth group were those who had been relieved of their gallbladder symptoms but who subsequently developed symptoms from extraneous conditions.

Weir and Snell[57] add to their reasons for failure of surgery to relieve the symptoms of gallbladder disease, failure in diagnosis, surgical errors resulting in stricture of the bile passages, and visceromotor disturbances such as biliary dyskinesia.

Reich[58] has suggested denervating the distal end of the common duct in an effort to reduce the spasm of the sphincter of Oddi which he believes is the cause of biliary dyskinesia, and one of the causes of continuation of symptoms following cholecystectomy. Unfortunately, he reports the result of but one case. However, it is an interesting idea on which to speculate and warrants further study.

Foss and Lillie suggest a strong tendency to look upon cholecystectomy for so-called "noncalculous cholecystitis" as a surgical error and responsible for a high percentage of failures in curing symptoms. They question seriously the identity of this disease.

Fallis and McClure reported 15.1 per cent of 278 patients returned for treatment. Of these half complained of the same symptoms as before operation. All of these were relieved by medical treatment. Forty-five per cent of the remainder of those who returned had symptoms of common-duct stone. About one-quarter of these had a choledochostomy, of which stones were removed in three and two were operated upon for common-duct stricture with relief.

Parsons in his study quoted Kunath who reported a cure in the noncalculous group of 69 per cent and in the calculous group 84 per cent. He compares the results of medical treatment by quoting Finsterer's report of 89 patients treated medically. Eleven died from gallstone trouble, seven died

of perforation of the gallbladder, and two from carcinoma. Schittenhelm[59] estimated that 40 per cent of patients discharged had subsequent recurrent attacks and 25 per cent died or required surgery for severe complications. They were the same complications that produce the majority of surgical deaths.

Finsterer[60] concludes by saying he believes 87.8 per cent of patients are cured by cholecystectomy.

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TRANSACTIONS
OF THE
PHILADELPHIA ACADEMY OF SURGERY

THE YEAR 1939

Meeting of January 16, 1939, College of Physicians. The President, DR. J. STEWART RODMAN, in the Chair.

SCIENTIFIC PROGRAM

Memoir

EDWARD B. HODGE, M.D. — Memoir of the Late Dr. John H. Girvin.

Case Reports

WILLIAM T. LEMMON, M.D., AND G. W. PASCHAL, JR., M.D. (By Invitation.) — Total Gastrectomy for Carcinoma of the Stomach (Report of a Successful Case). Annals of Surgery, 112:No. 1, p. 31.)

ADOLPH A. WALKLING, M.D. — Total Gastrectomy for Carcinoma of the Stomach (Report of a Successful Case).

ELDRIDGE L. ELIASON, M.D., AND FRANCIS WOOD, M.D. (By Invitation.) — A Case of Calcified Constricting Pericardium. (Annals of Surgery, 111:No. 3.)

Paper

JOHN F. MCCLOSKEY, M.D. AND JAMES A. LEHMAN, M.D. — Living Fascial Suture in the Repair of Large Inguinal Hernia. (Annals of Surgery, 111:No. 4)

Meeting of February 6, 1939, College of Physicians. The President, DR. J. STEWART RODMAN, in the Chair.

SCIENTIFIC PROGRAM

Memoir

HENRY P. BROWN, JR., M.D. — Memoir of the Late Dr. Addinell Hewson

Case Report
FREDERICK A. BOTHE, M.D. — Calcinosis.

Papers
JOHN C. HOWELL, M.D. — The Treatment of Breast Cancer.

L. KRAEER FERGUSON, M.D.
AND
W. D. THOMPSON, M.D.
(By invitation.) — Internal Derangement of the Knee Joints: A Review of 100 Operative Cases with Follow-ups.
(Annals of Surgery, 112:No. 3, p. 454.)

Demonstration
(Moving Picture)
W. W. BAKER, M.D. — Surgical Treatment of Horseshoe Kidney with Special Reference to Division of the Isthmus.

Joint Meeting of the Philadelphia Academy of Surgery and the New York Surgical Society at the New York Academy of Medicine, Wednesday, February 8, 1939.

SCIENTIFIC PROGRAM

Case Reports
DR. ARTHUR S. W. TOUROFF
Discussed by:
DR. HENRY P. BROWN, JR. — Major Surgical Procedures in Premature Infants:
Congenital Gastric Obstruction Due to Complete Transverse Prepyloric Mucous Membrane Septum in a 5-day Old One-month Premature Baby.

DR. DEWITT STETTEN
Discussed by:
DR. WALTER E. LEE — Major Surgical Procedures in Premature Infants:
Duodenojejunostomy for Congenital Atresia at the Duodenojejunal Junction in a 3-day Old One-month Premature Baby.

DR. HENRY H. M. LYLE
Discussed by:
DR. LEON HERMAN — Ombredanne's Pouch Operation for Hypospadias: 3 Illustrative Cases.

DR. GEORGE J. HEUER
Discussed by:
DR. JOHN B. FLICK — Chronic Constrictive Pericarditis: Report of 7 Operated Cases.

Papers
DR. FENWICK BEEKMAN
AND
DR. JOHN E. SULLIVAN
(By Invitation.)
Discussed by:
DR. EDWARD T. CROSSMAN — Treatment of Blood-borne Pyogenic Infections of Bones and Joints.

DR. BYRON STOOKEY
Discussed by:
DR. FRANCIS C. GRANT — Herniation of Nucleus Pulposus: A Clinical Study.

DR. R. FRANKLIN CARTER
AND
DR. M. BRUGER
(By invitation.)
Discussed by:
DR. NORMAN E. FREEMAN — Nephro-omentopexy in Arterial Hypertension.

Meeting of the Boston Surgical Society with the Philadelphia Academy of Surgery, March 6, 1939, in Mitchell Hall, College of Physicians. The President, DR. J. STEWART RODMAN, and DR. WILLIAM E. LADD, in the Chair.

