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1929-
1931

TRANSACTIONS

OF THE

PHILADELPHIA

ACADEMY OF SURGERY

VOLUME XXV

1929-1930-1931

PHILADELPHIA
PRINTED FOR THE ACADEMY
1932

NOTICE

The present volume has been prepared according to the plan adopted in 1929. The memoirs of the Fellows, deceased since the last publication, are reproduced. This volume is printed without cost to the Academy through the courtesy of the J. B. Lippincott Company, publishers of the ANNALS OF SURGERY.

CALVIN M. SMYTH, JR.
Recorder.

PRINTED IN THE UNITED STATES OF AMERICA

CONTENTS

CONSTITUTION AND BY-LAWS

(As Amended to 1929.)

CONSTITUTION

ARTICLE I

The name of the Society shall be "THE PHILADELPHIA ACADEMY OF SURGERY," and it shall consist of Fellows and Honorary Fellows.

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

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 IV

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 V

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

ARTICLE VI

The Candidate for admission as a Fellow must be a graduate of a reputable Medical School, not less than twenty-eight (28) years of age, practising his profession in the City of Philadelphia or within thirty miles of the City, and must have earned some reputation as a practitioner of Surgery, a Teacher, an Author, or an original Investigator.

ARTICLE VII

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 VIII

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

ARTICLE IX

NON-RESIDENT FELLOWS

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.

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

SECTION V

SECRETARY

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

SECTION VI

TREASURER

It shall be the duty of the Treasurer to receive all moneys and funds belonging to the Academy, unless otherwise provided for; he shall pay all bills when properly ordered at the instance of the Academy, collect all dues and assessments as promptly as possible, and present an annual account for audit.

SECTION VII

RECORDER

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

SECTION VIII

COUNCIL

The Council shall consist of six 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, and 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.

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 discussions; 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.

LIST OF OFFICERS, 1932

President

JOHN SPEESE, M.D.

Vice-Presidents

WALTER ESTELL LEE, M.D.
DAMON B. PFEIFFER, M.D.

Secretary

DEFOREST P. WILLARD, M.D.

Treasurer

WILLIAM B. SWARTLEY, M.D.

Recorder

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

Business Committee

EDWARD T. CROSSAN, M.D.
HENRY P. BROWN, JR., M.D.
With the Recorder

Council

HUBLEY R. OWEN, M.D.
GEORGE P. MULLER, M.D.
With the President, First Vice-President, Secretary and Treasurer

Trustees of the Samuel D. Gross Prize

WILLIAM J. TAYLOR, M.D.
JOHN H. JOPSON, M.D.
EDWARD B. HODGE, M.D.

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

VICE-PRESIDENTS

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

SECRETARY

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

CORRESPONDING SECRETARY

ELECTED
1880 THOMAS G. MORTON
Office abolished after 1889 by amendment to By-Laws.

TREASURER

ELECTED
1880 WILLIAM HUNT
1891 WILLIAM G. PORTER
1904 JAMES P. HUTCHINSON

ELECTED
1911 EDWARD B. HODGE
1920 DUNCAN L. DESPARD
1922 WILLIAM B. SWARTLEY

RECORDER

ELECTED
1880 JOHN B. ROBERTS
1881 DEFOREST P. WILLARD
1884 C. B. G. DENANCREDE
1884 J. EWING MEARS
1891 LEWIS W. STEINBACH

ELECTED
1902 JOHN H. GIBBON
1905 JOHN H. JOPSON
1915 JOHN SPEESE
1920 HENRY P. BROWN, JR.
1922 J. WILLIAM BRANSFIELD
1926 CALVIN M. SMYTH, JR.

LIBRARIAN

ELECTED
1880 OSCAR H. ALLIS
Office abolished after 1889 by amendment to By-Laws.

PATHOLOGICAL HISTOLOGIST

ELECTED
1880 SAMUEL W. GROSS
Office abolished after 1889 by amendment to By-Laws.

COUNCIL

ELECTED
1880 JOHN ASHHURST, JR.
1880 JOHN H. BRINTON
1894 WILLIAM B. HOPKINS
1895 HENRY R. WHARTON
1898 THOMAS R. NEILSON
1900 W. JOSEPH HEARN
1902 ROBERT G. LECONTE
1906 THOMAS R. NEILSON
1910 J. CHALMERS DACOSTA
1920 CHARLES F. MITCHELL

ELECTED
1922 GEORGE G. ROSS
1922 JAMES H. BALDWIN
1923 WILLIAM J. TAYLOR
1924 JOHN H. JOPSON
1924 JOHN SPEESE
1925 EDWARD B. HODGE
1926 DAMON B. PFEIFFER
1927 CHARLES F. MITCHELL
1930 ASTLEY P. C. ASHHURST
1930 HUBLEY R. OWEN

With President, Vice-President, Secretary and Treasurer.

PUBLICATION COMMITTEE

ELECTED
1880 JOHN H. PACKARD

ELECTED
1880 WILLIAM W. KEEN

With the Recorder.
Office abolished after 1894 by amendment to By-Laws.

BUSINESS COMMITTEE

ELECTED
1895 WILLIAM J. TAYLOR
1895 DEFOREST P. WILLARD
1896 RICHARD H. HARTE
1897 ROBERT G. LECONTE
1900 G. G. DAVIS
1902 JOHN H. JOPSON
1905 GEORGE G. ROSS
1908 FRANCIS T. STEWART
1914 JOHN SPEESE
1916 W. E. LEE
1916 MORRIS BOOTH MILLER

ELECTED
1917 DAMON B. PFEIFFER
1917 A. P. C. ASHHURST
1919 A. BRUCE GILL
1919 J. STEWART RODMAN
1920 ARTHUR BILLINGS
1922 DAMON B. PFEIFFER
1924 DEFOREST P. WILLARD
1928 WALTER E. LEE
1930 EDWARD T. CROSSAN
1930 JOHN B. FLICK
1931 HENRY P. BROWN, JR.

With the Recorder

COMMITTEE ON SAMUEL D. GROSS PRIZE FUND AND LIBRARY

1884–1891
D. HAYES AGNEW
SAMUEL W. GROSS
J. EWING MEARS
SAMUEL ASHHURST
WILLIAM HUNT

1892–1893
J. EWING MEARS
SAMUEL ASHHURST
WILLIAM HUNT
JOHN ASHHURST, JR.
WILLIAM W. KEEN

TRUSTEES OF THE SAMUEL D. GROSS PRIZE FUND AND LIBRARY

1894
J. EWING MEARS
JOHN ASHHURST, JR.
WILLIAM W. KEEN
With Samuel Ashhurst and William Hunt to serve with them on distribution of the 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 P. 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

ACTIVE FELLOWS OF THE PHILADELPHIA ACADEMY OF SURGERY

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., F.A.C.S., 2201 Chestnut Street. Professor of Clinical Surgery, University of Pennsylvania; Chief Surgeon (B Service) to the Episcopal Hospital; Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Colonel Auxiliary, United States Army.

1917. Baldwin, James Harvey, A.M., M.D., F.A.C.S., 1426 Pine Street. Surgeon to the Methodist Hospital.

1928. Bates, William, B.S., M.D., F.A.C.S., 2029 Pine Street. Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Babies' Hospital.

1922. Beltran, Basil R., A.M., M.D., F.A.C.S., 2109 Locust Street. Surgeon to the Misericordia Hospital; Assistant Professor of Surgery, Graduate School of Medicine; Assistant Surgeon to the Graduate Hospital of the University.

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

1898. Boger, John A., A.M., M.D., F.A.C.S., 2213 North Broad Street. Senior Surgeon to the Stetson Hospital.

1929. Bothe, Albert E., M.D., Chief Urologist, Misericordia Hospital; Director of Laboratory Surgical Pathology, University Hospital.

1928. Bothe, Frederick A., M.S., M.D., F.A.C.S., Medical Arts Building, Sixteenth and Walnut Streets. Assistant Surgeon to Presbyterian Hospital, Associate in Surgery, Post-Graduate School, University of Pennsylvania; Associate in Surgery, Children's Hospital; Surgeon to Stetson Hospital; Consulting Surgeon, Home for Incurables; Attending Surgeon to American Oncologic Hospital.

1921. Boykin, Irvine M., M.D., Elkins Park. Associate Surgeon to the Episcopal Hospital; Visiting Surgeon to the Abington Memorial Hospital; Instructor of Surgery, Undergraduate School, University of Pennsylvania; Instructor of Surgery, Post-Graduate School, University of Pennsylvania.

1921. Bransfield, John William, M.D., F.A.C.S., 2025 Walnut Street. Visiting Surgeon to St. Agnes', St. Vincent's and American Oncologic Hospitals.

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

1923. Buzby, B. Franklin, A.B., M.D., F.A.C.S., 414 Cooper Street, Camden, New Jersey. Orthopædist, Cooper Hospital, Camden, New Jersey; Consulting Orthopædist, Camden County Tuberculosis Sanitarium; Instructor in Surgery, University of Pennsylvania; Orthopædist, Burlington County Hospital; Assistant Surgeon, Philadelphia Orthopædic Hospital.

1907. Carmany, Harry S., M.D., F.A.C.S., 366 Green Lane, Roxborough. Surgeon to the Memorial Hospital, Roxborough; Associate Surgeon to the Episcopal Hospital; Surgeon to the Dispensary of the Episcopal Hospital.

1909. Carnett, John B., M.D., 2012 Spruce Street. Vice Dean and Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon to the Graduate Hospital and to the Radiological Department of the Philadelphia General Hospital.

1919. Crossam, Edward T., M.D., 257 South Sixteenth Street. Associate Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital; Instructor in Surgery, University of Pennsylvania.

1922. Davis, Warren B., M.D., F.A.C.S., 135 South Eighteenth Street. Associate in Surgery, Jefferson Medical College; Chief of Maxillo-Facial Clinic, Jefferson Hospital; Assistant Surgeon to the Frankford Hospital; Consulting Maxillo-Facial Surgeon, Kensington Hospital for Women; Consulting Otolaryngologist, St. Agnes' Hospital and Philadelphia Skin and Cancer Hospital.

1930. Deibert, Irvin E., M.D., F.A.C.S., 618 Benson Street, Camden, New Jersey. Instructor, Department of Operative Surgery, University of Pennsylvania School of Medicine; Surgeon to the Cooper Hospital; Attending Surgeon to the Camden County General Hospital and Camden County Hospital for Treatment of Tuberculosis, Lakeland.

1916. Dorrance, George Morris, M.D., F.A.C.S., 2025 Walnut Street. Surgeon to St. Agnes' Hospital; Consulting Oral Surgeon to the University Hospital and Philadelphia General Hospital; Professor Maxillo-Facial Surgery, Thomas Evans Institute, University of Pennsylvania; Surgeon to American Oncologic Hospital.

1928. Downs, T. McKean, M.D., 6013 Greene Street. Assistant Surgeon to the Pennsylvania, Germantown and Bryn Mawr Hospitals.

1921. Eliason, Eldridge Lyon, A.B., M.D., F.A.C.S., Sc.D., 326 South Nineteenth Street. Surgeon to the University of Pennsylvania, Philadelphia General, Mt. Sinai and Delaware County Hospitals; Professor of Clinical Surgery, University of Pennsylvania School of Medicine and University of Pennsylvania Graduate School of Medicine; Consulting Surgeon to the Lebanon Sanitarium, Burlington County Hospital and Kent General Hospital.

1909. Elmer, Walter G., B.S., M.D., F.A.C.S., 1801 Pine Street. Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Orthopædic Surgery, Woman's Medical College of Pennsylvania; Visiting Orthopædic Surgeon to the Philadelphia General Hospital, the Graduate Hospital of the University of Pennsylvania and the Hospital of the Woman's Medical College; Consulting Orthopædic Surgeon to the Jewish Hospital; Visiting Surgeon to the Pennsylvania Training School for Feeble-minded Children at Elwyn.

1929. Englerth, Louis D., M.D., F.R.C.S. (Edin.), F.A.C.S., 1710 Locust Street. Surgeon to the Frankford Hospital; Surgeon to St. Joseph's Hospital; Consulting Surgeon to Grandview Hospital, Sellersville, Pennsylvania.

1931. Ferguson, Lewis Kraeer, A.B., M.D., F.A.C.S., Associate in Surgery, University of Pennsylvania; Assistant Surgeon to the Hospital of the University of Pennsylvania and Philadelphia General Hospital.

1926. Flick, John B., M.D., 1608 Spruce Street. Associate in Surgery, Jefferson Medical College; Surgeon to the Pennsylvania Hospital; Assistant Surgeon to the Jefferson, Bryn Mawr and Philadelphia General Hospitals.

1931. Fleming, Bruce, M.D., 20th and Chestnut Streets. Assistant Surgeon to the Delaware County Hospital; Assistant Surgeon to the Memorial Hospital, Roxborough; Clinical Assistant in Surgery to the Jefferson Hospital.

1898. Frazier, Charles Harrison, M.D., F.A.C.S., Sc.D., 3400 Spruce Street, Room 700. John Rhea Barton, Professor of Surgery, University of Pennsylvania; Chief of Neurosurgical Service to the University Hospital.

1899. Gibbon, John H., M.D., 1608 Spruce Street. Emeritus Professor of Surgery, Jefferson Medical College; Consulting Surgeon to Jefferson, Pennsylvania and Bryn Mawr Hospitals.

1914. Gill, A. Bruce, A.B., M.D., 2001 Aldine Trust Building, 1930 Chestnut Street. Professor of Orthopædic Surgery, Medical School, University of Pennsylvania; Professor of Orthopædic Surgery,

Graduate School of Medicine, University of Pennsylvania; Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Orthopædic Surgeon to the Presbyterian Hospital; Chief Surgeon to the Widener Memorial Industrial School for Crippled Children.

1928. GILMOUR, WILLIAM R., A.B., M.A., M.D., Central Medical Building. Associate Professor of Operative Surgery and Surgical Anatomy, Women's Medical College; Instructor in Applied Anatomy, University of Pennsylvania; Attending Surgeon, Woman's College Hospital; Assistant Surgeon to the Methodist Episcopal Hospital and Northeastern Hospital.

1902. GIRVIN, JOHN H., M.D., F.A.C.S., 2120 Walnut Street. Gynæcologist to the Presbyterian Hospital; Associate Professor of Gynæcology, Graduate School of Medicine, University of Pennsylvania; Consulting Gynæcologist to the Home for Incurables.

1925. GRANT, FRANCIS CLARK, A.B., M.D., 133 South Thirty-sixth Street. Assistant Professor of Neurological Surgery, School of Medicine, University of Pennsylvania; Associate in Neurosurgery, Graduate School of Medicine, University of Pennsylvania; Consulting Neurosurgeon to the Children's Hospital; Visiting Neurological Surgeon to the Abington Memorial Hospital; Consulting Surgeon to the Chestnut Hill Hospital; Visiting Neurological Surgeon to the Philadelphia General Hospital.

1913. HEARN, WILLIAM P., M.D., F.A.C.S., 2119 Spruce Sreet. Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the Jefferson Hospital.

1922. HERMAN, J. LEON, B.S., M.D., Medical Arts Building, Sixteenth and Walnut Streets. Associate Surgeon to the Pennsylvania Hospital, in charge of Urology; Urologist to the Methodist Hospital.

1890. HEWSON, ADDINELL, A.B., M.D., F.A.C.S., 326 South Fifteenth Street. Professor of Anatomy and Histology, Temple University, Dental School.

1925. HINTON, DRURY, M.D., F.A.C.S., 1826 Pine Street. Instructor, Health Examinations, Department of Physical Education, University of Pennsylvania; Instructor in Physical Diagnosis and First Aid, Summer School; Visiting Chief Surgeon to the Delaware County Hospital; Adjunct Surgeon to Mt. Sinai Hospital; Visiting Surgeon to the Graduate Hospital.

1905. HODGE, EDWARD B., A.B., M.D., 2019 Spruce Street. Surgeon to the Presbyterian Hospital; Chief of Surgical Service to the Germantown Hospital; Chief Surgeon to the Chester County Hospital.

1898. HUTCHINSON, JAMES P., A.B., M.D., Media, Pennsylvania. Consulting Surgeon to the Pennsylvania Hospital and the Methodist Episcopal Hospital.

1915. IVY, ROBERT HENRY, M.D., DD.S., F.A.C.S., 1930 Chestnut Street. Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Chief of Maxillo-Facial Surgery to the Hospital of the Graduate School of Medicine; Consulting Plastic Surgeon to the Children's Hospital; Consultant in Maxillo-Facial Surgery to the Walter Reed General Hospital, Washington, D. C.

1922. JOHN, RUTHERFORD L., M.D., 256 South Twenty-first Street. Instructor in Surgery, University of Pennsylvania Medical School; Associate Surgeon (Orthopædic Service) to the Episcopal Hospital; Assistant Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Orthopædist, Chief of Orthopædic Dispensary, St. Christopher's Hospital for Children.

1915. JONES, JOHN F. X., B.S., A.M., M.D., F.A.C.S., 1737 Chestnut Street. Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals.

1900. JOPSON, JOHN HOWARD, M.D., F.A.C.S., 1824 Pine Street. Professor of Clinical Surgery, Medical Department, University of Pennsylvania; Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon to the Presbyterian Hospital and the Hospital of Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon to the Children's, Shriner's and Norristown State Hospitals, and Philadelphia Home for Incurables.

1914. KEENE, FLOYD E., M.D., F.A.C.S., 133 South Thirty-sixth Street. William Goodell Professor of Gynæcology, University of Pennsylvania; Gynæcologist to the University Hospital; Consulting Gynæcologist to Chestnut Hill, Bryn Mawr and Abington Memorial Hospitals.

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

1913. KLOPP, EDWARD J., M.D., F.A.C.S., 1611 Spruce Street. Professor of Surgery, Jefferson Medical College; Attending Surgeon to the Jefferson Hospital; Surgeon to the Pennsylvania Hospital and Chief of Out-Patient Clinic; Surgeon to the Delaware County and Memorial Hospitals.

1930. KNOX, HARRY E., M.D., 719 Sixty-sixth Avenue, Oak Lane. Chief Surgeon to St. Christopher's Hospital for Children; Chief Surgeon to the Philadelphia Hospital for Contagious Diseases; At-

tending Surgeon to the Germantown Hospital; Associate Surgeon to the Episcopal Hospital; Assistant Surgeon to Lankenau Hospital.

1914. Laws, George Malcolm, B.S., M.D., 1907 Spruce Street. Associate Gynæcologist to the Presbyterian Hospital; Assistant Professor of Gynæcology, Graduate School of Medicine, University of Pennsylvania.

1910. Lee, Walter Estell, M.D., F.A.C.S., 905 Pine Street. Professor of Surgery, Graduates School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr, Children's and Burlington County Hospitals and to the Hospital of the Graduate School of Medicine, University of Pennsylvania.

1926. Lipshutz, Benjamin, B.A., M.D., F.A.C.S., 1007 Spruce Street. Associate in Neuro-anatomy, Jefferson Medical College of Philadelphia; Surgeon to Mt. Sinai Hospital.

1900. Martin, Edward, A.M., M.D., F.A.C.S., Ph.D., LL.D., 135 South Eighteenth Street. Professor of Surgical Physiology, University of Pennsylvania.

1929. McCarthy, Patrick Andrew, M.D., Central Medical Building. Instructor in General Surgery and Anatomy, Jefferson Medical College of Philadelphia; Assistant Surgeon to the Philadelphia General Hospital; Staff, St. Mary's and Clark Hospitals; Assistant Surgeon, Out-Patient Department, Jefferson Medical College Hospital.

1931. Meade, R.H., Jr., M.D., 2116 Pine Street. Associate Surgeon to the Episcopal Hospital; Associate in Surgery, University of Pennsylvania Medical School.

1931. Mecray, Paul M., M.D., F.A.C.S., 405 Cooper Street, Camden, New Jersey. Chief Surgeon to the Cooper Hospital; Chief Surgeon to the Zurbrugg Memorial Hospital, Riverside, New Jersey; Consulting Surgeon to the Salem Memorial Hospital, Salem.