SCIENTIFIC PROGRAM

Case Report
ALEXANDER RANDALL, M.D.
Discussed by:
RICHARD H. MILLER, M.D. — Differential Diagnosis of a Case of Abdominal Tumor.

Paper
JOHN H. GIBBON, JR., M.D.
Discussed by:
STANLEY J. G. NOWAK, M.D. — Artificial Maintenance of the Circulation During Experimental Occlusion of the Pulmonary Artery, Followed by Survival.
(Surg., Gynec., and Obstet., 69: No. 5, p. 602.)

Case Report
GEORGE P. MULLER, M.D.
Discussed by:
ROBERT C. COCHRANE, M.D. — Excision of the Distal End of the Pancreas for Subacute Pancreatitis.

Papers
B. FRANKLIN BUZBY, M.D.
Discussed by:
HENRY H. FAXON, M.D. — Trophic Ulcer of the Plantar Surface of the Heel Secondary to Laceration of the Tendo-achillis.

JOHN S. LOCKWOOD, M.D. Discussed by: MARSHALL K. BARTLETT, M.D.	Some Bacteriological Considerations in Wound Healing.
Demonstration GILSON C. ENGEL, M.D., AND HANS MAY, M.D. (By invitation.) Discussed by: FRANK OBER, M.D.	Two-plane Direction and Range-finder in Treatment of Fractures of the Neck of the Femur. (Surg. Clin. N. Amer., 20:1721–1741, December, 1940.)
Paper VIRGIL H. MOON, M.D. Discussed by: ROBERT R. LINTON, M.D.	The Early Recognition of Shock and Its Differentiation from Hemorrhage. (Annals of Surgery, 110:No. 2, p. 260.)

Meeting of April 3, 1939, College of Physicians. The President, DR. J. STEWART RODMAN, in the Chair.

SCIENTIFIC PROGRAM

Papers LAWRENCE CURTIS, M.D.	Sarcoma of the Maxilla.
FRANCIS C. GRANT, M.D.	Surgical Treatment of Abscess of the Brain.
Case Reports J. WALTER LEVERING, M.D.	A Case of Situs Inversus with Acute Surgical Abdomen.
J. MONTGOMERY DEAVER, M.D.	Non-specific Inflammatory Lesions of the Cecum: Report of Two Cases.
DUNCAN CALDER, M.D. (By invitation.)	A Case of Complete Biliary Fistula of Four Years' Duration with Purpura.

Meeting of May 1, 1939, Thomson Hall, College of Physicians. The President, DR. J. STEWART RODMAN, in the Chair.

SCIENTIFIC PROGRAM

Case Reports DEFOREST P. WILLARD, M.D. AND	Cyst of the Internal Semilunar Cartilage.

JESSE T. NICHOLSON, M.D.	(Annals of Surgery, 112:No. 2, p. 305.)
HUBLEY R. OWEN, M.D.	Abdominal Aneurysm Treated with Installation of Gold Wire and Electrolysis.
ALAN P. PARKER, M.D.	Regional Ileitis with Unusual Manifestations.
Papers DEFOREST P. WILLARD, M.D. AND JESSE T. NICHOLSON, M.D.	Fracture-dislocations of the Odontoid and First Cervical Vertebra. (Annals of Surgery, 113: No. 3, p. 464.)
J. R. ELKINGTON, M.D. M. T. GILMOUR, M.D. W. A. WOLFF, PH.D. (All by invitation.)	Physio-chemical Determinations in the Management of Surgical Patients. (Annals of Surgery, 110:No. 6, p. 1050.)

Meeting of October 9, 1939, in Thomson Hall, College of Physicians. The President, DR. J. STEWART RODMAN, in the Chair.

SCIENTIFIC PROGRAM

Case Demonstration L. KRAEER FERGUSON, M.D. AND WILLIAM H. ERB, M.D. (By invitation.)	Ulcerative Colitis Treated by Colectomy.
Case Report JAMES B. MASON, M.D.	Recurrent Echinococcus Cyst in the Soft Tissues of the Thigh. (Surgery, 7:407–409.)
Papers ALBERT E. BOTHE, M.D.	Renal Hypertension. (Jour. Surg., November, 1939.)
BENJAMIN LIPSCHUTZ, M.D.	Regional Enteritis.

Meeting of November 6, 1939, College of Physicians. The President, DR. J. STEWART RODMAN, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

W. Emory Burnett, M.D.	Pneumonectomy for Benign Tumor.
Drury Hinton, M.D.	Case of Obstruction by Volvulus—Operation—Subsequent Attacks of Obstruction Relieved by Proctoscopy. (Annals of Surgery, **116**:147–149, July, 1942.)

Papers

Norman E. Freeman, M.D.	Physiological Principles in the Treatment of Varicose Veins. (Surg. Clin. N. Amer. **19**:1525, December, 1939.)
Hans May, M.D. (By invitation.)	The Plastic Repair of Scars. (Amer. Jour. Surg., **50**:754.)

Meeting of December 4, 1939, College of Physicians. The President, Dr. J. Stewart Rodman, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

L. A. Rademaker, M.D. (By invitation.)	A Case of Retractile Mesenteritis Associated with Early Carcinoma of the Gallbladder. (Amer. Jour. Surg., **52**:115–119.)
J. A. Lehman, M.D.	Pneumococcic Pericarditis.

Papers

William T. Lemmon, M.D.	Continuous Spinal Analgesia. (Annals of Surgery, **111**:1.)
S. Dana Weeder, M.D.	Mesenteric Adenitis.

SECRETARY'S REPORT FOR YEAR 1939

During the year 1939 there were seven regular and two joint meetings held. On February 8, the combined meeting with the New York Surgical Society was held in New York, forty-two members of the Academy attending. On March 6, the joint meeting with the Boston Surgical Society was held in Philadelphia.