1917. Mencke, J. Bernhard, A.B., M.D., 1816 Spruce Street. Associate Surgeon to the Lankenau Hospital; Surgeon to the Northwestern General Hospital.

1915. Merrill, William Jackson, A.B., M.D., F.A.C.S., 2017 Spruce Street. Associate Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Children's, Jewish and Misericordia Hospitals, St. Edmond's Home for Crippled Children, Children's Seashore House, Atlantic City, Lock Haven Hospital, City Hospital, Williamsport, Pennsylvania and Geisinger Memorial Hospital, Danville, Pennsylvania.

1904. Mitchell, Charles F., M.D., 2003 Pine Street. Surgeon-in-Chief to the Pennsylvania, Germantown and Bryn Mawr Hospitals; Consulting Surgeon to Chestnut Hill and St. Christopher's Hospitals.

1906. Muller, George P., M.D., F.A.C.S., 1930 Spruce Street. Professor of Clinical Surgery, University of Pennsylvania School of Medicine; Surgeon to the University Hospital, Misericordia Hospital and the Mary J. Drexel Home; Consulting Surgeon to the Women's Hospital, Rush Hospital, White Haven Sanitarium and the Chestnut Hill Home for Consumptives.

1921. Murphy, Eugene C., M.D., F.A.C.S., 1841 South Broad Street. Surgeon to St. Agnes' 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. Surgeon to the Episcopal Hospital.

1905. Nassau, Charles F., M.D., LL.D., Sc.D., F.A.C.S., 1710 Locust Street. Clinical Professor of Surgery, Jefferson Medical College; Assistant Surgeon to the Jefferson Medical College Hospital; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Girard College; Consulting Surgeon to the Kensington Hospital for Women; Consulting Surgeon to the Pottstown Hospital, Pottstown, Pennsylvania.

1890. Neilson, Thomas R., A.M., M.D., F.A.C.S., 1937 Chestnut Street. Surgeon Emeritus to the Episcopal Hospital; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania; Consulting Surgeon to St. Christopher's Hospital for Children.

1921. Outerbridge, George W., A.B., M.D., 1927 Spruce Street. Gynæcologist to the Abington Memorial Hospital; Associate Gynæcologist to the Methodist Hospital.

1915. Owen, Hubley R., M.D., 319 South Sixteenth Street. Professor of Clinical Surgery, Woman's Medical College; Instructor in Surgery, Jefferson Medical College; Surgeon to the Philadelphia General Hospital; Attending Surgeon to the Woman's College Hospital; Assistant Surgeon to the Orthopædic Hospital; Chief Surgeon to the Bureaus of Police and Fire of Philadelphia.

1912. Pfeiffer, Damon B., A.B., M.D., F.A.C.S., 1822 Pine Street. Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon-in-Chief to the Abington Memorial and Lankenau Hospitals; Associate Surgeon to the Presbyterian Hospital.

1919. Piper, Edmund B., B.S., M.D., F.A.C.S., 1936 Spruce Street. Professor of Obstetrics, University of Pennsylvania; Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania.

1916. RANDALL, ALEXANDER, M.A., M.D., F.A.C.S., Medical Arts Building, Sixteenth and Walnut Streets, Professor of Urology, School of Medicine, University of Pennsylvania; Consulting Urologist to the Germantown Hospital; Urologist to the Chestnut Hill Hospital.

1924. RAVDIN, ISADOR S., B.S., M.D., Medical Laboratories, University of Pennsylvania. J. William White Professor of Research Surgery, University of Pennsylvania; Surgeon to the University Hospital.

1928. ROBBINS, FREDERICK ROSS, B.S., M.D., 258 South Eighteenth Street. Associate Surgeon to the Children's Hospital; Assistant Surgeon to the Pennsylvania Hospital, Graduate Hospital, University of Pennsylvania, and Bryn Mawr Hospital; Assistant in Surgery, Graduate School of Medicine, University of Pennsylvania.

1913. RODMAN, JOHN STEWART, M.D., F.A.C.S., Medical Arts Building, Sixteenth and Walnut Streets. Professor of Surgery, Woman's Medical College; Surgeon-in-Chief to the Woman's Medical College Hospital; Attending Surgeon to the Bryn Mawr Hospital; Associate Surgeon to the Presbyterian Hospital.

1928. ROTHSCHILD, NORMANN S., M.D., 235 South Fifteenth Street. Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon to the Graduate Hospital; Surgeon to the Northern Liberties Hospital.

1913. RUGH, JAMES TORRANCE, A.B., M.D., LL.D., Medical Arts Building, Sixteenth and Walnut Streets. James Edwards Professor of Orthopædic Surgery, Jefferson Medical College; Orthopædic Surgeon to the Jefferson Hospital, Methodist Episcopal Hospital, Philadelphia General Hospital, and Montgomery Hospital of Norristown; Consulting Orthopædic Surgeon to the West Philadelphia Hospital for Women, Pottstown Hospital, Pottstown, Pennsylvania, and to the New Jersey State Institution for Feebleminded and Epileptics, Vineland, New Jersey.

1930. RYAN, THOMAS J., M.D., F.A.C.S., 4500 Chestnut Street. Surgeon to the Misericordia Hospital.

1920. RYAN, WILLIAM JOHN, A.B., M.D., F.A.C.S., Medical Arts Building, Sixteenth and Walnut Streets. Attending Surgeon to St. Mary's Hospital; Attending Surgeon to St. Vincent's Hospital.

1929. SEELAUS, HENRY K., M.D., 3015 North Broad Street. Surgeon to St. Mary's Hospital; Assistant Surgeon to the Philadelphia General Hospital; Demonstrator of Clinical Surgery, Jefferson Medical College.

1922. SHALLOW, THOMAS A., M.D., F.A.C.S., 2045 Walnut Street. Professor of Surgery to the Jefferson Hospital; Neuro-Surgeon to the Philadelphia General Hospital.

1903. SITER, E. HOLLINGSWORTH, M.D., F.A.C.S., Medical Arts Building, Sixteenth and Walnut Streets. Visiting Genito-urinary Surgeon to the Philadelphia General Hospital; Associate in Genito-urinary Surgery, University of Pennsylvania; Surgeon-in-Charge, Genito-urinary Clinic, University of Pennsylvania Hospital.

1924. SMYTH, CALVIN M., JR., B.S., M.D., F.A.C.S., 2021 Spruce Street. Assistant Professor of Surgery, University of Pennsylvania, Graduate School of Medicine; Surgeon to the Methodist Episcopal and Abington Memorial Hospitals.

1909. SPEESE, JOHN, M.D., F.A.C.S., 1832 Spruce Street. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Children's and Presbyterian Hospitals.

1911. STELLWAGEN, THOMAS C., JR., M.D., 220 South Sixteenth Street. Professor of Urology, Jefferson Medical College and Urologist to the Jefferson Hospital.

1919. SWARTLEY, WILLIAM BLAINE, M.D., F.A.C.S., 6002 Greene Street, Germantown. Surgeon to the Germantown and Chestnut Hill Hospitals; Demonstrator of Anatomy, Jefferson Medical College.

1890. TAYLOR, WILLIAM J., M.D., 1825 Pine Street. Attending Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Surgeon to St. Agnes' Hospital, the West Philadelphia Hospital for Women and the Woman's Hospital.

1911. THOMAS, T. TURNER, M.D., F.A.C.S., 2009 Spruce Street. Chief Surgeon to the Northeastern Hospital.

1915. THOMAS, W. HERSEY, M.D., F.A.C.S., 903 Central Medical Building, Eighteenth and Chestnut Streets. Professor of Genito-urinary Surgery, Medical Department of Temple University; Genito-urinary Surgeon to the Temple University Hospital; Urologist to the Philadelphia General Hospital.

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

1928. WALKLING, ADOLPH A., M.D., 2808 Girard Avenue. Assistant Surgeon to the Pennsylvania Hospital; Chief, Surgical Out-Patient

Division B, Jefferson Hospital; Demonstrator Fracture Dressings, Jefferson Medical College.

1928. WEEDER, S. DANA, M.D., 6110 Greene Street, Germantown. Surgeon to the Germantown Hospital; Assistant Surgeon to the Chestnut Hill Hospital.

1902. WHITING, A. D., M.D., 333 South Eighteenth Street. Surgeon to the Lankenau Hospital; Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania.

1919. WILLARD, DEFOREST P., M.D., 1729 Spruce Street. Professor of Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon to the Graduate School, Orthopædic, Shriner's, Bryn Mawr, Chestnut Hill, Delaware County and Abington Memorial Hospitals.

1927. WILLIAMSON, ERNEST G., M.D., F.R.C.S. (Edin.), F.A.C.S., 319 South Sixteenth Street. Associate Surgeon to the Children's Hospital; Assistant Surgeon to the Presbyterian Hospital; Assistant Surgeon, Out Patient Department, Jefferson Hospital.

1898. WOOD, ALFRED CONARD, M.D., F.A.C.S., 2035 Walnut Street. Assistant Professor of Surgery, University of Pennsylvania; Surgeon to the Howard and Philadelphia General Hospitals.

NON-RESIDENT MEMBERS

1908. SWEET, J. E., A.M., M.D., Sc.D., F.A.C.S., Cornell University Medical School, New York City. Professor of Surgical Research, Cornell University, School of Medicine.

1923. WELLS, J. RALSTON, M.D., Woolworth Building, Daytona Beach, Florida. Visiting Surgeon to the Halifax District Hospital, Daytona Beach, Florida.

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.....1900 or 1901.	
1881.	MOSES GUNN, Chicago, Illinois.....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, Massachusetts.	1927.
1881.	W. T. BRIGGS, Nashville, Tennessee.....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, Illinois.....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.	
1900.	W. H. A. JACOBSON, London, England.	
1900.	THEODORE KOCHER, Berne, Switzerland....July	27, 1917.

ELECTED		DIED
1900.	Vincenz Czerny, Heidelberg, Germany....	October 3, 1916.
1906.	William J. Mayo, Rochester, Minn.	
1906.	Dudley P. Allen, Cleveland, Ohio.....	January 6, 1915.
1906.	Robert Abbe, New York, N. Y.	1928.
1906.	C. B. G. deNancrede, Ann Arbor, Mich...	May 6, 1921.
1907.	John C. Munro, Boston, Mass.....	December 6, 1910.
1908.	J. Ewing Mears, Philadelphia, Pa.....	May 28, 1919.
1909.	Lewis Stephen Pilcher, Brooklyn, N. Y.	
1916.	W. W. Keen, Philadelphia, Pa.	
1920.	Henry R. Wharton, Philadelphia, Pa.....	December 1925.
1927.	John Chalmers DaCosta, Philadelphia, Pa.	
1929.	D'Arcy Power, London, England.	
1929.	Albin Lambotte, Esneux, Belgium.	
1929.	Henri Hartmann, Paris, France.	
1929.	Th. Tuffier, Paris, France.	
1929.	Joseph Guyot, Bordeaux, France.	
1929.	Georges Jeanneney, Bordeaux, France.	
1929.	F. de Quervain, Berne, Switzerland.	
1929.	Berkeley Moynihan, Leeds, England.	
1929.	Harvey Cushing, Boston, Mass.	
1929.	Edward W. Archibald, Montreal, Canada.	
1929.	John M. T. Finney, Baltimore, Md.	
1929.	Evarts Graham, St. Louis, Mo.	
1929.	Ellsworth Eliot, Jr., New York City, N. Y.	
1929.	Rudolph Matas, New Orleans, La.	
1929.	Dean D. Lewis, Baltimore, Md.	
1929.	Eugene H. Pool, New York City, N. Y.	
1929.	George W. Crile, Cleveland, Ohio.	
1929.	Edward Starr Judd, Rochester, Minn.	
1929.	Dallas B. Phemister, Chicago, Ill.	

FELLOWS DECEASED SINCE LAST PUBLICATION

DIED	
1930.	Benjamin Abraham Thomas
1930.	Emory Graham Alexander
1930.	Hiram Rittenhouse Loux
1931.	Harry Clay Deaver
1931.	John Blair Deaver

BENJAMIN ABRAHAM THOMAS

On the morning of May 29, 1930, the medical profession of Philadelphia was grieved by the announcement of the death of Dr. Benjamin Abraham Thomas. It had been well known that for several months Doctor Thomas had been ill, but the latest news from his bedside had been most encouraging and his recovery seemed assured. The announcement was therefore doubly a shock. Stricken with bronchopneumonia in the early Spring, he made a tardy but satisfactory convalescence, which was most unexpectedly interrupted by a severe hæmatemesis and in his weakened condition it seriously delayed his return of strength and subjected his vitality to an easy invasion of further complications. On the morning of his death he apparently suffered from an embolism of the artery of the foot and a short time following, a second embolus caused such a severe vascular shock that death ensued. This was the fifty-second year of his life.

Doctor Thomas was born at Valley Forge, November 7, 1878, the son of William B. Thomas and Mary J. (Abraham) Thomas. Raised in the Society of Friends, his preliminary training was at the Friends Central School from whence he entered Swarthmore College and received the Degree of Bachelor of Arts in 1899. Entering the Medical School of the University of Pennsylvania the following Fall, he graduated therefrom in 1903 with the Degree of Doctor of Medicine. Five years later in 1908, his Collegiate Alma Mater (Swarthmore) conferred upon him an Honorary Degree of Master of Arts, basing the same upon his earnest post-graduate work and the post-graduate studies that Doctor Thomas had pursued abroad.

Doctor Thomas was probably the first to enter practice in Philadelphia as a trained urologist. There had been others before him of course in this special field, but they were men who from personal interest devoted most of their time and surgical endeavor to genito-urinary diseases, or others who had adopted this work from the beginning, had taught themselves, and had gained recognition by long and varied experience. In contrast, Doctor Thomas came to Philadelphia a young man interested in the specialty, who had studied with the idea of limiting his work thereto, and who on completing his interneship went abroad and studied cystoscopy, pathology and diagnosis under the recognized masters at Vienna, so that on his return he started active practice a trained man. From then on he never wavered in his interest or advancement, and we who followed benefited by his steadfastness of purpose.

In order to hold his affiliation to surgery and his Alma Mater, Doctor Thomas worked for a number of years in the Surgical Dispensary of the University Hospital and when he resigned had been Chief of this Out-Patient Clinic for several years. In this interval he likewise administered

the Genito-Urinary Dispensary of the Polyclinic Hospital and when the latter became the nucleus of the Graduate School, it found him ready to head the Department of Urology as the Professor, to which was added subsequently the duties of Vice Dean. Further honor accrued to Doctor Thomas with the appointment as Chief of the Urological Service of the Presbyterian Hospital, and Consultant to the Milford Hospital, the Philadelphia Prison and the Pennsylvania Railroad.

Doctor Thomas was always a prolific writer and his contributions to urologic literature are numerous. He became the co-author with Dr. Edward Martin in re-editing White and Martin's textbook on *Genito-urinary Surgery, Venereal Diseases and Applied Immunology*, and collaborated with the editors in publishing *Diseases of Mid-Life*.

He was a member of the County and State Medical Societies, the American Medical Association, Philadelphia College of Physicians, American College of Surgeons, and this Academy of Surgery.

Four years ago he was Chairman of the Urological Section of the American Medical Association and three years ago initiated the organization of a Urologic Section in our Pennsylvania State Medical Society, and to insure its success, became its Secretary for the past three years and was to be its Chairman at this year's meeting.

A member of the American Urological Association since 1914 it was the exception that saw Doctor Thomas miss its annual meeting, and the writer, probably more than anyone else, was allowed to realize how widely he was known, and genuinely he was admired, when his untimely death cut off his attendance this year just one week before the annual meeting in New York City. It was then demonstrated by the innumerable inquiries that were made, and the sincere regrets that were expressed, how Doctor Thomas, through his work and the distribution of his graduate students, had made a country-wide acquaintance that was ever alive and constantly enlarging.

It was as one of the Founders of the Philadelphia Urological Society that Doctor Thomas will be long remembered by his local colleagues, for early in the existence of this society he foresaw the future more brilliantly than the rest of us, and through his own generosity deposited in a Philadelphia bank a sum of money to endow an annual lecture. Such has been regularly given and Philadelphia medicine has been enriched by the visits of recognized authorities. It was Doctor Thomas' wish that this lectureship should be unnamed until after his death, so that today, though deprived of his companionship, his inspiration and his advice, it will become our pleasant duty to perpetuate his memory and to yearly recall to mind his generosity and interest in urological surgery by being able to establish in perpetuity the Benjamin A. Thomas Lecture of the Philadelphia Urological Society.

Alexander Randall.

EMORY GRAHAM ALEXANDER

Emory Graham Alexander was born in Charlotte, North Carolina, October 1, 1880. He came from a family of distinction, one of his ancestors having been Secretary of State of the United States, while his father served for many years as a Congressman from North Carolina and was credited with being the father of the good roads movement in that state. His early schooling was received in the public schools of Charlotte and his collegiate education in the University of North Carolina, where he completed the first two years of his medical course. He entered Jefferson Medical College in this city in his junior year and was graduated from that institution. He took his interneship in the Episcopal Hospital of Philadelphia, which he completed in January, 1907. The same year he began practice in this city and from that time enjoyed a peculiarly close personal and professional relationship with Dr. Harry C. Deaver. He later married Miss Harriet Deaver, daughter of Dr. John B. Deaver, who with a son and daughter survive him.

Opportunity favored Doctor Alexander and during his career he occupied many important positions which brought him a wide and varied surgical experience. He served for years in the Out-Patient Department of the Episcopal Hospital and as Registrar of the hospital. During the World War, he went abroad with the Episcopal Hospital Unit and acted as Chief of the Surgical Service with the rank of Major for a number of months. He was later promoted to Colonel. On his return he was made Surgeon to the Episcopal Hospital. For several years he was Professor of Clinical Surgery in the Woman's Medical College. Other positions which came to him were Surgeon to St. Christopher's Hospital, to the Children's Hospital of the Mary J. Drexel Home, to the Abington Hospital and to the Philadelphia Hospital for Contagious Diseases. For several years also he had been Assistant Professor of Surgery in the University of Pennsylvania.

He was a member of a number of medical societies, notably of this Academy, to which he was elected in 1910, also the County, State and American Medical Associations, the College of Physicians and the American Surgical Association.

Doctor Alexander was an excellent surgeon, of experience and sound practical judgment. He was an agreeable colleague and a pleasant companion. Life had been kind to him and he returned in kind. Neither his environment, his achievements, nor his prospects could justly have yielded him any other feeling than one of satisfaction for the present and optimism for the future. He had the misfortune to have a chronic digestive disturbance which at one time had manifested itself by an attack of catarrhal jaundice. Whether the annoyance of this condition with attendant chronic

toxæmia, together with the pressure of a busy life and increasing responsibilities, whether the strain of surgery, to which William J. Mayo has recently called attention in reference to its effects upon the heart, in the case of Doctor Alexander found a point of lessened resistance in his temperament, will never be known. His tragic end in a sudden fit of depression was a shock to all who knew him.

Dámon B. Pfeiffer.

HIRAM R. LOUX

Hiram R. Loux was born in Bucks County, Pennsylvania, July 16, 1859. His preliminary education was obtained in the local schools of the county. He then entered Washington Hall at Trappe, Pennsylvania, from which institution he graduated and subsequently taught in the schools of Montgomery and Bucks counties.

His governing ambition had always been to become a surgeon. He fulfilled this primal desire of his life to his own satisfaction and the love and esteem of the ones to whom he ministered.

He entered upon his medical career as a student in the class of 1879 in the Jefferson Medical College from which he received his degree after completion of a three-year course in 1882. He acquitted himself with distinction and honors during his collegiate work and was the recipient of the J. M. DaCosta prize in medicine. He entered upon the practice of general medicine at Souderton, Pennsylvania, where he successively gained favor and a lucrative practice that occupied him for ten years.