During the year the average attendance at the meetings was thirty-seven members and thirty-five guests, which is an improvement of average one member, guests remaining the same as for 1938.

The following were elected to Fellowship (Active): Drs. Howard H. Bradshaw, David M. Davis, Donald C. Geist, Robert A. Groff, Alan P. Parker, and George Willauer. The present number of Active Fellows is 66.

In December four Active Fellows were transferred to the Senior List: Drs. Henry P. Brown, Jr., Edward T. Crossan, William B. Swartley, and DeForest P. Willard. The present number of Senior Fellows is 31.

Dr. Louis D. Englerth died August 16, 1939, having been elected to Fellowship in 1929. Dr. Thomas R. Neilson died October 25, 1939, elected to Fellowship in 1890. Dr. Louis H. Mutschler was appointed to read a Memoir of Dr. Neilson.

A memoir of Dr. John H. Girvin was read by Dr. Edward B. Hodge. The memoir of Dr. Addinell Hewson, written by his granddaughter, Mrs. A. L. Gucker, was read by the Secretary.

Dr. John B. Flick was appointed to present the Annual Oration for 1939.

There were nineteen case reports presented with fourteen discussions; twenty-two papers with twenty-one discussions and three demonstrations were made with two discussions.

The President announced the election to Army and Navy Fellowship of Commander F. L. Conklin, Commander William T. Lineberry, Lt. Col. Haskett L. Conner, Lt. Col. A. P. Hitchens and Lt. Col. John F. Corby.

The meeting of the Committee on the Bulletin of Surgical Clinics was held January 25, at which time Dr. George P. Muller's resignation from the Committee was accepted with sincere regret. The present officers were reelected for the ensuing year. A contribution of $500.00 from the Bulletin of Surgical Clinics to the Aid Association of the Philadelphia County Medical Society was approved.

It was decided to discontinue the recording of discussions of presentations before the Academy.

The following Amendment to the Constitution was adopted: Article VI, "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."

The Nominating Committee appointed by the President reported the following Nominations for 1940:

President—Dr. Eldridge L. Eliason.
First Vice-President—Dr. Robert H. Ivy.
Second Vice-President—Dr. Hubley R. Owen.
Secretary—Dr. John B. Flick.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Damon B. Pfeiffer and Dr. J. Stewart Rodman.
Program Committee—Dr. L. Kraeer Ferguson, Chairman, Dr. J. Montgomery Deaver and Dr. Adolph A. Walkling.

Respectfully submitted,
Henry P. Brown, Jr., M.D.
Secretary.

THE YEAR 1940

Meeting of January 10, 1940, in Thomson Hall, College of Physicians. The President, Dr. J. Stewart Rodman, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

Robert H. Ivy, M.D. — Bilateral Ankylosis of the Mandible. (Anales de Cirurgia, 6:No. 4, p. 3.)

Thomas A. Shallow, M.D. and Karl Houser, M.D. (By invitation.) — Retropharyngeal Lipoma.

John O. Bower, M. D. — Acute Intestinal Obstruction Due to a Hernia of the Transverse Colon Through the Diaphragm —Operation and Recovery.

Papers

L. M. Tocantins, M.D. (By invitation.) and W. A. Haus, M.D. (By invitation.) — Hypoprothrombinemia in Various Clinical Conditions; Effect of Peroral and Parenteral Administration of Crystalline Vitamin K. (Annals of Surgery, 113:276–283, February, 1941.)

John H. Gibbons, Jr., M.D. — Osteogenic Sarcoma; Diagnosis and Treatment with Illustrative Cases.

The combined meeting of the New York Surgical Society and the Philadelphia Academy of Surgery in Mitchell Hall, College of Physicians, Wednesday, February 14, 1940. The President, Dr. J. Stewart Rodman, in the Chair.

SCIENTIFIC PROGRAM

Case Report

Richard H. Meade, Jr., M.D. Discussed by: W. B. Parsons, M.D. and F. H. Amendola, M.D. — Massive Pancreatic Hemorrhage, Cholecystostomy and Severe Diabetes with Recovery.

Paper

Eldridge L. Eliason, M.D. and Julian Johnson, M.D. (By invitation.) Discussed by: P. Klingenstein, M.D. and F. E. Adair, M.D. — Acute Regional Enteritis. (International Clinics, 1:Ser. 3, 1940.)

Case Report

J. Montgomery Deaver, M.D. Discussed by: J. J. Russell, M.D. and R. F. Carter, M.D. — A Case of Delayed Rupture of the Spleen.

Paper

Hubley R. Owen, M.D. John Paul North, M.D. and Lewis C. Manges, Jr., M.D. (By invitation.) Discussed by: R. H. Kennedy, M.D., and D. Gordon, M.D. — Fractures of the Shaft of the Humerus Treated with the Hanging Cast. (Surg. Clin. N. Amer., December, 1940.)

Case Report

Gilson C. Engel, M.D., and Hans May, M.D. Discussed by: H. L. Wilson, M.D., P. A. Wade, M.D., and H. M. Bergaminin — The Regenerative Process in Bones in Fractures of the Neck of the Femur Treated with the Smith-Petersen Nail.

Paper

Thomas A. Shallow, M.D. Discussed by: Carl Eggers, M.D., and F. B. Berry, M.D. — The Operative Technique of Pulsion Pharyngeal Diverticulum and Traction Diverticulum of the Esophagus.

Combined Meeting of the Boston Surgical Society and the Philadelphia Academy of Surgery, in Boston, March 4, 1940.

SCIENTIFIC PROGRAM

Morning Session: Massachusetts General Hospital, George Robert White Building, Bigelow Amphitheatre.

Operations by members of the Staff.

W. A. Rogers, M.D.
Discussed by:
I. E. Deibert, M.D.
— Safe and Dangerous Reduction of Vertebral Body Fracture and Dislocation.