He then moved to the larger professional sphere of Philadelphia and was appointed a member of the surgical staff of the Jefferson Hospital under Dr. W. W. Keen, who was largely responsible for his coming to this city. He was appointed a Demonstrator of Surgery in the Jefferson Medical College. During this period of his life he taught fractures and fracture dressings. I well recall the eminently practical methods he advocated and the clear cut demonstrations and explanations offered in his course of lectures. The great example this man stood for was practical application of sound surgical principles. This was his surgical philosophy throughout his life. I am sure it accounted for much of his brilliant success. At all times he was an eminently sound thinker and his judgment consequently was untrammeled by immature surgical principles and procedures. Much of his success in practice, we believe, was due to the fact that he was primarily a well-trained and practical general physician. From this environment he passed to the field of general surgery wherein his basic training was equally thorough. When the chair of Genito-Urinary Surgery was founded in Jefferson he became Chief Clinical Assistant. His success in his chosen specialty was assured because of his preliminary training and thorough foundation gained in general practice.

He was successively appointed assistant Genito-Urinary Surgeon to the Jefferson Hospital and Assistant Professor of Genito-Urinary Surgery of the College. When the chair of Genito-Urinary Surgery was made vacant by the resignation of Dr. Orville Horwitz, Doctor Loux was made full Professor which position he occupied until his death on February 28, 1930. The staff of the Philadelphia General Hospital honored him by the appoint-

ment as Assistant Surgeon and later as an Attending Surgeon. For many years he was the Senior Surgeon of the staff.

To have been intimately associated with him was a great privilege and one that will be revered for many reasons. He was at all times the kindly and considerate doctor whom one naturally appealed to in times of trial and stress. I have seen him sorely tried both within and without the operating room by the emergencies of life and death but never did I witness a scintilla of harshness or a lack of consideration for his patient and those with whom he worked. He was at all times and in all ways an advocate of the greatest of religions—The Golden Rule. He presented many papers upon general surgical topics. His efforts in behalf of Genito-Urinary Surgery were always practical and based upon sound principles of diagnosis and technic which was his surgical religion.

He was a Fellow of the American Medical Association, the Philadelphia College of Physicians, the American College of Surgeons, the American Urological Association, the Philadelphia Academy of Surgery, the Pennsylvania State Medical Society and numerous other scientific bodies. With the passing of Hiram R. Loux, Philadelphia surgery and his Alma Mater suffered the loss of one of its soundest and sincerest surgical thinkers. We know that his colleagues and the younger men whom he taught and directed will always hark back and remember him as a teacher, a surgeon, and a Christian gentleman, worthy of love, esteem and respect.

Thomas C. Stellwagen

HARRY CLAY DEAVER

Harry Clay Deaver was born July 23, 1861, in Buck, Lancaster County, Pennsylvania. His father was a general practitioner covering a large territory about this small crossroads hamlet. He had three brothers, two of whom studied medicine. The oldest, Richard, became a successful general practitioner in Germantown, Pennsylvania. John B. Deaver became an internationally known surgeon.

Dr. Harry Deaver was graduated from the University of Pennsylvania in 1885 and served as Resident Physician in the Episcopal Hospital, Philadelphia, from April 1, 1886, to December 1, 1887. He was at once elected Dispensary Surgeon to that institution and in October, 1892, was made Visiting Surgeon, which position he held until his death. In 1892 he was also made Attending Surgeon to St. Christopher's Hospital for Children, holding this position until 1908 when he retired to become Consultant. He served also as Surgeon-in-Chief of the Children's Hospital of the Mary J. Drexel Home from 1901–1931. From 1909–1922 he was Professor of the Principles and Practice of Surgery and Clinical Surgery of the Woman's Medical College of Pennsylvania. He became a Fellow of the American Surgical Association in 1912. Doctor Deaver, while not so widely known as his brother John, enjoyed a very considerable local reputation and carried on a large practice throughout his life. He always kept a small proportion of general practice, chiefly among friends whom he had accumulated in the early period of his career. He liked these contacts and his brother John always maintained that it made him a better surgeon. However, the bulk of his work was surgical and was characterized by sound judgment, conservatism and thorough mastery of surgical technic. Had he chosen to write and speak to a larger public, there is no doubt that he could have achieved wide reputation. He preferred, however, a more secluded life and the well-merited confidence of his associates which he enjoyed. He died June 25, 1931, at his home in Wyncote, Pennsylvania, from cardiorenal complications following an attack of influenza.

Damon B. Pfeiffer

JOHN BLAIR DEAVER

John Blair Deaver was born near Buck, Lancaster County, Pennsylvania, July 25, 1855. He belonged to a medical family. His father was a physician and two of his brothers studied medicine. His older brother Richard was a successful practitioner in Germantown and his younger brother Harry became an active and well-known surgeon and a member for many years of this Academy. His son, Dr. J. Montgomery Deaver, has chosen surgery for his career.

Doctor Deaver's early education was received at the Nottingham Academy near his childhood home. He studied medicine at the University of Pennsylvania, being graduated in 1878. He served as interne in the Germantown Hospital and for an additional year in the Children's Hospital. He then began active practice in 1880 with an office at Sixteenth and Vine Streets. Shortly afterwards he was appointed Assistant Demonstrator of Anatomy in the University of Pennsylvania, which title he held for three years. The following years (1883–1884) he was Demonstrator of Osteology and Syndesmology. Then for three years, Demonstrator of Anatomy (1884–1887). Then Demonstrator of Anatomy and Lecturer on Topographical Anatomy (1887–1891) and Assistant Professor of Applied Anatomy (1891–1899).

At that period Anatomy was the recognized stepping stone into Surgery and Doctor Deaver made full use of his opportunities to become a profound surgical anatomist and an expert technician. Few indeed have been his equal and it is doubtful if any have been his superior in the skillful use of what he was fond of calling the "aseptic scalpel."

During this early period he made many connections in various capacities, mostly surgical, with Philadelphia hospitals, but his most important appointment came in 1886 when at the age of thirty-one he was made Chief Surgeon of the German Hospital, renamed during the World War, the Lankenau, after its great benefactor. Here Doctor Deaver found ample scope for his great energies. He rapidly built up a large surgical clinic and flung himself into the pioneering work of popularizing the new-found resources of surgical art. Instead of a last resort, always hateful and too often ineffective, surgery had to be made the first thought and early resort in order to save life and suffering in many emergencies. Especially was this true in appendicitis, and he rallied to the side of Fitz, McBurney, Senn and Murphy in teaching and urging early recognition and surgical treatment. His was a mind impatient of vacillating debate and hesitation. He cut quickly through to the essentials and took his stand. His great ability lay in making the impression which he desired. He had a whimsical humor before a medical audience which softened asperity of utterance. It came about that he exercised an influence upon medical practitioners second to none and though he saved many lives by his skillful surgery, his clear-cut teaching and advice saved far more. His

was a picturesque and colorful personality, the many sides of which would tax biographic skill and exceed the limits of this brief memoir. It should be recorded that he returned to the teaching staff of the University of Pennsylvania in 1911 as Professor of the Practice of Surgery and in 1918 was made head of this department as John Rhea Barton Professor of Surgery. In 1922 he was made Emeritus Professor of Surgery and retired from active teaching of undergraduates. He was also Professor of Surgery in the Graduate School of Medicine of the University of Pennsylvania from March, 1918, until July, 1931, when he was made Emeritus Professor in that school.

Always busy with surgery and clinical work which he loved, he was never fond of organization activities. He believed in organization, however, and was a member of many of the regular medical societies. He was elected to membership in this Academy in 1890. He was a member of the American Surgical Association and the International Surgical Society. He served as President of the American College of Surgeons and the Interstate Medical Association. Time does not permit even a recapitulation of his many memberships and activities nor of the numerous contributions to literature under his name. Truly he lived a full, successful and honorable life and one which must have been eminently satisfactory to himself.

He continued actively the practice of surgery and directorship of the surgical department of the Lankenau Hospital until the Summer of 1931 when failing health, due to progressive anæmia of unknown cause, made it impossible for him to continue. He died at his summer home in Wyncote, Pennsylvania, September 25, 1931.

DAMON B. PFEIFFER

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 Arterovenous Communications."—Dr. Emile Holman of Stanford University, San Francisco, California.

* 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.

ANNUAL ADDRESS FOR 1929

LYMPH EXUDATE AND FIBROUS TISSUE *

By Edward T. Crossan, M.D.

of Philadelphia, Pa.

associate surgeon, episcopal hospital

"The artless songs I sing do not deal with anything new or never said before."

These words of Rudyard Kipling are particularly fitting to the topic for this annual address. As the results of inflammation, lymph exudate and fibrous tissue remain today almost as great problems for the surgeon as they were when the processes of inflammation were described by Cohnheim in 1877. Nor do I have any new tales to add to those already told. The object of the discourse is to consider in detail the surgical application of the tales heard before about these tissues.

In surgery, fibrous tissue is an expression of recovery or it is the termination of irritation. To the patient, this scar tissue bears a sort of political relationship, for there are occasions when it is a mighty factor as an ally in the restoration of health, and, just as frequently, it can be found as a determined irresistible opponent to the restoration of function. Without this tissue, surgery would be impossible, and because of it surgery often is impossible.

As a process of repair and a medium for reconstruction, fibrous tissue is indispensable. Every wound, regardless of whether it is closed surgically or heals by granulation, depends upon fibrous tissue for the restitution of its structures or its coverings; in some wounds it provides strength, and in others it furnishes a scaffold for the growing epithelium or endothelium. For the restoration of function, fibrous tissue at times gives up its identity entirely, as, for example, in reconstruction operations fibrous tissue may become converted into cartilage, or in tenorrhaphies it takes on the shape of tendon. Then, too, in other reconstruction operations, fibrous tissue in the form of living sutures reproduces itself to restore anatomy.

Even in its disabling manifestations, fibrous tissue has had its inception for a beneficent purpose. Only in exceptional instances, as in keloid and certain cases of abdominal adhesions, have the reparative forces of the body run wild without undue cause. Many of the other deformities represent the body's efforts to repair destroyed tissue, and we have come to look upon these deformities as the toll the patient must pay for a fibrous tissue reaction to an excessive irritation. It is even possible that, after infections, some surgeons look upon the disabling fibrous tissue as a tribute to their art, in other words, the deformity to them is a proof of a violent contamination which has been cured by surgical procedures.

* The Annual Address before the Philadelphia Academy of Surgery, May 5, 1930.

To cure or improve is the primary object of surgery, but to cure or improve without deformity and disability is the ideal of surgery. Lymph exudate is a vital element in all surgical cures, both as a defensive mechanism and as the progenitor of fibrous tissue. Thus the problem is to control lymph exudate so that there may be cures with fibrous tissue as reparative as in the closure of wounds or in reconstructions. Viewed in this light, we may discover that many deformities are not tributes to surgery, and we will observe that a combination of a woman's touch and other qualities are not the only attributes of a good surgeon. Furthermore, if the surgeon appreciates the qualities of lymph exudate and fibrous tissue, he will understand how to deal with those deformities which have not or cannot be controlled.

My first practical lesson in lymph exudate occurred several years ago, and, though I have mentioned the instance at a previous meeting of this Academy, a repetition of the details still contains a lesson. The patient, a young girl, had weathered an attack of peritonitis of appendiceal origin, only to be faced a few days later by probable death from intestinal obstruction. In spite of morphine, lavage, and abstention from even liquids, the symptoms progressed. I reopened the abdomen and found coils of small intestine matted together in the pelvis and in the right iliac fossa by silvery membranes of exudate. Very carefully and thoroughly, I separated the films, saw the intestines resume their size and closed the abdomen with an absolute confidence that the obstruction was controlled. On the day following the second operation the patient again developed obstruction, on account of which I was forced to open the abdomen a third time. At the third operation, the intestinal coils were found matted together once more, but this time I showed better sense and performed an enterostomy, as the result of which the patient entirely recovered. My error at the second operation consisted in judging those membranes only as mechanical barriers, that is, I failed to think in terms of lymph exudate.

Lymph exudate is the body's first reaction to irritation, be it physical, chemical or bacterial. In the case just cited, if I had remembered this fact, I could have prognosticated the formation of more lymph exudate because the digital separation of the membranes was just another irritant. There is no operation, nor any infection which does not irritate the tissue, and in turn the tissues neutralize the irritant and repair the damage by the formation of lymph exudate. This defensive and reparative mechanism is not alone the pathologist's problem; surgical neglect of it is equivalent to thinking that sutures heal a wound or that drains and antiseptics cure infections, in short, it is the practice of mechanical surgery. The disciples of this school, not so many years ago, in peritoneal infections, were tearing away this defensive mechanism, the membrane which, as Boyd and others have said, "is more offensive to the surgeon's eye than to the patient's tissues." Nor is it necessary to carry on experiments to confirm this statement; a study of the components of the exudate will afford ample corroboration.

What are the constituents of the local reaction to irritation? Leucocytes, yes, leucocytes, and serum, fibrin, macrophages and monocytes—each a separate element but all acting in concert. The leucocytes phagocytose and liberate thrombin. The serum through the antitryptic element prevents any digestion of the tissues that would furnish nutriment to bacteria; also, serum brings opsonins, agglutinins, precipitins and fibrinogen, the latter to unite with thrombin for the formation of fibrin. Fibrin offers a foothold for the leucocytes, it shuts off absorption through lymphatics and, figuratively speaking, it ties the bacteria into knots. When the storm of irritation has passed away, the fibrin remains as a focus for the macrophages, leucocytes and monocytes to nourish and produce fibroblasts; in aseptic irritation Hertzler believes that the fibrin is actually converted into fibrous tissue. Taking infection as a specific example of irritation, we find that the exudate starves the bacteria, impedes their action or destroys them and prevents their toxins from entering into the circulation. What can a surgeon hope to add to such a defense? Obviously, only those measures which will nurture and stimulate the exudate.

However, the common practice of treating infections is based on an utter disregard for fibrin, leucocytes and serum. Pus to most surgeons seems to be an indication that the body defenses have broken down. Here, they say, we will destroy these bacteria by antiseptics, by frequent change of drains or by irrigation. What else do they destroy? Naturally, the defense mechanism, because anything that will kill the bacteria must also kill the white blood cells. If chemicals would kill organisms, one could afford to neglect the exudate. Does an antiseptic sterilize an infected wound?

Sir Almoth Wright has said, "If ever an antiseptic sterilized a heavily infected wound, that would deserve to be announced in all the evening and morning newspapers." Wright's inferences can be confirmed by daily visits to any hospital at the dressing hour. You will see pus pour forth from the same infected wounds day after day in spite of the use of varied antiseptics widely advertised as having a high phenol coefficient. If antiseptics are of value, it would seem reasonable that the abscess cavity should be sterilized when the incision is made, yet rarely is this done. Both Wright and Fleming have shown by experiments on wounds and in test tubes that (1) antiseptics cannot reach the bacteria in the tissue spaces; (2) antiseptics are rapidly diluted by the tissue juices to a point where they are no longer lethal for bacteria; and (3) antiseptics will not act in the presence of sloughs, and have very little action in serous discharges or in the presence of blood.

Antiseptics in infected wounds are not only useless but they are harmful. The destruction of leucocytes by the chemicals is not theoretical. Fleming, in 1919, showed that, with most of the antiseptics then in vogue, leucocytes were destroyed or their emigration inhibited. Fleming also demonstrated that some antiseptics actually increased the growth of bacteria, probably because the trypsin from the dead leucocytes encouraged digestion. The cure of an infection by the use of antiseptics is more probably

due to a generous incision than to a destruction of bacteria. It is possible that more recoveries and less loss of tissues from infections might be procured if surgical procedures did not destroy the defensive exudate.

Since the destruction of bacteria is not feasible, let us return to the proposition that protection and stimulation of the exudate are the rational procedures. Incision, drainage and infrequent change of drains—these measures are the only additions that a surgeon can hope to add to the body defenses. This passive method Wright has called the physiological treatment. Fleming has shown that there is one exception to this rule of treatment, namely, in the flat infected wounds. In this variety of infection a preliminary irrigation by hypertonic salt followed by eusol sterilizes the wound; the salt solution washes away the albuminous material and establishes a watery medium, the best medium for the action of antiseptics; the character of the wound permits the eusol to reach every crevice.

For years, surgeons have been forced to follow the physiological line of treatment in infections of the peritoneum. It is admitted by even the champions of antiseptics that no solution can reach all the recesses within the abdomen. No surgeon would think of using an irritant within the abdomen for fear of destroying endothelium, thereby increasing the absorption of toxins. Morphine, Fowler's position and nothing by mouth might cure some cases of peritonitis, but not many. Passive resistance by incision and undisturbed drainage, in conjunction with the measures noted, gives a high percentage of cures, if the treatment be inaugurated before the body has been overwhelmed by toxins. Here, in one of the most sensitive tissues of the body, are afforded daily examples of the efficacy of the passive treatment of infections.

Passive is not an exact qualifying term to apply to the physiological treatment because the method is actually stimulating and irritating to the lymph exudate. The fundamentals of the treatment, the incision and drainage, are more than mere mechanical procedures. The incision relieves pressure (Devine calls it detensionizing), and there is a resultant transudation of serum, an emigration of leucocytes and a backwash from the lymphatics. The drain, though it is inserted as a means for the egress of pus, also actually stimulates the formation of an additional defense. Let us consider in detail the subject of drains and their relation to lymph exudate.

Paradoxical as it may seem, the excretions from many a drained wound, excepting the discharges of the first twenty-four hours, actually are made possible by the closure of the drain with lymph exudate. Remove from your mind entirely the thought that the drain conveys pus and instead consider the drain in its true nature, a foreign body. Any foreign body introduced into a tissue space is an irritant which Nature in a very few hours surrounds by lymph exudate. If the entire circumference of the drain be in contact with tissue there is formed a channel through which pus reaches the surface. Consequently, lymph exudate can make of a drain, to use the words of Marion Sims, "a plug to keep the wound open." Of course, these statements, and those which follow, do not apply when fibrin actually penetrates the substance of the drain.

Now, let us revert to the thought that a drain creates an additional defense; that is, it does something more than prevent pus accumulation. In order for you to get a clear conception of this function, think first of an infection and picture to yourself the central cavity of pus surrounded by Nature's barrier of fibrin, leucocytes, *etc.* Introduce into that pus cavity a drain and then recall to your mind how the drain is excluded by lymph exudate. Now, you should have a mental picture of a wall within a wall or of a lymph exudate ring within a lymph exudate ring. Thus, the drain has been the cause for more fibrin and thereby the lymphatics have been more effectively occluded. Also, the more fibrin there is, the greater will be the surface for leucocyte attachment, consequently the more effective will be the phagocytosis, because leucocytes must be fixed to phagocytose. The addition of fibrin and leucocytes are not the only stimulants to the defense by the drain; there is also a constant out-pouring of serum as long as the drain is in the wound, a phenomenon that Horsley has termed a reversal of the lymphatic circulation. Arguments as to the relative merits of a drain are mere academic exercises when compared to a detailed knowledge of the relation of the drain to the lymph exudate.

Some one has said that it is easier to act than to think, a saying that is pertinent to the subject of drains and lymph exudate. For years and years drains have been pulled out of infected wounds daily without a single thought being given to the fact that fibrin and leucocytes were being destroyed. Mutilation of this defensive barrier results not only in an increased absorption of toxins, but also in a re-activation of the infection. A re-introduction of the drain, it is true, will erect another barrier; also, the re-introduction will kill more leucocytes and destroy more fibrin. The sum of the deleterious effects of pulling out the drain and putting it back will probably be greater than the beneficent results established by Nature in the interim between dressings.

Don't disturb the drain unless absolutely necessary, as has been mentioned, is one of the principles of the physiological treatment of infections. The practice of frequent change of drains is based on the fear that excretions will be dammed up by plugging. Every drain does become plugged, but, if the incision be extensive enough, there is no accumulation of pus; there usually is an increase of excretion through the attempts of the body to extrude the foreign object. If you have ever left packing in a wound for five or six days, or if you have ever seen a forgotten sponge removed from an abscess cavity you know that the foreign body was soft and mushy when removed, and you know that there usually was a profuse discharge until the object was taken out. In peritonitis cases drains are not disturbed and yet there seldom is any accumulation of pus as a result of plugging of the drains. Frequent removal of drains is unwarranted and is just as destructive to lymph exudate as is the use of antiseptics. When the penetration of the

fibroblasts into the lymph exudate imparts a rigidity to the channel, the drain can be changed occasionally or removed gradually without reactivation of the infection and without the production of excessive fibrous tissue.

Finally, in this discussion of drains and lymph exudate, there remains the topic of prophylactic use of drainage in the abdomen. Introduction of a drain to prevent dissemination of leakage is good surgery provided the surgeon realizes that the drain may actually encourage leakage. A drain introduced near the site of an anastomosis will build up additional lymph exudate; it will increase the sogginess at the stoma and the sutures may pull out. For the same reason, in the closure of perforated ulcers, if the subphrenic space is to be drained, be sure that the drain does not pass near the closure; neglect of this detail may be a cause for an abscess in the area that is being drained. If one is to judge from the literature, this principle has been entirely forgotten in the discussion of the merits of ideal cholecystectomy. Bile on the dressings after cholecystectomy with drainage may as well be an argument against the drain as it is in favor of it, in other words, the drain might have been the cause for the cutting through of the ligature. This subject cannot be dismissed without some mention about the relation of prophylactic drainage to adhesions, and it is in cholecystectomy that the topic has been given considerable prominence. Clinical experience has led me to believe that a drain within the abdomen for forty-eight hours, the period of our use of the prophylactic drain, never causes symptoms. Theoretically, the statement is perfectly plausible, because the lymph exudate formation ceases on removal of the irritant, and, at the end of forty-eight hours, there are comparatively few leucocytes and macrophages to nourish and stimulate fibroblasts. In cholecystectomy with drainage, in addition to the theory just given, there is the fact that the drain is the least of the irritants. The sutures used in the gall-bladder bed, the trauma to the free fold of the gastro-hepatic omentum, the ligatures on the cystic artery and cystic duct, the combination of these irritants produces more lymph exudate than does the drain, and, furthermore, the sutures, ligatures and raw area remain long after the drain has been removed. I doubt very much that the lymph exudate from prophylactic drainage is a major malefactor in the formation of adhesions.

So much for the detailed story of lymph exudate in infections, and as yet it is still a story in this presentation. On paper, in the test tube, and under the microscope it seems reasonable that incision and undisturbed drainage best serve the patient's welfare because they protect and irritate lymph exudate. However, bacteriological and pathological findings are just theories to the surgeon, unless he can prove them clinically. The statement "that is not my experience" is a convincing argument against any theory, an argument that can be contraverted only by examples of application of the theory. What are the clinical experiences with the physiological treatment of infection?

During the World War, Morrison, by the use of B. I. P. P. paste and

infrequent dressings, secured results which were said to have been as good as those obtained by any other method. Morrison attributed the effects to some sort of sterilization as a result of the interaction of the paste and the tissue juices; the paste itself had no antiseptic effect; in fact, it had to be sterilized before it was introduced into the wound. Fleming was never able to prove Morrison's theory by experiments in the test tube or in the wound, and he concluded that the good results were due to the infrequent dressings. Furthermore, in 1919, Fleming suggested that some bland substance for the wound packing would give results equally as good as B. I. P. P. Orr has supplied that bland substance in vaseline gauze and has combined it with passive wound treatment more radical than Morrison's. Orr's results have proven that Fleming's deductions were correct.

The physiological treatment of surgical infections, call it the Orr method, or the Morrison-Wright-Fleming-Orr method, gives brilliant results. With a generous incision and a bland greasy substance for wound packing that is not disturbed for ten days or longer, deformities seldom occur after infection. The packing never plugs, and when it is removed there is exposed a healthy, red wound, with little or no pus. Surgeons who have used this treatment know that antiseptics are useless; they have had proof that the ideal cure can be obtained by supporting and controlling lymph exudate.

Orr believes that the good results are due to an absence of re-infection by frequent dressings. If re-infection were probable, it is possible also that the accumulation of the wound secretions on the skin would re-infect, or that the cast and the thick unchanged dressings would cause a washing into the wound of the skin bacteria by perspiration. It is probably more exact to say that there is no re-activation of the wound infection by trauma to the defenses. Whether it be re-infection or re-activation, the end-result is the same: the excessive irritation leads to excessive lymph exudate and fibrous tissue.

Fibrous tissue is the deforming factor after infections treated aggressively. For instance, in a case of acute osteomyelitis that has been treated by antiseptics, *etc.*, there practically always occurs a sinus which is generally supposed to be due to a continued infection. Look at the sinus and see the inverted skin edges, feel the sinus wall and note its firm consistency, excise the sinus and transplant into the gap a flap of soft parts and see it heal up (as Reid and others have done), and you will be forced to the conclusion that the persisting sinus is due to the fibrous tissue. Or take a sinus in the soft parts after an infection that was treated similarly to the osteomyelitis, and if you strap the sides of that channel together, healing will occur in a few days; the cause for the drainage was due to the fibrous tissue that prevented collapse of the channel. You may well ask, if the sinus in osteomyelitis be due to fibrous tissue, why does sequestration, a remnant of the infection, so frequently occur?

Sequestration and sloughing tendons, we have been taught, are due to a destruction of the blood supply by the infection. When you have seen

cases of acute osteomyelitis heal without sequestration after treatment by the physiological method, you will have reason to doubt that the infection is the only factor in destruction of the blood supply, or even an important factor. Before infection *per se* can be condemned for the sloughing tendons and the sinuses, it must be proven that granulation tissue is blameless. Granulation tissue is known to destroy cartilage and bone in arthritis, and, in spite of the recent reports on parathyroid hyperplasia, it is probably the source of destruction in osteitis fibrosa cystica, local or diffuse. Granulation tissue is in excess wherever there is prolonged infection or irritation; therefore it is excessive in the old method of treating infection. Granulation tissue attached to any structure will shut off, by its bulk, the blood supply to that structure and this is the probable source of bone and tendon destruction after prolonged infections. This statement could be classed as rank heresy were it not for the fact, as previously mentioned, that sequestration and sloughed tendons seldom occur in the physiological treatment; that is, they are uncommon where there is a minimum of irritation and granulation tissue. If further proof be needed, cut away the fibrous tissue and granulation tissue, close the wound so that there will be no further irritation and see the wound heal without sequestration. In terminating this discussion on lymph exudate and its value in infections, let it be noted that skill is not essential to the ideal cures, and that deformities are not an index of the severity of an infection.

The lymph exudate of repair has the same qualities as the lymph exudate of defense. When the storm of infection has cleared away, and, to some extent during the infection, the defensive barrier changes to a reparative medium. The fibrin forms the scaffold for the ingrowing fibroblasts and the sprouting blood-vessels; thus, there comes into existence the granulation tissue. At first, the fibroblasts and the blood-vessels lie parallel to each other and at right angles to the surface of the wound. After organization is completed, the cells and fibrils have changed to a direction parallel to the surface of the wound, the blood-vessels have become obliterated and the cells have shrunk. Whether the fibroblasts spring from fibrous tissue, or from the polyblasts or the monocytes, seems of less practical importance than is the final arrangement of the fibrous tissue bundle.

Every soft pliable scar has the fibroblasts and fibrils arranged parallel to the surface of the scar, whereas, in adhesions and in some contracted scars, the bundles are at right angles to the coverings, the primitive arrangement in healing. Horsley, in his study of cicatricial contractures, came to the conclusion that there were no histological differences between the contracted and non-contracted scar, though he did note that in the contractures fibroblasts were found in an arrangement at right angles to the epithelium. Could it not be that it is just this difference in polarity of the cells that accounts for the tendency of scars in certain regions to contract?

Longitudinal incision in the neck, elbow, hand, axilla and popliteal space—all of them have a tendency to contract, regardless of whether there has

been healing by so-called primary intention or healing by granulation. These contractures stand out in whiplike cords and they mechanically impede motion. Inspection of these cords shows that the fascia in the region of the scar is pulled out beyond its surroundings. This traction outwards could be explained by the arrangement of the fibrous tissue bundles at right angles to the skin, an arrangement that would cause the fascia to be pulled up and out when the scar tissue contracted at organization. Why should the bundles have a different arrangement in contractures? Bunting and Eades may have answered this question by their experiments in which they were able to change the arrangement of the fibroblasts by changing the lines of traction on a wound. If they applied traction perpendicular to a wound, fibroblasts assumed that direction; if the traction was parallel to the line of the wound, the fibroblasts were in the same direction; if the forces were radial, the fibroblasts were also radial.

Now, it seems to me theoretically probable that muscle- and tendon-push on the skin determine the contractures mentioned. Feel your own antecubital fossa when the elbow is flexed and you will notice how the biceps tendon stands out like a cord and pushes up the skin. If there were a longitudinal wound in this region and if the elbow were held at a right angle or if it were moved frequently before organization, the fibroblasts would, according to Bunting and Eades' experiment, lie at right angles to the wound, and, after organization, they would pull out the deep fascia into a band. Or, again, abduct your shoulder and note how the axillary fold muscles make a downward traction on the skin of the axilla, and this is another site of web-like bands. I have noted, though it may be a coincidence, that none of the radical operations for carcinoma of the breast have contractures, and I have assumed that this was due to the removal of the axillary fold muscles and to the splinting of the arm against the chest.

What is the practical application of this knowledge of traction? First, of course, use transverse scars in the regions noted. Incidentally, you may wonder why traction does not affect the latter wounds. I believe that it is due to this fact, that the traction or push out in the center of the wound is neutralized by the pull in or transverse pull at both extremities. To return to the applications, incisions are not always surgical, and there are other injuries, such as burns. If traction be a factor in contractures, it would seem reasonable that in destruction of tissue in the axilla, the arm should be held against the side to eliminate the muscle-push and then depend on later stretching of the fibrous tissue to restore abduction. Also, in burns of the chest and neck, immobilization of some sort might prevent motions that make for contractures by taut muscles. When wounds occur longitudinally in the surface flexures, motion should be prohibited until after four or five weeks or until the fibroblasts have become settled in a line parallel to the scar.

The lymph exudate of primary healing requires very little discussion. The sutures and splints we employ insure healing from lymph exudate

through granulation, so that any defect is usually the result of mechanical error. However, there is some profit and considerable mental diversion secured by translating sutures, *etc.*, into terms of lymph exudate.

Every surgeon knows that the best cosmetic result is secured by the use of non-absorbable material. The answer is found in the fact that the absorbable suture is a greater irritant and more of a foreign body than the silkworm and like materials. Catgut requires more lymph exudate and requires it over a longer period of time because it is not removed, therefore, with these sutures, there will be more fibrous tissue.

In suturing, one should remember that every stitch more than required is a double irritant; first, by means of the extra foreign body, and secondly, through the trauma of introduction of that foreign body. If, as seems probable, purse-string sutures in appendectomy be unnecessary, then the practice of burying the stump should be abandoned, not on account of the change of form in the cæcum, but because of possible fibrosis and adhesions from unnecessary trauma. On the other hand, extra irritation and added trauma might be of advantage, as in herniorrhaphies; here many sutures closely placed will secure a firmer bond between the muscles and Poupart's ligament than will a few sutures. Likewise, close suturing of the abdominal wall is an added protection against prolapse of the intestines or a stretching of the scar. The use of many sutures in gastroenterostomies in order to prevent leakage may be unnecessary. It is probable that leakage would not occur provided the surgeon made use of interpolation—if only one line of sutures were used—because, as Hertzler has demonstrated, the interstices between the sutures are filled with lymph exudate in a few hours. This observation explains the absence of gastric juice leakage after this operation and accounts for the safety of water administration in small amounts by mouth after twelve hours.

The purpose of sutures is to hold firmly the lymph exudate scaffold. The suture material is absorbed or removed before the scaffold has disappeared and before the fibrous tissue has organized. It is in the interim between lymph exudate and fibrous tissue, between suture and organization, that great care is required. This is the period when we must guard not only against what the patient may do, but also against what others might do to the patient. Garlock had this danger period in mind when he advised early active motion and counseled against passive motion following tenorrhaphies. He knew, for instance, that the finger movements would be commensurate to the strength of the exudate bond, whereas the masseur might tear that bridge away.

Every discussion of lymph exudate is more or less intertwined with the subject of fibrous tissue, but a discussion of fibrous tissue does not of necessity involve lymph exudate. Fibrous tissue also springs from blood-clot, a formation in which lymph exudate is a secondary factor. Elimination of blood-clot is another method of fibrous tissue control. Some few years ago, Owen, at one of these meetings, showed some excellent functional results obtained by the frequent tapping of hæmarthrosis of the knee. From experience, we all know that blood left in a joint will clot and organize, with the result that there will be impeded motion by the mass, or ankylosis from destruction of the cartilage by granulation tissue. Organization of blood also occurs in the largest endothelial cavity, in the peritoneum, and to leave clots here is an invitation to the formation of fibrous tissue.

Fibrous tissue and lymph exudate are not related problems when the deformities come to the surgeon in full bloom. Then, control is no longer possible; the solution can be secured only by stretching or collapsing the wall. If one attempts forcibly to break up the adhesions, then there ensues another process of granulation and organization.

The treatment of fibrous ankylosis of joints is a prominent example of what stretching can do as opposed to what breaking adhesions will cause. The dramatic procedure of breaking up joint adhesions under anæsthesia is brutal and useless. Reverse Hunter's formula and "think—don't try," and you will never use this method. Breaking up adhesions means bleeding into and around the joints, more fibrous tissue and aggrevated ankylosis. Clinically, after these procedures, the joints are swollen and, in the end, the patient has less motion than at the beginning of the treatment. Put these cases on gradual stretching by plaster casts or by traction apparatus and a fairly good functional result will be obtained in many cases.

Stretching of adhesions, a stretching that is persistent, is an occasional cause for disability and pain. I have in mind the adhesions about the subastragalar joint after fracture of the os calcis. The efforts of surgery in this instance are directed against further stretching by abolition of the joint motion through arthrodesis.

Stretching is a big item in the surgery of fibrous tissue; sometimes we use it; occasionally we abolish it, and frequently in abdominal wounds we guard against it by the use of belts. In the abdomen, for fibrous tissue to be truly reparative, it must be a narrow, firm band that joins together gaps in the muscle or fascia or joins muscle and fascia. Where the scar is thin and wide, a belt might be of service if it could prevent lateral traction on the scar, which it rarely does. If the patient be very fat, then the belt does reduce stretching by preventing pressure from the inside. However, to consider the belt as a protection against muscle-pull is a delusion, because when the patient is stooped over, as in the act of lifting, two hands can be inserted between the belt and the belly. For the linear scar, the belt gives comfort to the patient but nothing else, for be it remembered, that these scars become organized in three weeks.

The most efficient treatment of fibrous tissue is that which has for its object the excision of the scar or the collapse of the sinus wall. Already, this treatment has been noted with respect to the sinuses of osteomyelitis, and it is the principle behind thoracoplasty in chronic empyæma. Fistula in ano is also a sinus encircled by fibrous tissue; if it were possible to strap the sides of this channel together, healing sometimes would occur without opera-

tion. Incision of the roof of this quasi-fistula results in healing, though delayed healing, by a collapse of one part of the fibrous tissue. Excision of the entire tract and immediate closure of the wound, as is done in Ashhurst's clinic at the Episcopal Hospital, gives a rapid cure, and a union by adhesion in the majority of the cases. The prolonged dressings for many sinuses could be prevented by excision of the entire fibrous tissue barrier.

What about the fibrous tissue riddles of keloid and peritoneal adhesions? Whoever solves keloid will answer the question of tumors and their related subjects of tissue tension, internal secretion, growth and inherited properties. As for abdominal adhesions, it does not seem probable that any substance introduced into the peritoneal cavity will prevent their growth, since even physiological salt solution within the belly is a cause for a leucocyte emigration.

At the end of the scale, I have placed non-union of fracture. Cowan's brilliant work seems to show that non-union is the result of fibrous tissue, and that the fibrous tissue does not represent an attempt at bone repair. By experiments on dogs, and by clinical observation, Cowan came to the conclusion that the fibrous tissue from the periosteum grew between the bone ends because the fragments were separated. This band, he reasoned, grew faster than the medullary callus could fill the gap, and it succeeded in holding the bones apart. Perhaps this difficult complication will be solved by the simple method of excluding the periosteum, as Cowan has done, when operating on cases of non-union.

I might continue for some time this theorizing and recital of old tales, but I fear that I would be committing the error of repetition of details. Details are all I have had to bring you, for which I have, as excuse, the saying attributed to Michelangelo that "success is due to details, but success is no detail."

ANNUAL ADDRESS FOR 1930

SURGICAL MANAGEMENT OF CRANIAL INJURIES *

By John Stewart Rodman, M.D.
of Philadelphia, Pa.

I have chosen the "Surgical Management of Cranial Injuries" as the subject of this address, first, because there are no general surgeons exempt from the necessity of handling such cases; second, because in spite of the voluminous literature on this subject, there is still no absolute uniformity of opinion as a whole, and third, because it is a subject in which I have been interested for some years. It is not a subject to which I can bring revolutionizing ideas, however, but rather one which I shall consider from the general surgeon's point of view. The neuro-surgeons have added considerably to our knowledge of the many problems presented by head injuries in general. I have drawn freely from the experience and comments of this especially interested group.

Head injuries are at the same time the oldest and newest subject in surgery. Even prehistoric man developed a certain skill in trephining. Just how early the practice of cutting holes in skulls to relieve fractures and to let out evil spirits was followed has not been determined, but Dr. George Duncan, anthropologist, has stated in recent studies of prehistoric disease, medicine and surgery, that such was undoubtedly done "in a great many unrelated places on the earth's surface."

One skull found in Peru showed that some early physician worked feverishly in an attempt to relieve a basal fracture of the skull before death overtook the patient. Indentations, epilepsy, melancholia, headache, and even magic, seemed to have been indications for trephining. Certain it is that some of these prehistoric people lived among their fellows with soft spots on their heads—the bone was cut or scraped out with flint, and later, with metal knives: skulls having been found with as many as five trephine openings. Such skulls have been found, not only in Peru, but in France, Denmark and Algiers.

The earliest records come from Egypt, which show that such trephinings were done there. We also find in the poems of Homer, which may be accepted as dating from about 1000 B.C., allusions to head injuries, as well as to many other parts of the body. Hippocrates, writing on this subject, begins his comments on such injuries by one of the very few inaccurate observations he was guilty of in describing the sutures of the cranium—namely, that the number and position of the sutures varies with the form of the head; that the coronal suture is wanting when there is no anterior protuberance of the skull and that the lambdoid suture is lacking if there

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is no posterior protuberance. He is the first, however, of whom I have found record who has devised a classification of head injuries—a form of amusement to surgeons ever since then.

Hippocrates thus divided fractures of the skull into four classes: (*a*) simple fissure, (*b*) contusions without fracture or depression, (*c*) indentations of the outer table and (*d*) fractures by contra coup.

Celsus speaks at some length on the subject of head injuries, quoting from Hippocrates. He seems to have more clearly understood indications for conservative or radical treatment as the particular case might warrant. To distinguish a fissure from a suture, he advises the pouring of ink on the part and then scraping the bone; if there is a fissure, the ink will mark it. All depressed bone is to be removed, but no more is to be taken away than is absolutely necessary. He would delay operating otherwise unless dangerous symptoms arise—for at least five days. Galen believed that depressed bone should be removed, but that fissures do not require operation. Paulus seems to have followed Galen, so that Greek and Roman methods of that early day did not differ, in some respects at least, from the conservative attitude toward operation of the majority of this time.

Like surgery in general, no real advance in the management of head injuries was made during the early centuries following the Christian era. Many references may be found referring to this subject, however, amply proving the interest which brain injury has always aroused, as well as the frequency of such injuries long before the motor car of today was dreamed of.