C. Lyons, M.D.
(By invitation.)
Discussed by:
J. L. Lockwood, M.D.
— Colon Bacillus Toxemia.

A. W. Allen, M.D.
Discussed by:
I. S. Ravdin, M.D.
— Surgery of the Biliary Passages.

L. S. McKittrick, M.D.
Discussed by:
A. A. Walkling, M.D.
— Early versus Delayed Operation in Acute Obstruction of the Small Intestine.

E. K. Churchill, M.D.
Discussed by:
H. H. Bradshaw, M.D.
— Metastatic Tumors of Lung—Diagnosis and Treatment.

Afternoon Session: Boston City Hospital, Dowling Building, Cheever Amphitheatre.

W. E. Ladd, M.D.
— Diaphragmatic Hernia.
(New England Jour. Med., **228**: 917–925, December 5, 1940.)

H. M. Clute, M.D.
Discussed by:
H. P. Brown, Jr., M.D.
— Diaphragmatic Hernia.

C. J. Mixter, M.D.
Discussed by:
D. B. Pfeiffer, M.D.
— Case Report.

D. Munro, M.D.
Discussed by:
R. A. Groff, M.D.
— Care of the Back in Spinal Cord Injuries — A Consideration of Bed Sores.
(New England Jour. Med., **223**: 391–398, September 12, 1940.)

J. Homans, M.D.
Discussed by:
N. E. Freeman, M.D.
— Minor Causalgias.
(New England Jour. Med., **222**: 870–874, May 23, 1940).

G. E. Haggart, M.D.
Discussed by:
J. R. Moore, M.D.
— Surgical Treatment of Degenerative Arthritis of the Hips and Knee Joints.
(Jour. Bone and Joint Surg., **22**: 717–729, July, 1940.)

C. W. Walter, M.D.
(By invitation.)
— Sterilization Technique.
(Motion Picture Demonstration.)

Meeting of April 1, 1940, in Cadwalader Hall, College of Physicians. The President, Dr. Eldridge L. Eliason, in the Chair.

SCIENTIFIC PROGRAM

Memoir

L. H. Mutschler, M.D.
— Memoir of the Late Thomas R. Neilson, M.D.

Case Reports

Thomas J. Summey, M.D.
— A Massive Goiter.

Calvin M. Smyth, Jr., M.D.
— Nontraumatic Stricture of the Hepatic Duct.

Papers

I. S. Ravdin, M.D.,
and
J. S. Lockwood, M.D.
(By invitation.)
— Use of Sulfanilamide in the Prevention of Peritonitis Following Resections of the Large Bowel.
(Surgery, **8**:No. 1, pp. 43–55.)

T. McKean Downs, M.D.
— Congenital Anomalies of the Appendix a Familial Disease.

Meeting of May 6, 1940, in Cadwalader Hall, College of Physicians. The President, Dr. Eldridge L. Eliason, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

William J. Ryan, M.D.
— Hemorrhage into the Thyroid Gland as a Result of Muscular Effort, with Review of the Literature.
(Annals of Surgery, **115**:No. 3, p. 469.)

ALAN P. PARKER, M.D. — Stab Wound of the Heart, Recurring Tamponade and Recovery.

Papers

LOUIS KAPLAN, M.D. (By invitation.) — Observations on the Relationship of the Scalenus Anticus to Shoulder Pain. (Pa. Med. Jour., 44:289–297, December, 1940).

JAMES A. LEHMAN, M.D. — Prevention of Complications in Thyroid Surgery.

Meeting of October 7, 1940, Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

W. EMORY BURNETT, M.D. — A Successful Case of Total Gastrectomy for Carcinoma.

WILLIAM T. LEMMON, M.D. — Spontaneous Rupture of the Stomach and Lower Esophagus. (Annals of Surgery, 114:No. 6.)

Annual Oration for 1939

JOHN B. FLICK, M.D. — Lobectomy for Chronic Pulmonary Suppuration. (Bulletin of the Ayer Clinical Laboratory, 3:No. 17, p. 375.)

Meeting of November 4, 1940, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

I. S. RAVDIN, M.D., AND J. E. RHOADS, M.D. (By invitation.) — Reconstruction of the Common Bile Duct After the Method of Speese and Ginsberg.

THOMAS A. SHALLOW, M.D. — Primary Carcinoma of the Hepatic Ducts, Report of Two Cases.

Papers

FREDERICK A. BOTHE, M.D. — Mesenteric Adenitis. (International Clinics, 4:77–88, December, 1941.)

RICHARD H. MEADE, JR., M.D. — Healing of Abdominal Wounds in Perforated Peptic Ulcers.

Meeting of December 2, 1940, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

THOMAS J. SUMMEY, M.D., AND FRANCIS F. BORZELL, M.D. (By invitation.) — Trichobezoar. (Pa. Med. Jour., 45:No. 2, p. 142.)

DRURY HINTON, M.D. — Leiomyoma of the Stomach.

Papers

L. M. TOCANTINS, M.D. (By invitation.) AND J. F. O'NEILL, M.D. (By invitation.) — The Bone Marrow Cavity as a Route for the Parenteral Administration of Blood and Other Fluids. (Exper. Biol. and Med., 45:292–296, October, 1940.)

NORMAN E. FREEMAN, M.D., AND HUGH MONTGOMERY, M.D. (By invitation.) — Lumbar Sympathectomy in the Treatment of Intermittent Claudication.

SECRETARY'S REPORT FOR YEAR 1940

DURING the year 1940 the Philadelphia Academy of Surgery held six regular stated meetings and two combined meetings. On February 14, 1940, forty-four members of the New York Surgical Society met with the Fellows of the Academy of Surgery in Philadelphia. On March 4, 1940, twenty-five Fellows of the Academy of Surgery met with the members of the Boston Surgical Society in Boston.