Head injuries have always been a large part of the army surgeon's concern. Thus, in the thirteenth century, Roland, of Parma, wrote a book on the subject, dealing largely with fractures of the skull and wounds of the head, for this period followed closely on the age of chivalry. Then knights in full armor carried on war and their weapons were the lance, mace and battle axe. These warriors were always mounted, and the head was the part aimed at. Roland's knowledge of the important clinical signs of various types of head injury was considerable, and his observations at times would do justice to a well-informed surgeon of today. Thus, he states that fractures of the skull may occur with or without a wound of the scalp, but that the important matter is the injury which has involved the brain or its immediate coverings. This thought has been repeated many times since the thirteenth century, but how many today still think of a given case as having died of a fracture of the skull! Roland went so far as to attempt to differentiate between injuries to the dura and to the pia. In injury to the dura, he states that there is pain in the head, redness of the face, inflammation of the eye, wanderings of the mind and blackness of the tongue. When the pia is injured, he thought there was loss of consciousness, loss of voice, pustules on the face, and, if the injury is severe, rigor of body which is a certain sign of death.

Ambrose Paré reported in the sixteenth century rather numerous observations of his cases of head injury. For example, one of these patients had been unconscious fourteen days after having been hit on the head with a cannon ball! This man had vomited blood as well as having bled from the nose, mouth and ears, and also having had convulsive tremors—evidently an instance of fracture of the base of the skull with markedly increased intracranial tension. Paré trephined this patient and in his notes states that while he had operated, "God healed him and today he is still living." With all of our surgical progress since then, it is still quite an achievement to save a case of this kind, and there are certainly times when I suspect that influences other than ours have played the larger part. In 1559 Paré's master and friend, Henry II of France, was wounded by a lance blow in a tournament. The King lived eleven days. Surgeons could not find the lance splinters which had penetrated the brain, although they secured the heads of four criminals that had been beheaded, and experimented upon them to try to determine the probable course of the lance splinters. The King evidently died of a subdural hæmorrhage plus infection. Some of Paré's other reports are most interesting, but we must not let historical data delay us too long from the present day.

That versatile and great surgeon of the eighteenth century, Percival Pott, wrote a monograph on head injuries in 1759. He was an ardent advocate of early operation, and one finds cropping up in his opinions such statements as "the more pressing the symptoms are, the sooner the operation should be performed,—and all other attempts are worse than useless." Again—"Inflammation of the membrane always requires, and if slight, sometimes yields to large bleeding, gentle purging and a general febrifuge cooling regimes—even though perforation of the cranium should become necessary." In so far as fracture is concerned, he thought that unless there was a depression, there was no need for operation—that when infection occurred it was due to a "putrefaction" of the dura mater. He further states that fractures of the skull, considered abstractly, are not so dangerous a thing as they are commonly supposed to be. This fact, though stressed one hundred and seventy years ago, has been generally ignored since and until its repeated reiteration in the last few years. Pott advised that all gunshot wounds should be immediately trephined.

Abernethy in the early part of the nineteenth century must have been much interested in head injuries, and I have found the report of cases from his service at St. Bartholomew's Hospital quite interesting. He seems to have been as much of a conservative as his distinguished predecessor, Pott, was a radical. In beginning his report of cases, he states that while it had been largely the custom of the times, no doubt due to Pott's teaching, to trephine early, that in his experience "many cases have occurred of late where, even in fractures with depression, the patients have done well without an operation." He then reports in considerable detail five case histories to prove his point. Repeated bleeding, purging, saline medicines and antimonials were the treatment used in all of these cases—all showing fracture

of the skull, some with depression and all with serious coincident brain injury. These cases all recovered. Abernethy's comments are interesting: "It appears (he states) very clearly from these cases, as well as from a great number of others to be found in books, that a slight degree of pressure does not derange the functions of the brain for a limited time after its application. That it does not do so at first is very obvious, as persons are often perfectly sensible and free from headache and dizziness immediately after the injury. Whether it may not produce such an effect at some remote period is not so easily determined since this cannot be ascertained but by a continued acquaintance with the person who had received the injuries." He continues, however, by stating that all whom he has had an opportunity of knowing for any length of time after the accident continued as well as before. He quotes Mr. Hill as substantiating this belief. Abernethy must have been fortunate in the cases he "followed up." Certainly it is contrary to the majority opinion of the present day that those who recover from serious brain injury remain entirely free from later trouble. I think it can be said safely that all know of instances of persistent headache, dizziness and even Jacksonia epilepsy occurring as sequellæ, regardless of the type of treatment at the time the injury was received, and immediately thereafter. In fact, the writer recalls hearing that wonderful teacher of a few years ago, John B. Murphy, state he would much rather die than to recover after having been unconscious from brain injury for more than five minutes. While this statement seems somewhat pessimistic, one is never justified in too great optimism that any given case of serious brain injury resulting in unconsciousness will entirely resume a normal life should immediate recovery follow.

Much more could be said on the historical side of this question. Many of the surgically great seem to have written something on the subject of head injuries. One can get a fair estimate, I think, of the practice of the times if the opinions of a few of these be considered as we have done. From Abernethy's time to the dawn of modern brain surgery twenty-five years ago, some progress was made, of course, but not of great moment as it has only been since then and more especially during the past ten years, that surgeons in general have had a better understanding of the underlying pathology of brain injuries. During the century that has elapsed, bleeding has gone and obvious skull injuries have been repaired where possible. Purging, however, has come back now on a more scientific basis. The advent of anæsthesia and asepsis has greatly increased the patient's chances.

There is still, however, and probably always will be, that irreducible minimum of immediately fatal brain injuries because, in spite of the centuries which have elapsed, and a certain increased storing with utilitarian knowledge, the brain is now the same as it was in the pre-Christian era. There is no axiom in surgical pathology more true than the one which states that the higher the degree of specialization of a tissue, the less is its power of regeneration! The tissues of the central nervous system, being the highest

of all, have none! Therefore, if certain vital brain centres are destroyed, life goes with them, and there is no help for such a situation. There is, however, that increasingly large group which fall between the immediately fatal and the trivial. All of the latter will get well, provided complications do not arise, and most of the intervening group should and do, under intelligent, modern management. These patients should not only get well, however, but if an intelligent plan of management is now used, should get well with a reasonable chance of staying well.

It is not easy to say just when the modern management of brain injuries began. A better understanding, at least, of such injuries seems to have gone hand in hand with the growth of modern brain surgery. For much of this progress, therefore, we general surgeons must give the credit to that especially interested group in the last twenty-five years—the neuro-surgeons.

It is perfectly apparent, of course, that one cannot outline clearly the management of an injury unless the underlying pathology of that injury is clearly understood. The difficulty of anyone, therefore, developing a rational plan of treatment of brain injuries during the centuries which have now elapsed, has been that until very recent years, a clear understanding of brain tissue reaction to trauma has not been definitely known. Even now we cannot say just what happens in the mild cases of concussion, since these cases almost invariably get well. My own better understanding of this matter, however, came with the work of Weed and McKibben, who, in the *American Journal of Physiology,* vol. xlviii, 1919, published the result of their experimental work on the alteration of brain bulk. They showed by direct observation of the brain in experimental animals, that hypertonic solutions of saline injected intravenously, certainly reduced the size of the brain, presumably by decreasing the fluid within the cranial chamber while, conversely, hypotonic solutions (water) injected into the veins, increased the size of the brain, presumably by the escape from the vessels of the watery element; further, histological changes within the brain cells were shown to have occurred where the increased tension was unrelieved—as for example, by trephining to allow more room for expansion—but these changes did not occur when such relief had been afforded. Since that time we ceased thinking of these injuries in the time-honored terms of *concussion, contusion,* and *compression* of the brain or *fractures* of the skull alone, and have since thought of them much more in the light of intracranial tension. These terms have been retained for clinical diagnosis since it has proven to be impossible to get surgeons in general to think of intracranial tension alone. In our own work, however, the latter was the keynote and gave the indications for proper management—as put forth in a paper on "A Plan of Management of Cranial Injuries Based on a New Grouping of Such Injuries," read before this Academy in 1924, and published in the ANNALS OF SURGERY in April of that year. We began about that time to group our cases for the purpose of proper management into three groups, depending entirely on the presence or absence of increased intracranial tension as follows:

Group 1.—No increase in intracranial tension. Sub-group A.—Inevitably fatal. Sub-group B.—Mild concussion. Group 2.—Moderate increase in intracranial tension. Group 3.—Marked increase in intracranial tension.

I will not repeat now what has been said in that paper except that further experience since then with a much larger group of cases has in no way made me change my mind about the efficacy of the simple plan of handling these cases presented then—on the contrary, reducing this heretofore complicated question to a comparatively simple one has been of the greatest comfort to us and has, I am firmly convinced, definitely added to the success with which these cases have been handled. I do not mean to suggest that any grouping will change in one iota the result in some of these cases receiving serious brain injury at the time of the accident. Such fall into the inevitably fatal class in whom death ensues quickly and who do not last long enough to develop any increased intracranial tension—these die of shock plus probably a complete overwhelming of the medullary centres. In this type of case, treatment is of no avail. We classify all of those cases falling into this "inevitably fatal" sub-group who never recover consciousness—are inevitably markedly shocked—whose temperatures usually remain subnormal, although a rapid rise to hyperpyrexia is seen in some and whose pulse remains rapid and whose blood-pressure remains low. Incidentally, in such patients one can usually find evidence of fracture of the base of the skull on X-ray examination. Often there has been bleeding from the mouth, nose and ears and even the escape of brain tissue. Death ensues within a very few to twenty-four hours after the accident. All one can do is to treat shock. There is no need here to attempt to reduce intracranial tension since, as has been said, these patients do not last long enough to develop it. The literature on this subject has become enormous and as a result many clinical groupings have been proposed. All of them, however, recognize this inevitably fatal type of case for which little or nothing can be done. It would serve no useful purpose were it even possible to do so, to mention here the different ways in which many of those who have been interested in this subject, group their cases. Such groupings have no real value unless a definite plan of management can be built thereon. I do not wish to make any especial plea for the adoption of my own. To me it has been useful because I have been able to outline much more clearly what each case, as it comes along, shall have in the way of treatment. In the mild types of Concussion Group 1, Sub-group B (no increase in intracranial tension) the spinal pressure will be normal, and the clinical picture will be that of mild concussion. All of this type, unless complications ensue, will get well. The underlying pathology of this group must be imagined from what we know of brain tissue reaction to trauma. There is little more than a temporary anæmia, closely akin to ordinary surgical shock—the only difference being a transient loss of consciousness, or dazing, since the anæmia affects the medullary centres—when the anæmia has been recovered from, the blood-vessels will dilate again, and, as a rule, the mentality will clear and remain so, unless the next step in brain tissue

reaction to trauma is taken, namely, œdema. We do not find œdema occurring, however, in appreciable amounts until the case has progressed to Group 2 (moderate increase in intracranial pressure) with a spinal pressure reading of from 10 to 20 millimetres of mercury. These cases will show clinically, marked concussion, or even contusion with an increased period of unconsciousness, slow pulse and raised blood-pressure. If, when consciousness has returned, the patient is restless and lapses readily when undisturbed into sleep, we may fairly surmise that brain tissue has been minutely lacerated and minute hæmorrhage has added itself to the usual compressing force—œdema. In spite of treatment calculated to reduce this increased intracranial tension at times, the case will progress rapidly to Group 3 (marked increase in intracranial pressure) with a spinal pressure reading 20 millimetres or above. These cases will show the clinical picture of compression—unconsciousness lasting for days or even weeks, giving way to coma if the pressure be unrelieved, with a slow pulse later to become fast and weak, and with an elevated blood-pressure falling as the picture progresses toward a fatal termination. There is no need, in addressing this audience, to enlarge further on these separate pictures with which we are all familiar. So closely does the clinical picture fit the spinal-pressure reading, however, that we have given up doing it as a necessary step in the milder cases of concussion for diagnostic reasons.

The surgical management of any uncomplicated case falling into any of these groups is directed solely at relieving intracranial tension where it is increased. Since there is no increase in Group 1, Sub-group B, the simplest plan is called for—rest in bed three or four days, early and free evacuation of the bowels by magnesium sulphate enemas and a limited fluid intake will suffice, the latter two procedures being routinely done more with the idea of forestalling the development of œdema than of reducing it. In those cases falling into Group 2—to these measures will be added an intravenous injection of a 50 per cent. glucose solution (the latter having taken the place of hypertonic saline in our hands and *most others*) and possibly therapeutic spinal puncture, removing sufficient fluid daily to reduce the reading to slightly above normal, 12 millimetres on an average. In our group of uncomplicated cases, falling into Group 2, there was a small mortality. A greater death rate will, of course, be found in Group 3 (marked increase in pressure above 20 millimetres of mercury). To the plan of management already mentioned for Group 2, one must consider the addition of decompression. After having seen a great many cases since the beginning of this work on the above plan, I must confess to being still somewhat in doubt as to the wisdom of ever doing a subtemporal decompression. As a general rule, it will do no good in the cases which fail to respond to the non-operative plan already given—and yet there is the occasional case, progressing slowly toward the terminal stage of compression in spite of the most active non-operative plan that will, I believe, benefit by decompression. I have seen such benefit a few times, but more often have been disappointed in accom-

plishing nothing. In such a case, however, no general anæsthetic is necessary and this simple procedure can do no harm. I intend to keep on doing it, though I believe it will only occasionally be thought of as a probable help.

With increasing experience, spinal tap has become with us more of a treatment than a diagnostic procedure. The character of the fluid means much as, of course, bloody fluid can only mean damage beneath the membranes and, naturally, makes the prognosis more grave.

I have referred to the "uncomplicated case" repeatedly. We have considered, in this plan, such frequent accompaniments of head injuries as laceration of the scalp, fractures of the skull, localizable intracranial hæmorrhage as complications in themselves, each with their own operative indication. There is no need to go into the matter further at this time as many papers have already done so, and I take it we are all agreed that such lesions usually call for surgical interference, except in the linear fractures of the vault or base. Again we shall repeat, however, that it is the injury to the brain which matters and to which we must direct our chief attack, and this means combating intracranial tension. I have not quoted most of the splendid original work which has helped in doing this, as those who have done it are well known to us, and have long since received the credit which they so rightly deserve.

These impressions have been based largely on seeing many of the 800 cases which have occurred on the surgical services at the Presbyterian Hospital since 1925. I am greatly indebted to Doctors Jopson, Hodge and Speese for making it possible for me to have done so. While the series does not include those cases seen prior to 1925 at the Presbyterian Hospital, nor those seen on my services at the Bryn Mawr and the Woman's College Hospitals, I felt that it was better for the purposes of this report to consider only this particular group occurring at one hospital since it is of sufficient size to warrant drawing conclusions from.

Summary

Total number of cases studied 800

Group I. (No increase in intracranial tension)

A. Inevitably fatal	36
B. Concussion (mild)	578
Group II (Moderate increase in intracranial tension)	65
Group III (Marked increase in intracranial tension)	121
Total	800

Total mortality of series, 60 or 7.5 per cent.

		L.	D.	Per Cent.
Group I	604			
Sub Group A.		0	36	100
Sub group B.		574	4	.7
Group II	65	59	6	9.2

		L.	D.	Per Cent.
Group III	121	107	14	11.5
Mortality of those showing serious brain injury (Groups 1A, 2 and 3)		166	56	25.2

(One case in every four in this series died of serious brain injury.)

Causes of death

Group I

A. Inevitably fatal	36 cases
(General and cerebral shock—brain laceration.)	
B. Mild concussion	4 cases
Pulmonary embolism	1
Terminal pneumonia	1
Hæmopneumothorax	1
Shock (multiple fractures)	1
	4
Group II	6 deaths
General and cerebral shock—mild brain laceration	2
Peritonitis (simultaneous rupture of intestine)	1
Diffuse subdural hæmorrhage	1
Chr. alcoholism—terminal pneumonia	1
Hæmorrhage into left lateral ventricle and left internal capsule	1
	6
Group III	14 deaths
(All died of brain compression.)	

ANNUAL ADDRESS FOR 1931

THE PROBLEM OF DELAYED UNION AND UNUNITED FRACTURE

By Hubley Raborg Owen, M.D.
of Philadelphia, Pa.

In treating the police and firemen we deal with a large number of men who perform hard, manual work and who are very liable to severe injury. We have a large number of these two surgical conditions to treat. The memory of our surgical failures as well as those of other surgeons is constantly with us. The surgeon who does not make mistakes makes no great progress. The best of men and the most earnest workers will make enough mistakes to make them humble.

The object of this address will be to emphasize some of the etiological factors of delayed union and non-union, and to suggest certain methods of reducing the liability of these complications. It is realized that delayed union, non-union and pseudo-arthrosis may be three separate entities, often with varied etiological factors and requiring different treatment, but for the purpose of this paper they will be considered together.

Wilcox[1] has aptly said: "This is an age of acceleration, mass production, high-speed machinery, automotive and ærial activity." This ever-increasing demand for greater speed and more thrills connected with the desire for more hazardous sports is largely accountable for the increasing number of physical injuries, especially fractures. One often wonders when this craze for thrills and speed will end.

In order to emphasize some of the points with a view of appreciating exactly the incidence of ununited fracture, 11,683 fracture cases treated at the Philadelphia General and the Jefferson hospitals from 1921 to 1931 have been reviewed. There were 101 cases treated for non-union.

The occurrence of delayed union, according to Von Bruns, is one-half of 1 per cent.; according to Scudder, 2 to 3 per cent.; according to Heygroves, 4 to 5 per cent.

As the number of fractures is ever increasing, so the complications of fractures, including non-union, will increase. With the advent of motorized fire apparatus, motor-cycles, sidecars and bandit chasers our problem of dealing with fractures is a serious one. Severe accidents at fires are not as frequent as heretofore. This reduction is due to saner methods of fire prevention, fewer fire hazards and better construction of buildings. Transportation to and from fires is far more hazardous because of congested traffic.

The problem of fracture heretofore was one which concerned principally the surgeons of our city hospitals. There were two reasons for this: First,

* Annual Address before the Philadelphia Academy of Surgery for 1931.

the majority of fractures occurred within the city limits; secondly, the more severe fractures which occurred in the rural districts were usually transported to a city hospital. At the present time the frequency of automobile accidents on the crowded public highways renders it imperative that the physician in each hamlet, village or even at crossroads be prepared to render proper treatment for fractures. These cases are now transported to a nearby hospital rather than to a city hospital, so that the surgeons of these suburban hospitals are now called upon to treat the more serious cases occurring in the suburbs.

Regarding the treatment of fractures by the general practitioner, Wilson and Cochran[2] remind us that, notwithstanding the lack of suitable facilities, the same standards of treatment and the same qualities of results as obtained in the best hospitals are demanded by both the public and the courts.

Modern text-books on surgery divide the etiology of delayed and non-union into two main classifications—constitutional causes and local causes. These statements, like many others in our text-books, have been repeated from one edition to another without contradiction.

Constitutional factors have but little, if anything, to do with the union of fractures. The diseases usually enumerated as causative factors of delayed and non-union are syphilis, tuberculosis, diabetes and blood dyscrasias. In our series of 101 cases, the Wassermann was positive in six cases, negative in ninety-one, not taken in four; the blood chemistry was normal in eighty-nine, abnormal in six, five of which showed a hyperglycæmia and one a hypoglycæmia, not being taken in six; pulmonary tuberculosis was noted in two cases.

Campbell[3] states we do have constitutional ailments which prevent union but that they are so rare that, excepting in those fractures which are infected, union will occur in 95 per cent. of the cases, and that non-union of fracture is undoubtedly caused by local interference with the physiological or nature's attempt at producing union.

Newell[4] does not believe that systemic conditions have anything to do with union or non-union. He considers the condition an entirely local one.

Wilson,[5] from his studies, agrees with Henderson[6] that local causes are far more significant in the development of non-union of fracture than general or systemic causes, but states that the chemical analysis of the blood to determine the calcium and sulphur content is valuable in a small number of cases.

Henderson, in discussing this subject two years ago, stated that in The Mayo Clinic up to that time, so far as he knew, there had been only one case of non-union which could actually be ascribed to syphilis.

Culbertson[7] has found that in normally healing fractures the catabolism of calcium shows but little change. There is a marked loss of nitrogen, phosphorus and sulphur, the main excretory path for these catabolities being the kidney. He, further, found that metabolic analysis did not throw any light upon the problem of ununited fracture. A catabolic loss of sulphur, nitrogen and phosphorus may result from injury to tissues other than bone.

The majority of writers, as pointed out by Lacey,[8] do not believe that the calcium and phosphorus index can be used as a prognostic index of union or non-union.

Bohler[9] believes that such diseases as tuberculosis and lues, and even osteomalacia and rickets, only delay but do not prevent union. He adds that non-union after fractures

of the shaft is chiefly found in powerful and healthy individuals and disproportionately more common in men than in women. We found in our series seventy-three cases of ununited fracture in the male and twenty-eight in the female. One reason for the disproportionate frequency of non-union in men may be due to the fact that men are subjected to more severe injury, and, being more muscular, there results greater shortening and greater trauma in attempts to reduce this shortening. Certainly rickets cannot be a cause of non-union as orthopædic surgeons deliberately fracture rachitic bones and non-union is practically unknown.

As quoted by Pat Fite,[10] there are some surgeons who believe that osteoblasts occur either from migration at the ends of bone or are formed from newly made connective tissue by metaplasia, and that due to the activating enzymes produced in these cells calcification is brought about. Other workers at the Presbyterian Hospital in New York fairly recently have sought to show that from their experiments this is not the case, and, further, that the available source of calcium at the site of the fracture is due to tissue death and that an enzyme if formed is due to the death of this tissue and is probably affected in no way by the blood calcium.

Fite also notes the fact that non-union of the tibia occurs where there is poor lateral blood supply and also believes that in certain cases of fracture, as in the case of the neck of the femur or tarsal scaphoid, that union is affected by being bathed in synovial fluid. He believes that fractures bathed in synovial fluid are hampered to some extent in union because of slow and perhaps imperfect callus formation. Other surgeons deny this.

Bohler cites as the causes of non-union lack of reduction of fragments and interposition of soft parts, and emphasizes that the most common cause is insufficient and inadequate early treatment.

It is interesting in this connection to consider the thoughts of some of the earlier surgeons. Black,[11] writing in 1797, states that his era was "an age of investigation of fractures in which mankind was eager to attain even absolute perfection." He observes: "Although the science of surgery has reached a very high degree of perfection, yet there still exists considerable disagreement of sentiment on many points and perhaps none more than on which is the best mode of treating fractures." He adds that "falls, blows and bruises are said by many writers to be the common causes of fracture, but a little examination and reflection will be sufficient to prove that muscular action is a very principal agent in the production of fractures." Black further reflects that "in addition to this, we may observe that persons who are drunk or under the operation of any cause that produces relaxation of the muscular system, although they fall from a considerable height, seldom receive a broken bone." Even now, as then, a strange providence watches over those who violate the spirit of the eighteenth amendment.

Certainly fractures by muscular action are not as frequently complicated by non-union as fractures received by direct force. In our series of cases the primary injuries were:

Falls	47
Automobile accidents	24
Blows by external forces other than automobile accidents	24
Crushing injuries	4
Gunshot wound	1
Stab wound	1

We found that non-union occurred in the first decade of life in three cases; in the second decade of life in eight cases; in the third decade of life in nineteen cases; in the fourth decade of life in nineteen cases; in the fifth decade of life in sixteen cases; in the sixth decade of life in sixteen cases; in

the seventh decade of life in eleven cases; in the eighth decade of life in seven cases; in the ninth decade of life in two cases. Of those occurring in the first decade, one was in a child three and one-half years old who was struck by an automobile and sustained a fracture of the left femur. The child was admitted to the hospital two months after the injury with non-union of the fracture. The second case was a child six years old with a fracture of the left humerus and non-union for two years and eight months. The third case was that of a child ten years of age with a fracture of the right femur, non-union duration of three months. These are unusual, as union occurs far more readily in the young than in the old. In this connection it is interesting to note that Gross reported firm union in a fracture of the humerus occurring in a patient whose age was one hundred years.

Local causes as etiological factors in delayed and non-union are far more important than constitutional factors. The most frequent of the local causes are: Lack of proper apposition or approximation of fragments; interposition of foreign tissue; injury to the blood supply; presence of foreign bodies in fracture zone; infection; and lack of proper fixation.

The question of nerve injury as an etiological factor is doubtful. There was but one case of our series which was associated with a nerve injury, that of a child thirteen years old, who sustained a fracture of the left humerus associated with musculospiral palsy. The case was admitted to the hospital eight months after the accident with non-union and wrist drop, but this does not necessarily mean that the musculospiral palsy was a factor in the non-union.

Injury to the blood supply is a well-recognized cause of non-union.

Cox[12] has emphasized the necessity of allowing for free circulation through veins and lymphatics as well as through arteries as through the medium of the veins and lymphatics the old structure is torn down and absorbed, thus permitting the growth of new vessels to form and the establishment of anastomosis so essential to bone repair. If resistance to expansion (swelling) occurs, its effect will be to diminish or occlude the veins, lymphatics and arteries, thus preventing an adequate supply of blood from reaching the interior of the callus. It is of great importance to prevent hæmorrhage, swelling and inflammatory reaction, especially during the early days of a fracture.

Impairment of the blood supply as an etiological factor in non-union was emphasized by Lacey in his excellent paper read before this Academy in 1929.

Bankhart[13] believes that the one common cause for non-union is the absence of hæmorrhage from between and around the fracture surfaces. He states that bones bleed when they are broken and usually there is considerable hæmorrhage between and around fracture surfaces. Extravasated blood, acting as a stimulus, produces the first step in the repair of the fracture. When for any reason sufficient hæmorrhage does not occur at the site of fracture, the natural stimulus for repair is lacking. He states that this is distinctly common in the case of fracture of the neck of the femur in an elderly person, not, as usually stated, on account of malnutrition of the fragments but simply on account of the absence of hæmorrhage, for these fractures are practically dry. He does not think imperfect immobilization is a causative factor, because, as he quotes: "Fractures of the ribs and other bones unite readily in spite of constant movement." He believes that systemic diseases are often causative in non-union because they are associated with arterial changes and, therefore, the bleeding around the ends of the fractured bone is very slight. For

delayed union he advocates the injection of autogenous blood around the site of the fracture. Bier has also used this method. Bier has also advocated the use of hyperæmia for delayed union. Diathermy and the use of certain therapeutic lights may be used to increase vascularity.

In this connection, it is interesting to note that it has been recognized for many years that irritation between the fragments will often stimulate osteogenesis. Celsus used acupuncture and introduced bristles between the fragments to stimulate union. The insertion of ivory tacks and the injection of bone-marrow was used by Wyeth, in 1878. Thomas percussed the tissues over the ends of the fragments with a rubber hammer. Lannelongue and Menard[15] advocate the injection of a few drops, 1 to 10 solution, of zinc chloride between the fragments.

Proper allowance should always be made for the expansion of the soft parts. This has been further dwelled upon by Newell,[16] who contends that the most important consideration in the treatment of a fracture is to maintain a good blood supply to the broken bone and to allow for free anastomosing circulation with the blood-vessels coming from the soft parts to the bone. He further directs that there should be as little manipulation of the bone as possible so as not to disturb the periosteum.

Cowan[17] attributes the cause of non-union to local anatomical conditions resulting from laceration of the periosteum, usually flush with the fracture line and separation of the fragments.

Callus is formed from bone-marrow from the broken splinters of bone, periosteum from the muscles attached to the bone and from the hæmatoma which is mixed with the fat of the bone-marrow.

Boehler[14] believes that too short and interrupted immobilization of fractured bones is the cause responsible for non-union. Extensive detachment of the periosteum produces a large subperiosteal callus. Bohler has shown that in transverse fractures of the lower third of the tibia and in the middle third of the radius there is produced only a very slow-growing callus. He believes that to obtain firm union in fracture of the tibia from ten to twenty weeks are necessary and for fracture of the radius from six to twelve weeks, believing that the cause of delayed union in these bones is based on the fact that the bone-marrow in these fractures is open only in a small area. He reiterates that the cause of delayed union in fractures of the tibia is due to the fact that the anterior and medial surfaces are not covered by muscles and the blood supply to this area is, therefore, poor. It can be shown by means of the X-ray that in fractures of the lower third of the tibia callus formation takes place only on the lateral and posterior surfaces, while upon the medial and anterior surfaces, not covered by muscle, bone formation is frequently very slow.

In speaking of the rôle of bone-marrow in union, I am reminded of the quotation of an English surgeon[18] who said to his students: "You may think, gentlemen, the shaft of a long bone is occupied by marrow, but do not believe it; the medullary cavity is filled with base, black ingratitude which flows out when the bone is broken."

By what means may we possibly minimize the occurrence of delayed and non-union? Groves[19] has aptly stated that the three main types of mistakes in the treatment of fracture are neglect, delay and error of judgment. Delay in the efficient primary treatment of fracture accounts for three-fourths of the failures, which are frequently due to sheer carelessness or procrastination. Far too often a patient is admitted to the ward, and

nothing is done until the next day, when a skiagram is taken and the fracture may not be seen by the surgeon for a day or two unless some grave symptom arises. Every compound fracture should receive the same immediate care as a fractured skull or acute abdomen and the full surgical team should set about the problem. Extensive examination should take place at once, with no more delay than in the case of a ruptured gastric ulcer.

For the proper treatment of fractures one must understand the mechanics of fracture. We cannot agree with the deduction of Russell, who states: The only one bone being fractured finds in gravity an ally and help in treatment is the humerus and for this reason fracture of the humerus should be the easiest of all fractures to treat. He also adds: "Gravity aided by a sling and bandage, with very little guidance from the surgeon, will do the rest." He speaks of the Balkan frame "with iron splints" as the crucifixion method of treatment.

The successful surgeon is one who is mechanically minded. It is realized that surgery is becoming more and more specialized. We have beds assigned to the neurological surgeons, thoracic surgeons, genito-urinary surgeons and other surgical specialties. In many of the larger hospitals fracture cases are now segregated. It is by no means advocated that the treatment of fracture should be considered as a specialty, but every hospital should have a fracture service. The surgeon who is not interested in the treatment of fractures should graciously refer these cases to the surgeon who is adapted for this work. This procedure should add to efficiency. The segregation of fracture cases facilitates the use of the necessary equipment and adds to the convenience of the surgeon.

There was once an old adage:[20] "It is not necessary to go to a fracture as you would to a fire." This statement is untrue. Every fracture should be considered as an emergency.

Wilcox[21] remarks that "until not long ago the arrangement and treatment of fractures have been left to internes or junior house officers. Medical students should be taught that a broken bone is something more than a mishap and the laity should be made to realize the importance of this subject. There should be provision for immediate X-ray examination, both night and day. Assisting staff, internes and nurses should be definitely instructed in the methods to be employed for the treatment of fractures."

Findlay[22] strongly advocates that internes assigned to ambulance service should receive special instructions as to the first-aid treatment of fractures and additional instruction should be given ambulance drivers in methods of assisting the interne. He concludes that:

(1) The internes appreciate having specific equipment and knowing definite methods for treating any particular fracture.

(2) Their work is carried out more accurately, quickly, and gently with this knowledge and equipment.

(3) When ambulance chauffeurs have been given specific instruction, they coöperate with and assist the internes.

(4) The patients are more comfortable.

(5) Shock is lessened or prevented.

(6) Our results in the treatment of fractures in general have been definitely improved since proper treatment has been instituted at the site of accident.

Bancroft[23] emphasizes that immediate replacements of fractures is imperative because "several hours after fracture the swelling due to hæmorrhage which infiltrates the muscle bundles is so excessive that replacement becomes difficult." Non-union may result from the fact that extremities often have to be suspended for several days in order to allow the

swelling to subside before reduction can be attempted. He, too, emphasizes that allowance must be made for free and adequate circulation and that in cases where reduction is not attempted for several days the gelatinous consistency of the callus interferes with manual correction of overriding and to correct this deformity only long-continued traction may suffice.

Ashhurst[24] insists that reduction should be secured within a few hours of the injury "because if the surgeon postpones reduction he finds it increasingly difficult to secure because the reparative processes of nature will not await his convenience." He further emphasizes that delay and especially oft-repeated attempts at reduction not only do great injury to the soft parts, but are apt to hinder the progress of union with the result of delayed union or even non-union.

When the case is admitted to the hospital, before manipulation is attempted, X-ray studies should be made. The value of the biplane fluoroscopical screen cannot be overemphasized. A single picture is of no value, excepting to diagnose the existence of a fracture, and it may fail to do even that. An X-ray of the entire bones should be made, with more than one plane taken. Some surgeons advocate taking an X-ray of the corresponding bone of the opposite extremity. The X-ray should be repeated after the fracture is reduced. Fractures should be reduced preferably by the findings of an X-ray and not by manipulation. We thoroughly disagree with Russell,[25] who writes that "X-rays are the indirect cause of much unnecessary operating on fractures," and that "X-rays can rarely demonstrate anything of importance which cannot be more easily demonstrated with a tape measure," but we do agree with him in his thought that the inspection of fracture films by patients may have serious psychological results because far too often the laity, and especially juries, lay too much stress upon a line of fracture *per se*.

To reduce a fracture muscular relaxation is imperative. This was recognized by the early surgeons, who obtained muscular relaxation either by means of alcoholic beverage, opium or blood-letting. Black states that "the reduction of a fracture is in general attended by little trouble since extension and counter-extension be all that in a majority of cases are requisite." Furthermore, that "unless laceration be great or the muscular system in such a state as to be easily thrown into convulsive action, the resistance given is seldom so powerful as to baffle the judicial efforts of two or three persons," but, he adds, "it has been not uncommon for us to see or hear of six or eight or as many men as can stand around the patient to use their utmost force to extend a limb, which force has sometimes even been so great as to tear the muscles to pieces while the unhappy sufferer must calmly submit to a fate fashioned by custom."

Black advocates the treatment of bleeding *ad delquim animi*, producing a suspension of muscular action; that is, bleeding just to the fainting point. This, he states, is often successful and by this practice one can get sufficient relaxation of muscles so that the force of two or three persons will be all that is necessary to reduce a fracture. This practice of bleeding to the fainting point was also popular because the patient felt no pain from extension of the limb and replacement of the fragments. Fainting, it was said, could be produced with safety and readiness in proportion to the size and velocity of the stream of blood. Should the vein be small and the bleeding slow it was often necessary to open an artery. The radial artery at the wrist was the one usually attacked. Black frequently practiced this procedure with a lancet and stopped the flow by compress and bandage.

Black was not enthusiastic about splints. One of his chief objections to the use of splints for fracture was for the reason "men in general have a greater or lesser degree of bandy-legs and by the use of splints following fractures the leg is made to deviate from its former shape and from that of the sound limb which would be the case if completely straight."

He mentions the case of a man who had been accustomed to drink ardent spirits freely and whose whole system was in an irritant state. This patient fell to the floor and received "a very oblique fracture" of the bones of the right leg. During the first attempt

to reduce these parts severe spasms occurred and continued four or five days. During this time the lancet was used freely to dispose of the spasms. This was effected by repeated bleeding, cathartics and a vegetable diet. From the day of the accident to the ninth day he lost 125 ounces of blood, which conquered the disposition to spasm.

Black remarks that there are "some unfortunate cases in which the ends of the bone do not unite. The inflammation subsides and the integrants resume their former appearance, but the bones remain separate." This, he says, is usually due to the consequence of some disease in the part which destroys the first bond of union. In some cases certain authors advised an incision be made down to the bone and the ends of it be sawed off. This surgical procedure was practiced first by White, of England, in 1760.

Black also mentions a mode of practice which has been successful in his hands and at least merits a trial; namely, to make the patient exercise his limb by weight-bearing so as to produce inflammation, thus causing an extravasation of coagulated lymph and the parts are united by a kind of granulation, but he spoils this modern thought of treatment by adding "if this is unsuccessful all that can ever be necessary is to reduce it to the state of a compound fracture by a simple incision, for there are few, if any, cases in which union does not take place in compound fractures." He ends his argument by the following statement: "I will close this dissertation on fractures by recommending free use of the lancet."

Instead of rendering a patient insensible either by the use of intoxicating liquor or by blood-letting, we now resort to anæsthesia to obtain muscular relaxation.

Local anæsthesia may be used. This form of anæsthesia in the reduction of fracture was first used by Conway,[26] in 1885, who reduced three cases of Colles' fracture and one posterior dislocation of both bones of the elbow by aid of a local anæsthetic. He advised injection directly into the fracture site, even though the fracture extended into a joint or into the hæmatoma about the site of fracture. This method was used by Reclus,[27] in 1903, Lerda,[28] in 1907, Quenu,[29] in 1908 and Braun, in 1913.

Hosford[30] advocates the use of novocaine, infiltrating around the fracture or into the hæmatoma at the site of fracture, or by injecting suitable peripheral nerves.

Mage[31] reports the use of regional anæsthesia by nerve block and advocates two methods to use: first, a field block which consists in creating an encircling wall of anæsthesia by infiltrating the tissues around the operative field, and second, nerve block which consists in making novocaine injection in close proximity to the nerve or nerves whose conductivity it is desired to cut off.

Gray[32] advocates the use of novocaine plus adrenalin chloride, but Wells in the discussion advised against using adrenalin because of the fact that the addition of adrenalin retards the desired rapid absorption of the novocaine. According to Wells one does not get good relaxation in local anæsthesia for the reduction of fractures in from 25 to 35 per cent. of the cases.

Bohler does not use general anæsthesia for the reduction of fractures. He advocates local anæsthesia to reduce all recent cases. In isolated cases he uses regional anæsthesia, infiltrating the tissues around the fracture with twenty to fifty cubic centimetres of a 2 per cent. solution of novocaine. In cases of fracture which are over two weeks' duration Bohler[33] blocks the brachial plexus to reduce fractures of the arm and advocates the use of spinal anæsthesia for fractures of the lower extremity.

The advantages of local anæsthesia are:

A surgeon may attempt reduction when an assistant or anæsthetist is not available.

Reduction may be attempted in those who are too shocked to receive a general or a spinal anæsthetic, or in those who refuse a general anæsthetic.

The patient can go to the X-ray room alone.

The duration of the anæsthesia is from two to three hours and if the first attempt is not successful subsequent attempts can be made.

Individuals with fractures of the upper extremities may go home immediately unassisted.

It may also be used in cases of decompensated heart or conditions which would contra-indicate the use of general anæsthesia.

The disadvantages of local anæsthesia in the reduction of fracture are:

It cannot be satisfactorily used in very young children.

It usually takes longer than a general anæsthetic.

It is contra-indicated in open fractures and cannot be used when there is very much swelling present because it is difficult to inject novocaine exactly at the site of fracture.

Spinal anæsthesia may be used to reduce fractures of the lower extremity when general anæsthesia is contra-indicated, but cannot be used in cases of shock associated with low blood-pressure, or in certain cases where the patient, because of associated injury, cannot be placed on his side.

My personal experience with local anæsthesia having been limited, my preference is a general anæsthetic, although for a short case such as a Colles' fracture nitrous oxide and oxygen may be used. Ethylene would be ideal were it not for the fact that proper reduction must often be accomplished in the fluoroscopic room, which renders ethylene impracticable.

General anæsthesia at the present time seems preferable, but with more experience in practice with local anæsthesia it may become popularized. General anæsthesia is not without risk because if the patient is not well under he may move at the crucial moment.

It is not my intention to discuss the pros and cons of operative or non-operative treatment of fractures. In this connection I am reminded of the words of Wilhelm Von Humboldt: "I lay very little stress either upon asking or giving advice. Generally speaking, they who ask advice know what they wish to do and remain firm in their intentions. A man may allow himself to be enlightened on various points, even upon matters of expediency and duty, but after all he must determine his course of action for himself."

An attempt has been made to emphasize the importance of early reduction with the least trauma. For reasons enumerated above any mechanical device which extensively tears the periosteum or traumatizes the tissues surrounding the fragments preëmpts the probability of delayed or non-union.