Excluding the combined meeting with the New York Surgical Society, the average attendance at the Academy meetings held in Philadelphia, was Fellows thirty-seven, and guests forty-five. The average number of Fellows remained the same as in 1939, but the average number of guests increased by ten. Eight papers and twelve case reports were presented by Fellows and three papers and one case report by guests. There were forty-six discussions of papers or case reports by Fellows and three by guests. These figures do not include the combined meetings.

There was only one election to Active Fellowship during the year, that

of Dr. Hans May, and no one was transferred to the Senior membership. The number of Active Fellows at the end of the year was sixty-seven and the Senior Fellows was thirty.

At the January 10th meeting, the President announced that a portrait of Dr. Astley P. Ashhurst had been presented by several Fellows of the Academy and had been placed in Cadwalader Hall. At the same meeting the Academy voted to contribute two hundred dollars ($200.00) from the funds of the Bulletin of Surgical Clinics toward the purchase of a First Edition Volume of "Cinq livres" by Ambroise Paré, the volume to be placed in the Samuel D. Gross Library of the College.

A memoir of the late Dr. Thomas R. Neilson was read by Dr. L. H. Mutschler at the April meeting of the Academy.

Lieutenant Colonel Arthur P. Hitchens represented the Academy at the Eighth American Scientific Congress held in Washington on May 10 to 21, 1940.

The Committee on the Samuel D. Gross Prize announced on May 6 that by January 1, 1940, eighteen manuscripts had been submitted in competition and that after careful study, the Committee awarded the prize to Dr. Frederick Fitzherbert Boyce of New Orleans for his essay entitled "The Role of the Liver in Surgery."

Dr. Charles F. Nassau died on August 11, 1940. He was elected to Active Fellowship in 1905.

Lieutenant Commander H. L. Pugh and Commander T. C. Anderson were elected to Army and Navy Fellowship during the year.

The Annual Oration for 1939 was given by Dr. John B. Flick in October, 1940. The title was "Lobectomy for Chronic Pulmonary Suppuration."

At a meeting of the Committee on the Bulletin of Surgical Clinics held on January 10, 1940, Dr. Hubley R. Owen's resignation was accepted with regret. Dr. James A. Lehman was elected to membership to fill the vacancy. At a special meeting held on December 2, 1940, Dr. Henry P. Brown, Jr., was elected Chairman, this position having been held by the late Dr. Charles F. Nassau. The resignation of Dr. Eldridge L. Eliason was accepted with regret and Dr. W. E. Burnett was elected to take his place.

Beginning in December, 1940, The Bulletin of Surgical Clinics has furnished each hospital subscribing to the Bulletin, a mimeographed program, on a sheet separate from that which lists operations, a week prior to the meeting of the Philadelphia Academy of Surgery.

The volume labeled "Constitution and By-Laws" and the Academy seal which had not been in use since 1931, were located in the vault of the College of Physicians by Mr. W. B. McDaniel, 2nd, and those who had not signed the "Constitution and By-Laws" were given the opportunity to do so. The Academy voted that certificates of Fellowship be given those requesting them, and that in the future, certificates be issued to every new Fellow.

In the Fall, the Academy presented the College of Physicians with a new projection screen for use in Cadwalader Hall.

The Nominating Committee, appointed by the President, made the following nominations for the year 1941:

President—Dr. Eldridge L. Eliason.
First Vice-President—Dr. Robert H. Ivy.
Second Vice-President—Dr. Hubley R. Owen.
Secretary—Dr. John B. Flick.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council (elected members)—Dr. J. Stewart Rodman and Dr. Edward B. Hodge.

Program Committee—Dr. L. Kraeer Ferguson, Chairman, Dr. Adolph A. Walkling and Dr. J. Montgomery Deaver.

Respectfully submitted,

JOHN B. FLICK, M.D.,
Secretary.

THE YEAR 1941

Meeting of January 13, 1941, in Mitchell Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Guest Speaker

HENRIK DAM,
Associate Professor, Biochemical Institute, University of Copenhagen.

Vitamin K, Its Role in Human Pathology and Its Application to Therapeutics.
(Klin. Wchnschr., **19**:729–732, July 20, 1940.)

Meeting of February 3, 1941, in Cadwalader Hall, College of Physicians. The First Vice-President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Memoir

THOMAS A. SHALLOW, M.D.

Memoir of Charles F. Nassau, M.D.

Case Reports

JAMES B. MASON, M.D.

Mediastinal Goiter.
(Annals of Surgery, **116**:No. 5, 795.)

HANS MAY, M.D.

Nicola Operations for Fracture Dislocation of the Head of the Humerus.
(Jour. Bone and Joint Surg., **25**: 78, Jan., 1943.)

Papers

CLARE C. HODGE, M.D. (By invitation.) — The Ambulatory Treatment of Fractures of the Leg and Foot by Means of the Skate Type of Walking Iron.

KENNETH E. FRY, M.D. (By invitation.) — Peritoneoscopy, A Report of Over One Hundred Cases. (Surg. Clin. N. Amer., 20:1849–1858, December, 1940.)

Combined Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery, February 12, 1941, in New York. New York Academy of Medicine, New York City.

SCIENTIFIC PROGRAM

FENWICK BEEKMAN, M.D. Discussed by: F. A. BOTHE, M.D. — Hurthle Cell Tumor of the Thyroid with Tracheal Obstruction and Subtotal Thyroidectomy at the Age of Six Weeks—Recurrence Four Months Later.

GEORGE HUMPHREYS, M.D. (By invitation.) Discussed by: JULIAN JOHNSON, M.D. (By invitation.) — Ligation of Patent Ductus Arteriosus—Three Illustrative Cases.

BEVERLY C. SMITH, M.D. Discussed by: S. DANA WEEDER, M.D. — Cyst of the Common Duct.

JOHN J. WESTERMAN, M.D. Discussed by: DAMON B. PFEIFFER, M.D. — Preliminary Colostomy — Secondary Subtotal Gastrectomy.