A large majority of fractures, if seen sufficiently early, can be reduced under an anæsthetic. The fragments can be held in good position by means of a properly molded plaster-of-Paris splint or case, skeletal traction or open operation. When reduction of the fracture is delayed or if the fracture is associated with swelling of the soft parts or considerable shortening of fragments exists any attempt to make forcible reduction by means of severe and rapid traction will do irreparable damage to the soft parts. The method of Soutter[34] is for this reason not advocated. For the same reason, although being a great admirer of Moorehead, his instrument is thought to be too

drastic. Gradual traction by means of ice tongs or pins is preferable to any rapid method as such gradual traction produces less trauma to the soft parts.

Hippocrates[35] mentions the use of extension and states that "for the most part two strong men will suffice." He advocates, "when the parts are adjusted you should apply the bandages while the limb is in a stretched position and when the limb is bandaged it should be placed upon some smooth or soft object so as not to be distorted to one side or the other, and that there may be no protrusion of the bones backward or forward, and for this purpose nothing is more convenient than a cushion or something similar, either of linen or wool, and not hard." He states that he is at a loss to advise that "gutters" below the fractured legs should be used, but found that a board is an uncomfortable thing to have a limb laid upon unless something be placed above it, but that it is useful in making subsequent arrangement of the bed or in going to stool. He found that as the swelling subsides in a satisfactory manner the bandaged limb will become more slender, the bones will be more mobile and yield more readily to traction. He states that the bones of the leg should get consolidated in forty days if properly treated and he cautions against tight bandaging. For the swelling which complicates a fracture he advised the part should be wrapped in unscoured wool, washed with wine oil and anointed with serate before bandaging, and if splints give pain they should be slackened. In speaking of fractures of the lower limb, he states that when the outer bone of the leg is broken the patient should soon walk about, but in fractures of the inner bone it is a long time before they can walk. "It is a disgrace," he says, "to exhibit a shortened thigh, for an arm when shortened may be concealed and the mistake is not so bad." In fact, he says that "when the sound leg is placed beside the broken one, the sound one being longer than the other, exposes the mistake and, therefore, it would be to the advantage of the person who would be improperly treated that both of his legs should be broken, for in this case one would be the same length as the other." In his experience the thigh bone should be consolidated in fifty days.

In speaking of the treatment of compound fracture, Hippocrates notes that: "In those cases of fracture in which the bones protrude and cannot be restored to their place the following mode of reduction may be practiced: Some small pieces of iron are to be placed below the levers which the cutter of stone makes use of, one being rather broader and the other narrower. There should be three of them at least and still more so that you may use those that suit best, and then, along with traction, we must use these levers, applying the under surface of the piece of iron to the under fragment of bone and the upper surface to the upper bone. In a word, we must operate powerfully with the lever as we would do upon a stone or a piece of wood. The lever treatment along with traction must be had recourse to on the day of the accident or the next day, but by no means on the third, fourth or fifth day for if delayed too long convulsions are apt to occur." In other words, Hippocrates realized the fact that when operation was indicated it should be performed early.

If the fragments of a fracture cannot be satisfactorily held by conservative measures it is far better to anticipate delayed or non-union and operate early, for, as pointed out by Whitman:[36] "Operative treatment in fractures is indicated when a satisfactory reduction cannot be obtained and maintained by non-operative methods, provided there is no contra-indication and when the expected result of the open method is sufficiently better than the closed to justify the additional risk." Open operation when employed should preferably be undertaken during the first week after injury. Fractures should be reduced immediately after the injury, but only when proper fixation apparatus is at hand.

The method of operation by means of internal or external fixation or by wiring, plating or bone graft is not the theme of this paper. It is interesting, however, to note that Horeau, of France, first advocated the wiring of fractures in 1805.

Early weight-bearing with proper fixation is of great assistance in the treatment of delayed union of the lower extremity, especially the tibia and fibula.

Memmel[37] quotes the axiom of Aristotle that movement is life. He never preached that immobilization was death, but only that immobilization carried too far was responsible for many evils and certainly immobilization is frequently carried too far in the treatment of fractures.

Bohler, on the other hand, warns against too short a period of immobilization for fear that "new shortening or bending take place." He advocates early active motion of joints and early weight-bearing provided there is proper external fixation. He prefers active motion to passive motion.

Although constitutional diseases may have but little effect on union, yet naturally the general physical condition of the patient must always be considered. Beneficial effects or irradiated ergosterol, vigantol and feeding of other foods rich in vitamins has been employed. Phosphorus has been given internally and the newer osophyte has been used.[38]

Knoflach,[39] in the administration of ergosterol in doses of five to ten milligrams daily, noted an increase of the amount and density of the callus as compared with that in the controls. This increase began in the third week of fracture and was particularly great in elderly persons and children. In persons aged more than fifty-five the time required for bony union was distinctly shortened, but in other patients and other periods of life the difference in this respect was not significant.

H. A. Swart,[40] with his studies on the effect of irradiated ergosterol in the treatment of fractures of rabbits concluded that the administration of this vitamin did increase the rate of healing or the amount of callus formation in fractures of the tibia and fibula. He showed that there is some evidence that the administration of irradiated ergosterol to animals by mouth will produce a calcification in blood-vessels and other soft tissues. He found the most significant factor in the rate of healing and in the union of experimental fractures of animals is the degree of apposition of the fragments.

Israel and Frankel[41] found that fractures in guinea-pigs do not heal if the animals are kept on a diet free from vitamin C. A mere restriction of the vitamin does not impair the formation of callus.

Sir Ashley Cooper, over a hundred years ago, in his lectures on delayed and non-union, mentioned the fact that the improper diet of sailors on long cruises might be a causative factor in non-union of fracture so prevalent among these sea-faring men.

In conclusion it is urged that:

(1) More time be spent in teaching the medical student and the interne the proper mechanical factors in the treatment of fractures.

(2) Cases of fracture be considered more as emergencies which require immediate reduction.

(3) Such reductions should not be attempted unless proper fixation apparatus is at hand.

(4) An anæsthetic is usually indicated.

(5) Those cases of fracture in which the fragments cannot be properly approximated and held be operated upon early.

(6) Immobilization should not be carried over too long a period of time.

(7) Active and passive motion should be instituted early.

(8) Weight-bearing with proper external fixation is advantageous.

(9) Proper coöperation between the surgeon, his assistant, the interne staff, the laboratory and the röntgenologist be demanded.

By these methods we can improve our fracture service, although probably never reach the ideal, for "ideals are like stars; you will not succeed in touching them with your hands but, like the sea-faring man on the desert of waters, you choose them as your guides and, following them, you reach your destiny."

BIBLIOGRAPHY

[1] Wilcox: Jour. Lancet, vol. i, p. 130.
[2] Wilson, and Cochran: Quoted by Wilcox (*ibid.*).
[3] Campbell: Quoted by Cox: Jour. Tennessee State Med. Assn., vol. ii, 1921.
[4] Newell: Quoted by Cox (*ibid.*).
[5] Wilson: Jour. Am. Inst. of Homeop., vol. xxiii, 1930.
[6] Henderson: Jour. Am. Med. Assn., vol. lxxxvi, 1926.
[7] Culbertson: Bio-Chem. Jour., vol. xxiv, pt. 2, 1930.
[8] Lacey: ANNALS OF SURGERY, 1929.
[9] Bohler: Treatment of Fractures.
[10] Pat Fite: Jour. Oklahoma State Med. Assn., vol. xxiv, No. 7, July, 1931.
[11] Black: Dissertation on Fractures, 1797.
[12] Cox: *ibid.*
[13] Bankhart: Brit. Med. Jour., vol. i, p. 8, 1930.
[14] Boehler: *ibid.*
[15] Lannelongue, and Menard: Modern Surgery, DaCosta.
[16] Newell: *ibid.*
[17] Cowan: Transactions of Am. Surg. Assn., vol. xlvi, 1928.
[18] Makel: Military Surg., vol. lxiv, 1929.
[19] Groves: Brit. Jour. of Surg., vol. xviii, 1930 and 1931.
[20] Carothers: Intern. Jour. of Med. and Surg., vol. xlii, 1929.
[21] Wilcox: *ibid.*
[22] Findlay: Jour. Bone and Joint Surg., vol. xiii, No. 4, 1931.
[23] Bancroft: ANNALS OF SURGERY, vol. xc, 1929.
[24] Ashhurst: ANNALS OF SURGERY, vol. xc, 1929.
[25] Russell: Journal Col. of Surg., Australia, vol. iii, p. 32, 1930 and 1931.
[26] Conway: New York Med. Jour., vol. xlii, 1885.
[27] Reclus: L'Anestesie localisee par la cocaine. Paris, Masson & Cie, p. 266, 1903.
[28] Lerda, G.: Die fraktische Anwendung der lokal Anasthesie bie Frakturen. Zentralbl., f. Chir., vol. xxxiv, pp. 1417, 1907.
[29] Quenu: De L'anesthesie locale par la cocaine dans la reduction des fractures. Bull. et mém. Soc. de Chir. de Paris, vol. xxxiv, p. 976, 1908.
[30] Hosford: Brit. Jour. of Surg., vol. xviii, 1930 and 1931.
[31] Mage: ANNALS OF SURGERY, 1927.
[32] Gray: Jour. Florida Med. Assn., vol. xvi, 1929.
[33] Bohler: *ibid.*
[34] Soutter: Jour. Am. Med. Assn., vol. xciv, p. 1547, 1930.
[35] Hippocrates: Works of Hippocrates, vol. ii, p. 50.
[36] Whitman: Quoted by Shearer: U. S. Veteran's Bur. Med. Bull., vol. vi, 1930.
[37] Memmel: Clinical Jour., vol. lviii, 1928.
[38] Bohler: *ibid.*
[39] Knoflach: Quoted by Swart: Jour. Bone and Joint Surg., New Series, vol. xii, p. 365, 1930.
[40] Swart, H. A.: *ibid.*
[41] Israel, and Frankel: Klin. Wochenschrift, vol. v, 1926.

TRANSACTIONS
OF THE
PHILADELPHIA ACADEMY OF SURGERY

Meeting of January 8, 1929, in Thomson Hall, College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Presentation.
Dr. William Bates. — Stab wound of left ventricle.
Vol. LXXXIX, No. 4, p. 625.

Papers.
Dr. Selling Brill. — Mortality of intestinal obstruction.
(By invitation.) Discussed by Doctors Deaver, Muller, Pfeiffer and Nassau.
Vol. LXXXIX, No. 4, p. 541.

Dr. John B. Deaver. — Perforated peptic ulcer.
Vol. LXXXIX, No. 4, p. 529. Discussed by Doctors Nassau, Muller and Deaver.

Meeting of February 4, 1929, in the Hall of the College of Physicians. Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Presentation.
Dr. Alexander Randall. — X-ray destruction of kidney.
Vol. LXXXIX, No. 6, p. 949. Discussed by Doctors Stellwagen and Randall.

Case Reports.
Dr. B. A. Thomas. — Nephrectomy for unilateral polycystic kidney.
Vol. LXXXIX, No. 6, p. 946. Discussed by Doctors Thomas, Nassau and Outerbridge.

Dr. Leon Herman
Dr. Lloyd B. Greene. — New growths of the renal pelvis.
(By invitation.) Discussed by Doctors Bishop, Bauer, Thomas, Greene, Stellwagen and Randall.
Vol. LXXXIX, No. 6, p. 682.

Presentation of Dressing.
Dr. Thomas C. Stellwagen. — New scrotal dressing.
Vol. LXXXIX, No. 6, p. 954.

The volume and page number following the name of the author refers to the location of the article in Annals of Surgery.

Annual Conjoint Meeting with the New York Surgical Society, February 13, 1929. Dr. Frank S. Mathews, President of the New York Surgical Society, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Cases.
Dr. Carl Eggers.
Vol. XC, No. 1, p. 118.
1. Tuberculosis of the thyroid gland with secondary lymph-node involvement.
2. Gastrotomy for hæmorrhage following gastroenterostomy.
Discussed by Doctor Billings.

Dr. James I. Russell.
Vol. XC, No. 1, p. 121.
1. Pyonephrosis.
Discussed by Doctor Herman.
2. Carcinoma of cæcum—intussusception.
Discussed by Doctor Jopson.

Dr. Franz Torek.
Vol. XC, No. 1, p. 124. Carcinoma of the tonsil and adjacent tissues.

Dr. Allen O. Whipple.
Vol. XC, No. 1, p. 127. Mycotic cysts of the liver.
Discussed by Doctors Brickner, Speece and Whipple.

Papers.
Dr. Richard Lewisohn.
Vol. XC, No. 1, p. 69. Factors of safety in resection of the stomach for gastroduodenal ulcers.
Discussed by Doctor Muller.

Dr. Eugene H. Pool.
Vol. XC, No. 1, p. 132.
1. Reconstruction of common duct.
Discussed by Doctors Klopp and Nassau.
2. Diverticulum of duodenum.
Discussed by Doctors Gibbon and Mathews.

Dr. Walton Martin.
Vol. XC, No. 1, p. 47. The spread of bacteria from the gall-bladder to the liver.
Discussed by Doctor Ravdin.

Meeting of March 4, 1929, in the Hall of the College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Papers.

Dr. Charles H. Frazier.
Vol. LXXXIX, No. 6, p. 801.

Use of iodized rape-seed oil for röntgenographic exploration.

Dr. James Lacey.
(By invitation.)
Vol. LXXXIX, No. 6, p. 813.

Non-union of fractures.
Discussed by Doctor Eldridge L. Eliason.

Dr. Charles H. Frazier.
Dr. W. B. Mosser.
(By invitation.)
Vol. LXXXIX, No. 6, p. 849.

The effect of iodine and thyroid feeding on the thyroid gland.

Dr. Selling Brill.
(By invitation.)
Vol. LXXXIX, No. 6, p. 857.

Effect of abdominal thermal applications on the intraperitoneal temperature.
Discussed by Doctor Bartgis McGlone.

Dr. I. S. Ravdin.
Dr. C. M. Smyth, Jr.
Dr. M. E. Morrison.
(By invitation.)
Vol. LXXXIX, No. 6, p. 878.

Bile ascites and bile peritonitis.
Discussed by Doctor George P. Muller.

Dr. Norman Rothschild.
Vol. LXXXIX, No. 6, p. 878.

Safety factors in mesenteric ligations.
Discussed by Doctor John H. Jopson.

Dr. Frederick A. Bothe.
Vol. LXXXIX, No. 6, p. 886.

The fate of the free omental graft in abdominal surgery.
Discussed by Doctor John Speese.

Meeting of April 1, 1929, in the Hall of the College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. J. Stewart Rodman.
Vol. XC, No. 3, p. 433.

1. Multiple myeloma.
2. Extensive metastasis to bone from carcinoma of breast.

Dr. Hubley R. Owen.
Dr. Temple Fay.
(By invitation.)
Vol. XC, No. 3, p. 434.

Chordotomy for gastric crises complicated by acute intestinal obstruction.
Discussed by Doctors Grant and Fay.

Dr. Hubley R. Owen.
Vol. XC, No. 3, p. 440.

Gastric crisis complicating cancer of pylorus.
Discussed by Doctors Nassau, Lee and Scott.

Dr. Calvin M. Smyth, Jr.
Vol. XC, No. 3, p. 441.

Post-operative massive atelectasis: bronchoscopic aspiration.
Discussed by Doctors Nassau and Lee.

Presentation of Case.
Dr. Benjamin Lipshutz.
Vol. XC, No. 3, p. 444.

Bullet wound of intestines and kidney with nephrectomy.
Discussed by Doctor Nassau.

Paper.
Dr. John H. Gibbon.
Vol. XC, No. 3, p. 367.

Review of operations done on the gall-bladder and ducts.
Discussed by Doctors Jopson, Muller, Rodman, Smyth, Ashhurst and Gibbon.

Meeting of May 7, 1929, in Thomson Hall, College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Case.
Dr. DeForest P. Willard.
Vol. XC, No. 3, p. 448.

Tendon transplantation for wrist drop.
Discussed by Doctor Elmer.

Case Reports.
Dr. Frederick A. Bothe.
Vol. XC, No. 3, p. 449.

Adenoma of thyroid with torsion of larynx.

Dr. John B. Flick.
Vol. XC, No. 3, p. 450.

Gangrenous infection of the hand and forearm following human bite.
Discussed by Doctor Owen.

Dr. Eldridge L. Eliason.
Dr. James Lacey.
(By invitation.)
Vol. XC, No. 3, p. 451–3.

1. Case of Diffuse Gangrenous Pancreatitis.
2. Incarcerated retrocæcal hernia containing an acute gangrenous appendix.
3. Perforated peptic ulcer.

Annual Oration.
Dr. Edward J. Klopp.
Vol. XC, No. 3, p. 454.

Surgery in breast tumors.

Meeting of October 7, 1929, in the Hall of the College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Case.
Dr. Hubley R. Owen. — Duodenal ulcer complicating appendicitis.
Vol. XCI, No. 2, p. 303.

Case Reports.
Dr. William Bates.
Dr. J. Gershon-Cohen. (By invitation.) — The association of vertebral fractures with fractures of the os calcis.
Vol. XCI, No. 2, p. 304.

Dr. George M. Dorrance. — Pulsating exophthalmos. Discussed by Doctors Jopson and Shallow.
Vol. XCI, No. 2, p. 306.

Paper.
Dr. P. A. McCarthy. — Treatment of aneurysms of the thoracic aorta and innominate artery by distal arterio-venous anastomosis. Discussed by Doctor Muller.
Vol. XCI, No. 2, p. 161.

Meeting of November 4, 1929, in the Hall of the College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.
Dr. Ralph Goldsmith. (By invitation.) — 1. Bleeding œsophageal varices due to hepatic cirrhosis. Discussed by Doctors Ravdin, Owen and Goldsmith. 2. Subacute hæmorrhagic pancreatitis.
Vol. XCI, No. 3, p. 427.

Dr. Damon B. Pfeiffer. — Recurrent tuberculous lymphadenitis of the axilla and neck. Discussed by Doctors Eiman, Pfeiffer, Jopson and Dorrance.
Vol. XCI, No. 3, p. 432.

Papers.
Dr. F. S. Hammett. (By invitation.) — Natural chemical stimulus for cell division.
Vol. XCI, No. 3, p. 435.

Dr. Stanley P. Reimann. (By invitation.) — The use of the natural chemical stimulus for cell division in wound healing. Discussed by Doctor Ashhurst.
Vol. XCI, No. 3, p. 435.

Meeting of December 2, 1929, in the Hall of the College of Physicians. The President, Dr. Astley P. C. Ashhurst, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Case.
Dr. Norman Rothschild. — Hemolytic icterus with multiple abscesses of spleen, splenectomy.
Vol. XCI, No. 3, p. 436.

Case Reports.
Dr. Edward J. Klopp. — Perforated duodenal ulcer with multiple sequelæ.
Vol. XCI, No. 3, p. 438.

Dr. H. F. Ulrich. (By invitation.) — Ruptured adenoma of the thyroid gland.
Vol. XCI, No. 3, p. 440.

Papers.
Dr. Richard H. Overholt. (By invitation.) — Phrenic nerve stimulation in diaphragmatic hernia. Discussed by Doctor Muller.
Vol. XCI, No. 3, p. 381.

Dr. George P. Muller. — Mortality and end-results of operation for abscess of the lung.
Vol. XCI, No. 3, p. 361.

SECRETARY'S REPORT FOR YEAR 1929

During the year 1929 there were held eight regular meetings of the Philadelphia Academy of Surgery; one joint meeting of the Philadelphia Academy of Surgery with the New York Surgical Society which was held at the Medical Center in New York City on February 13, 1929; and one special meeting of the Philadelphia Academy of Surgery, held on April 22, 1929, at 3 P.M., in the Mitchell Hall of the College of Physicians, in celebration of the fiftieth anniversary of the founding of the Philadelphia Academy of Surgery.

The average attendance at the regular meeting was twenty-four Fellows and twenty-six guests.

During the year there were fifteen cases reported with seventeen discussions; nine presentations of patients with ten discussions; sixteen papers with

thirty-three discussions; one exhibition of surgical apparatus and one moving-picture demonstration.