EDWARD W. PETERSON, M.D. Discussed by: J. MONTGOMERY DEAVER, M.D. — Tumor of the Carotid Body.

SAMUEL STANDARD, M.D. Discussed by: I. S. RAVDIN, M.D. — Intestinal Obstruction Due to Intussusception of Benign Fibroma of the Jejunum.—Resection and Anastomosis—Recovery.

HERBERT WILLY MAYER, M.D. Discussed by: WILLIAM T. LEMMON, M.D. — Extensive Linitis Plastica Involving the Entire Stomach and Lower Esophagus—Total Gastrectomy and Partial Esophagectomy—Intrathoracic Esophagojejunostomy—Recovery.

ARTHUR S. W. TOUROFF, M.D. Discussed by: JOHN B. FLICK, M.D. — Unusual Traumatic Perforation of the Cervical Esophagus — Prompt Operation and Recovery. (Annals of Surgery, 114:369–375, September, 1941.)

JAMES T. PILCHER, M.D. Discussed by: GEORGE P. MULLER, M.D. — Pancreatic Calculosis.

Meeting of March 3, 1941, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

J. WALTER LEVERING, M.D. — An Unusual Case of Intestinal Obstruction.

ERNEST G. WILLIAMSON, M.D. — Two Cases of Primary Tuberculosis of the Stomach.

Papers

DAVID M. DAVIS, M.D. — How Safe Is Prostatic Surgery? (Urol. and Cutan. Rev., 44:No. 11.)

FRANCIS C. GRANT, M.D. — Spinal Epidural Abscess, Diagnosis, and Treatment. (Trans. Amer. Neurol. Asso., 67: 99–103, 1941.)

Meeting of April 7, 1941, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Memoir

HUBLEY R. OWEN, M.D. — Memoir of Louis D. Englerth, M.D.

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HENRY P. ROYSTER, M.D. (By invitation.) AND I. S. RAVDIN, M.D. — Five Perforations of a Gastric Ulcer.

ADOLPH A. WALKLING, M.D. — Pancreatic Cyst Treated by Partial Pancreatectomy.

LLOYD W. STEVENS, M.D. (By invitation.) AND L. KRAEER FERGUSON, M.D. — Unilateral Elephantiasis Treated by Modified Kondoleon Operation.

Papers

JOHN PAUL NORTH, M.D. — Chronic Tenosynovitis.

BENJAMIN LIPSHUTZ, M.D. — Imperforate Anus Associated with Rectovesical Fistula.

Meeting of May 5, 1941, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

JOHN B. FLICK, M.D. AND FRANK F. ALLBRITTEN, JR., M.D. (By invitation.) — Teratoma of the Mediastinum.

Papers

CHARLES H. HARNEY, M.D. (By invitation.) — Multiple Primary Malignant Disease.

HUBLEY R. OWEN, M.D., AND LEWIS C. MANGES, JR., M.D. (By invitation.) — Treatment of Bimalleolar Fractures and Fractures of the Lower Third of the Tibia and Fibula.

EDWARD F. MCLAUGHLIN, M.D. (By invitation.) — Ovarian Lesions Simulating Appendicitis.

Meeting of October 13, 1941, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

FREDERICK A. BOTHE, M.D. — Hyperthyroidism with Osteoporosis and Spontaneous and Traumatic Fractures of the Vertebrae.

HUBLEY R. OWEN, M.D., AND LEWIS C. MANGES, JR., M.D. (By invitation.) — Neurogenic Sarcoma, Two Cases.

BENJAMIN LIPSHUTZ, M.D. AND LOUIS KAPLAN, M.D. (By invitation.) — Traumatic Aneurysm of the Vertebral Artery.

Annual Oration for 1940

FRANCIS C. GRANT, M.D. — Brain Abscess.

Meeting of November 3, 1941, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

WILLIAM T. LEMMON, M.D., AND GEORGE W. PASCHAL, JR., M.D. (By invitation.) — Splenic Abscess with Drainage and Recovery.

L. KRAEER FERGUSON, M.D., WILLIAM H. ERB, M.D., AND EDWARD S. DILLON, M.D. (By invitation.) — Adenoma of the Pancreas.

Paper

ALLAN D. WALLIS, M.D. (By invitation.) AND MARGARET J. DILWORTH, B.S. (By invitation.) — Odor in the Orr Treatment of Osteomyelitis and Its Prevention by Lactose. (Jour. Amer. Med. Asso., 120:pp. 583.)

Paper-of-the-Evening

JOHN S. LOCKWOOD, M.D. (By invitation.) — Chemotherapy in Surgery. (Bancroft System of Surgical Therapeusis, Vol. 2, Appleton-Century Co., N. Y.)

Meeting of December 1, 1941, in Thomson Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

ORVILLE C. KING, M.D. — A Case of Traumatic Appendicitis.

ROBERT B. BROWN, M.D. (By invitation.) AND ROBERT C. MCELROY, M.D. (By invitation.) — A Case of Abortion with Prolapse and Gangrene of the Ileum.

Paper

ALBERT G. MARTIN, M.D. (By invitation.) — A Simple Method of Treating Metacarpal Fractures.

Annual Oration for 1941

WILLIAM BATES, M.D. — The Control of Somatic Pain. (Amer. Jour. Surg., 59:83, January, 1943.)

SECRETARY'S REPORT FOR YEAR 1941

DURING the year 1941, the Philadelphia Academy of Surgery held eight regular stated meetings. In addition, on February 12, 1941, thirty-eight Fellows of the Philadelphia Academy met with members of the New York Surgical Society in New York. Following an excellent scientific program, dinner for the combined societies was held at the Harvard Club. After dinner, Dr. Allen O. Whipple presented Dr. George Woolsey with a memento on behalf of the New York Surgical Society in commemoration of Doctor Woolsey's fifty years as a member of the Society. Doctor Woolsey gave many interesting reminiscences. Dr. Philip D. Wilson gave an account of his recent experiences in England.

The customary combined meeting of the Boston Surgical Society and the Philadelphia Academy of Surgery was not held, the President of the Boston Surgical Society declining with regret the invitation of the Philadelphia Academy, owing to the uncertainty of the times.

Excluding the combined meeting with the New York Surgical Society, the average attendance at the Academy meetings was Fellows thirty-three and guests fifty-five. The average number of Fellows was four less than that of 1940, but the average number of guests was increased by ten. The latter is accounted for, at least in part, by the very large attendance at the January 13th meeting (one hundred sixty-seven) when Dr. Henrik Dam addressed the Academy on "Vitamin K, Its Role in Human Pathology and Its Application to Therapeutics."

Six papers and twelve case reports were presented by Fellows and eight papers and three case reports by guests. There were forty-five discussions by Fellows and six by guests. These figures show a decided increase in the participation of guests in the Academy meeting.

Four Active Fellows became Senior Fellows during the year and four new Fellows were elected. One Active Fellow, having moved to Winston-Salem, North Carolina, resigned, and was made a Non-resident member. The number of Active Fellows at the end of the year was sixty-four, of Senior Fellows thirty-five.

Dr. James H. Baldwin, elected to Fellowship in 1917, died on April 18, and was buried in the National Cemetery in Baltimore on April 22, 1941.

The Annual Oration for 1940 was given by Dr. Francis C. Grant on October 13. The title was "Brain Abscess." The Annual Oration for 1941 was given by Dr. William Bates on December 1. The title was "The Control of Somatic Pain."

During the year, the Samuel D. Gross Library received a copy of the monograph "The Role of the Liver in Surgery" by Frederick Fitzherbert Boyce, recipient of the Samuel D. Gross Prize for research surgery awarded by the Philadelphia Academy of Surgery in 1940. Also, the gift by Dr. J. Stewart Rodman, of a copy of Dr. C. G. Morton's "Human Anatomy."

At a stated meeting held on May 5, 1941, a resolution was adopted whereby "Upon written request to the Council, any Fellow of the Academy who enters Government Service, the Red Cross, or similar activity and thereby is unable to continue in practice, may have his annual dues remitted subject to the approval by Council."

At a stated meeting held on October 13, 1941, a resolution was adopted authorizing payment from the treasury of the expenses of the out-of-town guests attending the combined meeting of the New York Surgical Society and Philadelphia Academy of Surgery in Philadelphia on February 12, 1942.

The Nominating Committee, appointed by the President, proposed the following nominations for the year 1942:

President—Dr. Robert H. Ivy.
First Vice-President—Dr. Hubley R. Owen.

Second Vice-President—Dr. John B. Flick.
Secretary—Dr. L. Kraeer Ferguson.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Thomas A. Shallow and Dr. Eldridge L. Eliason.
Business Committee—Dr. Calvin M. Smyth, Jr., Chairman, Dr. J. Montgomery Deaver and Recorder.

Respectfully submitted,
JOHN B. FLICK, M.D.
Secretary.

THE YEAR 1942

Meeting of January 5, 1942, in Cadwalader Hall, College of Physicians. The President, DR. ELDRIDGE L. ELIASON, in the Chair.

SCIENTIFIC PROGRAM

Memoir
CALVIN M. SMYTH, JR., M.D. — Memoir of Dr. James H. Baldwin.

Papers
JOHN O. BOWER, M.D., AND (By invitation) L. A. TERZIAN, PH.D., E. A. PEARCE, M.D., J. C. BURNS, M.D., H. B. TRACHTENBERG, M.D. — Appendicitis-Peritonitis — Report of Blood Changes Occurring in Acute Appendicitis and Appendicitis-Peritonitis in Man and Dog Similar to Those Observed in Shock. A Preliminary Report of the Use of Plasma in the Management of Appendicitis-Peritonitis in Man.

C. WILMER WIRTS, M.D. (By invitation.) — The Diagnosis of Early Carcinoma of the Stomach.

Paper-of-the-evening
GEORGE P. MULLER, M.D. — Surgical Aspects of Carcinoma of the Stomach.

Annual Conjoint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery, February 11, 1942, in Mitchell Hall, College of Physicians, Philadelphia, Pa. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Report
W. EMORY BURNETT, M.D. — Postanal Teratoma Removed 20 Hours After Birth, Followed by Plastic Repairs.

Paper
I. S. RAVDIN, M.D. AND HARRY VARS, M.D. (By invitation.) — The Effect of Diet in Man in Conditioning the Liver Against Minimal Injury Together with Observations on Regeneration.

Case Report
THOMAS A. SHALLOW, M.D., AND KENNETH E. FRY, M.D. (By invitation.) — Removal of Cysts from the Gastrocolic Omentum; Two Cases—(Motion Pictures).

Paper
LESLIE A. CHAMBERS, M.D. (By invitation.) T. N. HARRIS, M.D. (By invitation.) FRANCIS SCHUMANN, M.D. (By invitation) AND L. KRAEER FERGUSON, M.D. — The Use of Microcrystals of Sulfathiazole in Surgery.

Case Report
JOHN B. FLICK, M.D. AND FRANK F. ALLBRITTEN, JR., M.D. (By invitation.) — Ewing's Tumor of Rib.

LEANDRO M. TOCANTINS, M.D. (By invitation.) — Bone Marrow Infusion and Transfusion (Demonstration and Motion Pictures).

Meeting of March 2, 1942, in Cadwalader Hall, College of Physicians. Second Vice-President, DR. JOHN B. FLICK, in the Chair.

SCIENTIFIC PROGRAM

Case Report
WILLIAM H. ERB, M.D. — Epiphyseal Separation of the Lower End of the Femur With Growth Disturbance.