The following were elected to Fellowship during the year: Dr. Louis D. Englerth, Dr. Henry K. Seelaus, Dr. P. A. McCarthy, Dr. Albert E. Bothe.

The following were elected to Honorary Fellowship: *Foreign.*—Sir D'Arcy Power, London, England; Dr. Albin Lambotte, Esneux, Belgium; Dr. Henry Hartmann, Paris, France; Dr. Th. Tuffier, Paris, France; Dr. Joseph Guyot, Bordeaux, France; Dr. Georges Jeanneney, Bordeaux, France; Dr. F. DeQuervain, Berne, Switzerland; Baron Berkeley Moynihan, Leeds, England. *American.*—Dr. Harvey Cushing, Boston, Mass.; Dr. Edward W. Archibald, Montreal, Canada; Dr. John M. T. Finney, Baltimore, Md.; Dr. Evarts Graham, St. Louis, Mo.; Dr. Ellsworth Eliot, Jr., New York City, N. Y.; Dr. Rudolph Matas, New Orleans, La.; Dr. Dean D. Lewis, Baltimore, Md.; Dr. Eugene H. Pool, New York City, N. Y.; Dr. George W. Crile, Cleveland, Ohio; Dr. Edward Starr Judd, Rochester, Minn.; Dr. Dallas B. Phemister, Chicago, Ill.

HUBLEY R. OWEN, M.D., *Secretary.*

Meeting of January 7, 1930, in Thomson Hall, College of Physicians. The President, DR. ASTLEY P. C. ASHHURST, in the Chair.

SCIENTIFIC PROGRAM

Papers.

DR. WALTER ESTELL LEE.
DR. HARRY B. WILMER.
DR. HERBERT MARSHALL COBE. (By invitation.)
Vol. XCI, No. 5, p. 651.
Allergy and asthma in post-operative atelectasis.
Discussed by Doctor Lee.

DR. PAUL N. JEPSON. (By invitation.)
Vol. XCI, No. 5, p. 724.
Obstetrical paralysis.
Discussed by Doctors Gill, Ashhurst and Shallow.

DR. THOMAS J. RYAN. (By invitation.)
Vol. XCI, No. 5, p. 714.
Mortality from appendicitis.
Discussed by Doctors Bower, Crossan, Owen, Ashhurst and Ryan.

Presentation of Case.

DR. THOMAS A. SHALLOW.
Vol. XCI, No. 5, p. 761.
Oxycephalia.
Discussed by Doctors Grant and Lipshutz.

Annual Conjoint Meeting with the New York Surgical Society, February 13, 1930, at the Philadelphia General Hospital. The President, DR. GEORGE P. MULLER, in the Chair.

Papers.

DR. I. S. RAVDIN.
Vol. XCI, No. 6, p. 801.
Coagulation of blood.
Discussed by Doctor Bancroft.

DR. JOHN BERTON CARNETT.
Vol. XCI, No. 6, p. 811.
Bone metastasis in cancer of the breast.
Discussed by Doctors Lee, Semken and Carnett.

DR. ELDRIDGE L. ELIASON.
Vol. XCI, No. 6, p. 833.
Osteitis fibrosa.
Discussed by Doctors Douglas and Beekman.

DR. CHARLES H. FRAZIER.
Vol. XCI, No. 6, p. 848.
Spasmodic torticollis.
Discussed by Doctor Stookey.

DR. JOHN B. DEAVER.
Vol. XCI, No. 6, p. 841.
Curability of cancer.
Discussion by Doctor Rorek.

Presentation of Cases.

DR. HENRY P. BROWN, JR.
Vol. XCI, No. 6, p. 945.
Traumatic rupture of the diaphragm.
Discussed by Doctor Lilienthal.

DR. ROBERT H. IVY.
DR. LAWRENCE CURTIS. (By invitation.)
Vol. XCI, No. 6, p. 947.
Fractures of the mandible.
Discussed by Doctor Moorhead.

DR. JOHN H. JOPSON.
Vol. XCI, No. 6, p. 952.
Hour-glass stomach.
Discussed by Doctors Eggers, Gibson and Jennings.

Case Report.

DR. JOHN H. JOPSON.
Vol. XCI, No. 6, p. 951.
Pancreatic cyst.
Discussed by Doctor Jopson.

Paper.

DR. ALEXANDER RANDALL.
Vol. XCI, No. 6, p. 960.
Water intoxication.
Discussed by Doctor Beer.

Meeting of March 3, 1930, in Thomson Hall, College of Physicians. The President, DR. GEORGE P. MULLER, in the Chair.

Papers.

DR. GEORGE M. DORRANCE.
Vol. XCII, No. 2, p. 161.
Osteoperiosteal bone grafts.
Discussed by Doctors Wagoner, Ivy, Ashhurst and Dorrance.

DR. JOHN P. NORTH. (By invitation.)
Vol. XCII, No. 2, p. 277.
Welch bacillus antitoxin in intestinal obstruction.
Discussed by Doctor Eliason.

Dr. I. S. Ravdin.
Archives of Surgery, May, 1931.
Contractile function of the gall-bladder.
Discussed by Doctor Jopson.

Dr. James T. Lacey.
(By invitation.)
Vol. XCII, No. 2, p. 281.
Peritoneal adhesions.
Discussed by Doctor Pfeiffer.

Dr. Richard H. Overholt.
(By invitation.)
Intraperitoneal pressure.

Dr. Albert E. Bothe.
Vol. XCII, No. 2, p. 300.
Vesical neck obstruction.

Meeting of April 7, 1930, in Thomson Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. Benjamin F. Buzby.
Vol. XCII, No. 2, p. 302.
Reconstruction of an arm in brachial plexus injury.

Dr. Charles H. Frazier.
Vol. XCII, No. 2, p. 306.
Chronic arthritis showing improvement following lumbar sympathectomy.

Papers.

Dr. Thomas Shallow.
Vol. XCII, No. 2, p. 182.
Traumatic brachial plexus paralysis.

Dr. Lee A. Rademaker.
(By invitation.)
Vol. XCII, No. 2, p. 195.
Cause and elimination of reactions after intravenous infusions.

Dr. Edward W. Saunders.
(By invitation.)
Vol. XCII, No. 2, p. 222.
Gastric ulcer.
Discussed by Doctor Sweet.

Meeting of May 5, 1930, in Thomson Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. J. R. Veal.
(By invitation.)
Vol. XCII, No. 6, p. 1097.
Foreign body in heart.

Dr. William B. Swartley.
Vol. XCII, No. 6, p. 1093.
Intrahepatic cholelithiasis.

Dr. Edward J. Klopp.
Vol. XCII, No. 6, p. 1096.
Removal of rubber tube from common bile-duct.
Discussed by Doctor Muller.

Papers.

Dr. Bruce L. Fleming.
(By invitation.)
Vol. XCIII, No. 3, p. 730.
Cholecystostomy.

Annual Oration.

Dr. Edward T. Crossan.
Vol. XCII, No. 6, p. 1019.
Lymph exudate and fibrous tissue.

Meeting of October 6, 1930, in Cadwalader Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. George M. Dorrance.
Vol. XCII, No. 6, p. 1099.
Necrosis of bones of forearm following traumatic removal of periosteum.

Dr. Benjamin F. Buzby.
Vol. XCII, No. 6, p. 1100.
Blastomycetic osteomyelitis of femur.
Discussed by Doctor Buzby.

Papers.

Dr. Irvin E. Deibert.
(By invitation.)
Vol. XCII, No. 6, p. 1087.
Treatment of osteomyelitis.
Discussed by Doctors Eliason, Buzby, Willard and Rademaker.

Dr. Francis C. Grant.
Vol. XCII, No. 6, p. 998.
Cordotomy for the relief of pain.
Discussed by Doctor Frazier.

Meeting of November 3, 1930, in Cadwalader Hall, College of Physicians. The Vice-President, Dr. John Speese, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patient.

Dr. Richard H. Overholt.
(By invitation.)
Vol. XCIII, No. 3, p. 779.
Phrenic nerve exeresis for lung abscess.

Case Report.

Dr. Leonard G. Dobson.
(By invitation.)
Vol. XCIII, No. 3, p. 775.
Primary giant-cell tumor of the patella.
Discussed by Doctor Gill.

Papers.

DR. WILLIAM BATES.
Vol. XCIII, No. 3, p. 781.

Electrocauterization in treatment of human bites.
Discussed by Doctors Owen, Flick, Harkins and Bates.

DR. ELI SALEEBY.
DR. M. J. HARKINS.
(By invitation.)

Comparative studies of antiseptics in experimentally produced local infections.

DR. GEORGE P. MULLER.
(By invitation.)
DR. LEE RADEMAKER.
Vol. XCIII, No. 3, p. 755.

End-results in radical operations for carcinoma of the periampullar region.

Meeting of December 1, 1930, in Cadwalader Hall, College of Physicians. The President, DR. GEORGE P. MULLER, in the Chair.

SCIENTIFIC PROGRAM

Case Report.

DR. S. DANA WEEDER.
Vol. XCIII, No. 3, p. 782.

Perforated diverticulitis of the sigmoid.
Discussed by Doctors Pfeiffer and Muller.

Papers.

DR. JOHN JEFFRIES.
(By invitation.)
Vol. XCIII, No. 3, p. 761.

Torsion of the great omentum.
Discussed by Doctor Swartley.

DR. BENJAMIN LIPSHUTZ.
Vol. XCIII, No. 3, p. 766.

Carbuncle of the kidney.
Discussed by Doctors Laws, Muller and Lipshutz.

DR. GEORGE M. DORRANCE.
Vol. XCIII, No. 3, p. 786.

Cæcal drainage in acute suppurative appendicitis.
Discussed by Doctors Ravdin, Pfeiffer and Dorrance.

SECRETARY'S REPORT FOR YEAR 1930

DURING the year 1930 there were held seven regular meetings of the Philadelphia Academy of Surgery and one joint meeting of the Academy with the New York Surgical Society which was held in Philadelphia on February 13, 1930, in the Auditorium of the Philadelphia General Hospital.

The average attendance at the regular meeting was twenty-seven Fellows and eighteen guests.

During the year there were eleven cases reported with twenty-two discussions; three presentations of patients with five discussions; thirty papers with seventy-six discussions and one presentation of specimen.

The Annual Address in Surgery was delivered by Dr. Edward T. Crossan.

The following were elected to Fellowship during the year: Dr. Thomas J. Ryan, Dr. Irvin E. Deibert, Dr. Harry E. Knox.

The following deaths occurred during 1930: Dr. Hiram R. Loux, Dr. B. A. Thomas, Dr. E. G. Alexander.

HUBLEY R. OWEN, M.D., *Secretary.*

Meeting of January 5, 1931, in Thomson Hall, College of Physicians. The President, DR. GEORGE P. MULLER, in the Chair.

SCIENTIFIC PROGRAM

Presentation.

DR. GEORGE M. LAWS.
Vol. XCIII, No. 5, p. 1095.

Tuberculous peritonitis and recurrent umbilical hernia.
Discussed by Doctor Muller.

Case Report.

DR. GEORGE M. LAWS.
Vol. XCIII, No. 5, p. 1096.

Pendulous abdomen—lipectomy.

Paper.

DR. HENRY P. BROWN, JR.
Vol. XCIII, No. 5, p. 1075.

Subphrenic abscess.
Discussed by Doctors Eliason, Owen, Shallow and Lipshutz.

Annual Oration (for 1930).
DR. J. STEWART RODMAN.
Vol. XCIII, No. 5, p. 1017.

Surgical management of cranial injuries.

Meeting of February 2, 1931, in Thomson Hall, College of Physicians. The President, DR. GEORGE P. MULLER, in the Chair.

SCIENTIFIC PROGRAM

Presentation.

DR. JOHN B. FLICK.
Vol. XCIII, No. 6, p. 1240.

Lobectomy for bronchiectasis.

DR. DEFOREST P. WILLARD.
Vol. XCIII, No. 6, p. 1242.

Nicola operation for recurrent dislocation of the shoulder.
Discussed by Doctors Thomas, Nassau and Englerth.

Case Reports.

Dr. Eldridge L. Eliason. Vol. XCIII, No. 6, p. 1243.	Angio-endothelioma of the humerus.
Dr. Eldridge L. Eliason. Vol. XCIII, No. 6, p. 1245.	Traumatic emphysematous bulla. Discussed by Doctors Eliason and Flick.

Papers.

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The Annual Conjoint Meeting of the Philadelphia Academy of Surgery and New York Surgical Society was held at the Academy of Medicine, New York, on February 11, 1931.

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Dr. Charles L. Gibson (of New York). Vol. XCIV, No. 1, p. 117.	Exhibition of patient following operation for large ventral hernia. Discussed by Doctors George M. Laws and George P. Muller.
Dr. Burton J. Lee (of New York). Vol. XCIV, No. 1, p. 117.	Exhibition of patients illustrating the results of irradiation of primary operable carcinoma of breast. Discussed by Doctors John B. Carnett, of Philadelphia, Eugene H. Pool and Burton J. Lee, of New York.
Dr. Franz Torek (of New York). Vol. XCIV, No. 1, p. 121.	Treatment of undescended testicle. Discussed by Doctors Hubley R. Owen, of Philadelphia, William B. Coley, and Franz Torek, of New York.
Dr. Byron Stookey (of New York). Vol. XCIV, No. 1, p. 123.	Hemilaminectomy for exploration. Discussed by Doctors Charles H. Frazier, and Thomas A. Shallow, of Philadelphia, and Byron Stookey, of New York.

Dr. Richard Lewisohn (of New York). Vol. XCIV, No. 1, p. 80.	Hæmatologic studies as a basis for determining the surgical risk in jaundiced patients. Discussed by Doctor I. S. Ravdin.
Dr. John F. Connors (of New York). Vol. XCIV, No. 1, p. 38.	Treatment of empyæma and lung abscess by packing. Discussed by Doctors John B. Flick and George D. Stewart.
Dr. Fordyce B. St. John (of New York). Vol. XCIV, No. 1, p. 126.	Study of cases of carcinoma of the stomach treated at the Presbyterian Hospital of New York, 1916–30. Discussed by Doctor John B. Gibbon.
Dr. Otto C. Pickhardt (of New York). Vol. XCIV, No. 1, p. 128.	Bilateral phrenicectomy for persistent hiccough. Discussed by Doctor George P. Muller.
Dr. H. H. M. Lyle (of New York). Vol. XCIV, No. 1, p. 131.	Plastics for bone cavities. Discussed by Doctor B. Franklin Buzby.
Dr. William F. MacFee (of New York). Vol. XCIV, No. 1, p. 135.	Filarial lymphatic varix of the breast.
Dr. William Crawford White (of New York). Vol. XCIV, No. 1, p. 139.	Wilm's mixed tumor of the kidney. Discussed by Doctors Albert E. Bothe and Damon B. Pfeiffer.
Dr. John E. Jennings (of New York). Vol. XCIV, No. 1, p. 142.	Acute osteomyelitis of the vertebræ. Discussed by Doctor A. B. Gill.

Meeting of March 2, 1931, in Thomson Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

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Dr. Hubley R. Owen. Vol. XCIV, No. 3, p. 436.	Primary carcinoma of the œsophagus and stomach.
Dr. J. Bernhard Mencke. Vol. XCIV, No. 3, p. 436.	Intussusception of ileum.

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Dr. B. Franklin Buzby.
Vol. XCIV, No. 3, p. 397. — Internal derangements of the knee. Discussed by Doctors Elmer and Gill.

Meeting of April 6, 1931, in Thomson Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

SCIENTIFIC PROGRAM

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Dr. John B. Deaver.
Vol. XCIV, No. 3, p. 381. — Cancer of the uterus. Discussed by Doctors Nassau, Keene and Deaver.

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SCIENTIFIC PROGRAM

Presentation of Cases.

Dr. Hubley R. Owen.
Dr. Samuel B. Hadden.
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Vol. XCIV, No. 5, p. 945. — Multiple neuritis following prophylactic injection of tetanus antitoxin. Discussed by Doctor Owen.

Dr. Frederick A. Bothe.
Vol. XCIV, No. 5, p. 947. — Inflammatory reaction of the loops following gastroenterostomy. Discussed by Doctors Pfeiffer, Crossan, Muller and Bothe.

Case Report.

Dr. Hubley R. Owen.
Vol. XCIV, No. 5, p. 943. — Intrahepatic calculus-calculous cholecystitis common duct stone.

Papers.

Dr. Arthur E. Billings.
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Dr. Joseph Kreiselman, Washington, D. C.
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Vol. XCIV, No. 5, p. 885. — Avertin anæsthesia from the anæsthetist's standpoint.

Dr. Charles S. White, Washington, D. C.
(By invitation.)
Vol. XCIV, No. 5, p. 888. — Avertin anæsthesia from the surgeon's standpoint. Discussed by Doctors Frazier, Muller, Beach, White and Kreiselman.

Meeting of October 5, 1931, in Cadwalader Hall, College of Physicians. The Vice-President, Dr. John Speese, in the Chair.

SCIENTIFIC PROGRAM

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Dr. William J. Ryan.
Vol. XCV, No. 2, p. 295. — Duodenal ulcer. Unusual recurrences.

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Vol. XCV, No. 2, p. 296. — Osteochondritis dissecans of the femur.

Dr. V. W. Murray Wright.
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Dr. Adolph A. Walkling.
Vol. XCV, No. 2, p. 298. — Perforation of Meckel's diverticulum by a fish bone.

Presentation.

Dr. Eldridge L. Eliason.
Vol. XCV, No. 2, p. 299. — Traumatic rupture of the jejunum. Discussed by Doctor Pfeiffer.

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Dr. William R. Gilmour.
Vol. XCV, No. 2, p. 161. — Fractures of the pelvis.

Meeting of November 2, 1931, in Cadwalader Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

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Discussed by Doctor Smyth.

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Discussed by Doctors Crossan and John.

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Vol. XCV, No. 2, p. 308.
Forward dislocation of the knee.

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Vol. XCVI, No. 1.
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Discussed by Doctors Dorrance and Rugh.

Dr. Charles H. Frazier.
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Meeting of December 7, 1931, in Cadwalader Hall, College of Physicians. The President, Dr. George P. Muller, in the Chair.

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Vol. XCV, No. 5, p. 771.
Osteogenesis imperfecta tarda.

Dr. Astley P. C. Ashhurst.
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Vol. XCV, No. 5, p. 776.
Case of interlobar empyæma discharging through a bronchus (pleural vomica) and exploratory thoracotomy and extrapleural thoracoplasty.

Dr. I. E. Deibert.
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Compound fracture. Gas gangrene.
Discussed by Doctors Deibert, Hodge, Smyth, Speese, Pfeiffer, Brown and Rodman.

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Vol. XCV, No. 5, p. 788.
Fracture of both femora with unusual complications.
Discussed by Doctor Mecray.

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Vol. XCV, No. 5, p. 773.
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Annual Oration (for 1931).
Dr. Hubley R. Owen.
Vol. XCV, No. 5, p. 759.
Problem of delayed union and ununited fractures.
Discussed by Doctor Eliason.

SECRETARY'S REPORT FOR THE YEAR 1931

During the year 1931 there were held eight regular meetings of the Philadelphia Academy of Surgery, and one joint meeting of the Academy with the New York Surgical Society which was held in New York City, February 11, 1931, in the New York Academy of Medicine, at 2:45 P.M.

The average attendance at the regular meetings was thirty-three Fellows and thirty-six guests.

During the year there were twenty-four cases reported with twenty-five discussions; six presentations of patients with eight discussions; fourteen papers with nineteen discussions; one report with two discussions and one demonstration of X-rays with three discussions.

The Annual Oration for 1930 was delivered by Dr. J. Stewart Rodman; the Annual Oration for 1931 was delivered by Dr. Hubley R. Owen.

The following were elected to membership during the year 1931: Dr. Bruce Fleming, Dr. Paul Mecray, Dr. R. H. Meade, Jr., Dr. L. K. Ferguson.

The following deaths occurred during the year 1931: Dr. Harry C. Deaver, Dr. John B. Deaver.

De Forest P. Willard, M.D., *Secretary.*

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