Papers

EDWIN R. RISTINE, M.D. — Perforated Peptic Ulcer. Review of 12 Years' Experience at Cooper Hospital.

JONATHAN E. RHOADS, M.D. — Newer Methods in the Treatment of Burns.

Meeting of April 13, 1942, in Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Report

WILLIAM B. SWARTLEY, M.D., AND EDWARD F. MCLAUGHLIN, M.D. (By invitation.) — Thrombosis of the Arm Followed by Gangrene of the Hand and Forearm in a Patient with Polycythemia Vera.

Paper

JULIAN JOHNSON, M.D. — Total Pneumonectomy.

Paper-of-the-evening

RALPH M. TOVELL, M.D. of Hartford, Conn. (By invitation.) — The Department of Anesthesia in the Modern Hospital.

Meeting of May 4, 1942, in Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Report

NORMAN FREEMAN, M.D., AND ROBERT H. IVY, M.D. — Extensive Frost Bite of Both Feet with Recovery.

Papers

T. TURNER THOMAS, M.D. — Union Without Internal Fixation in Intracapsular Fractures of the Femoral Neck.

SAMUEL GOTTMAN, M.D. (By invitation.) — The Treatment of Spinal Anesthesia Headaches.

Paper-of-the-evening

EDWARD CROSSAN, M.D. — Acute Osteomyelitis.

Meeting of October 12, 1942, in Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Report

DAMON B. PFEIFFER, M.D., AND SIMMONS PATTERSON, M.D. (By invitation.) — Hereditary Polyposis of the Colon.

Paper

HANS MAY, M.D. — Immediate and Late Treatment of Burns (Motion Picture Demonstration).

FREDERICK A. BOTHE, M.D. — Granulomas of the Thyroid.

Paper-of-the-evening

P. C. COLONNA, M.D. (By invitation.) — Differential Diagnosis of Painful Hip of Childhood.

Meeting of November 2, 1942, in Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

J. STEWART RODMAN, M.D. GEORGE WILSON, M.D. (By invitation) AND CHARLES STEINER, M.D. (By invitation.) — Report of a Case Showing Total Unconsciousness Following Cranial Injury for a Period of 94 Days with Recovery.

GILSON C. ENGEL, M.D. — Mesenteric Cyst and Volvulus in an Infant Three Days Old.

Papers

ALBERT E. BOTHE, M.D. — Urologic Surgery at the National Naval Medical Center.

THOMAS A. SHALLOW, M.D. — Jejunal Carcinoma.

Meeting of December 7, 1942, in Cadwalader Hall, College of Physicians. The President, DR. ROBERT H. IVY, in the Chair.

SCIENTIFIC PROGRAM

Case Reports

James A. Lehman, M.D.	Penetrating Wound of the Abdomen Caused by Crowbar.
Thomas A. Shallow, M.D.	Choledochus Cyst. Resection of Cyst—Right and Left Hepaticoduodenostomy. (Annals of Surgery, 117:No. 3, pp. 355.)
William B. Swartley, M.D., and Lt. Col. William C. Sheehan (By invitation.)	Choledochus Cyst. Anastomosis of Cyst to Duodenum. Hepatic Abscess After One Year.

Annual Oration

S. Dana Weeder, M.D.	Cholecystitis — Cholelithiasis: A Review.

SECRETARY'S REPORT FOR 1942

During the year 1942 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.

The conjoint meeting of the New York Surgical Society and the Philadelphia Academy was held in Mitchell Hall, College of Physicians, on February 11, 1942. Forty-two members of the New York Surgical Society attended as guests of the Academy. The meeting was followed by a dinner at the Racquet Club.

During the year there were nineteen papers and fifteen case reports presented, of which thirteen papers and fourteen case reports were by Fellows, six papers and one case report by guests. One memoir was read. There were forty-eight discussions of these presentations of which forty were by Fellows and eight by guests.

Four Active Fellows were transferred to the Senior list and four candidates were elected to Active Fellowship. There was one election to the Special Army and Navy Fellowship.

Dr. James Torrance Rugh elected to the Fellowship in 1913 died on October 12, 1942.

The Annual Oration for 1942 was pronounced by Dr. S. Dana Weeder on December 7 on the subject of "Cholecystitis and Cholelithiasis: A Review."

The Nominating Committee consisting of Drs. Pfeiffer, Muller, and Eliason, appointed by the President, recommended the election of the following officers for 1943:

President—Dr. Robert H. Ivy.
First Vice-President—Dr. Hubley R. Owen.
Second Vice-President—Dr. John B. Flick.
Secretary—Dr. L. Kraeer Ferguson.
Treasurer—Dr. Harry E. Knox.
Recorder—Dr. Adolph A. Walkling.
Council—Dr. Thomas A. Shallow and Dr. Eldridge L. Eliason.
Business Committee—Dr. Calvin M. Smyth, Chairman, Dr. J. Montgomery Deaver and the Recorder.

Respectfully submitted,
Calvin M. Smyth, M.D.
Secretary Pro Tem.

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Rogers, W. A.
Royster, Henry P.
Ryan, William J.
Schumann, Francis
Shallow, Thomas A.
Sheehan, William C.
Smith, Beverly C.
Smyth, Calvin M., Jr.
Standard, Samuel
Steiner, Charles
Stetten, Dewitt
Stevens, Lloyd W.
Stookey, Byron
Sullivan, John E.
Summey, Thomas J.
Swartley, William B.
Terzian, L. A.
Thomas, T. Turner
Thompson, W. D.
Tocantins, L. M.
Touroff, Arthur S. W.
Tovell, Ralph M.
Trachtenberg, H. B.
Vars, Harry
Walkling, Adolph A.
Wallis, Allan D.
Walter, C. W.
Weeder, S. Dana
Westerman, John J.
Willard, DeForest P.
Williamson, Ernest G.
Wilson, George
Wirts, C. Wilmer
Wood, Francis