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TRANSACTIONS

OF THE

PHILADELPHIA

ACADEMY OF SURGERY

VOLUME XXIII

1921-1922-1923
1924-1925

PHILADELPHIA
PRINTED FOR THE ACADEMY
1926

NOTICE

With this issue the Business Committee announce the inauguration of a change in plan in the publication of the *Transactions*. In as much as all original contributions are published in full in the *Annals of Surgery*, the Academy of Surgery has decided not to duplicate their publication and to confine the contents of the volume to material of immediate interest to the Academy as an organization. The annual orations are reprinted in full and the programs otherwise given by title only. The Constitution and By-Laws are included for the first time. The present volume contains the proceedings of the Academy from January, 1921 to December, 1925 inclusive.

LIPPINCOTT PRESS, PHILADELPHIA

CONSTITUTION AND BY-LAWS

(As Amended to 1925)

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 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.

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, 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 monthly stated meetings, except in June, July, August and September, and special meetings may be held on the call of the President or on the call of any two 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 assign to each Fellow in alphabetical order the duty of the preparation and presentation of a paper for one of the meetings held during the year, 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 Fellows, who shall vouch for the character of the candidate, and declare immediately prior to the election his fitness for Fellowship. 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 one year.

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. 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 striken 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, 1926

President

CHARLES F. MITCHELL, M.D.

Vice-Presidents

ASTLEY P. C. ASHHURST, M.D.
GEORGE P. MULLER, M.D.

Secretary

HUBLEY R. OWEN, M.D.

Treasurer

WILLIAM B. SWARTLEY, M.D.

Recorder

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

Business Committee

DAMON B. PFEIFFER, M.D.
DEFOREST P. WILLARD, M.D.

Council

JOHN SPEESE, M.D.
EDWARD B. HODGE, M.D.
With the President, First Vice-President, Secretary and Treasurer

Trustees of the Samuel D. Gross Prize and Library

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

VICE-PRESIDENTS

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

SECRETARY

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

CORRESPONDING SECRETARY

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

TREASURER

ELECTED		ELECTED	
1880	WILLIAM HUNT	1911	EDWARD B. HODGE
1891	WILLIAM G. PORTER	1920	DUNCAN L. DESPARD
1904	JAMES P. HUTCHINSON	1922	WILLIAM B. SWARTLEY

RECORDER

ELECTED		ELECTED	
1880	JOHN B. ROBERTS	1902	JOHN H. GIBBON
1881	DEFOREST WILLARD	1905	JOHN H. JOPSON
1884	C. B. G. DENANCREDE	1915	JOHN SPEESE
1884	J. EWING MEARS	1920	HENRY P. BROWN, JR.
1891	LEWIS W. STEINBACH	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		ELECTED	
1880	JOHN ASHHURST, JR.	1910	J. CHALMERS DACOSTA
1880	JOHN H. BRINTON	1920	CHARLES H. MITCHELL
1894	WILLIAM B. HOPKINS	1922	GEORGE G. ROSS
1895	HENRY R. WHARTON	1922	JAMES H. BALDWIN
1898	THOMAS R. NEILSON	1923	WILLIAM J. TAYLOR
1900	W. JOSEPH HEARN	1924	JOHN H. JOPSON
1902	ROBERT G. LECONTE	1924	JOHN SPEESE
1906	THOMAS R. NEILSON	1925	EDWARD B. HODGE

With President, Vice-President, Secretary and Treasurer.

PUBLICATION COMMITTEE

ELECTED		ELECTED	
1880	JOHN H. PACKARD	1880	WILLIAM W. KEEN

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

BUSINESS COMMITTEE

ELECTED		ELECTED	
1895	WILLIAM J. TAYLOR	1916	W. E. LEE
1895	DEFOREST WILLARD	1916	MORRIS BOOTH MILLER
1896	RICHARD H. HARTE	1917	DAMON B. PFEIFFER
1897	ROBERT G. LECONTE	1917	A. P. C. ASHHURST
1900	G. G. DAVIS	1919	A. BRUCE GILL
1902	JOHN H. JOPSON	1919	J. STEWART RODMAN
1905	GEORGE G. ROSS	1920	ARTHUR BILLINGS
1908	FRANCIS T. STEWART	1922	DAMON B. PFEIFFER
1914	JOHN SPEESE	1924	DEFOREST P. WILLARD

COMMITTEE ON SAMUEL D. GROSS PRIZE FUND AND LIBRARY

1884–1891	1892–1893
D. HAYES AGNEW	J. EWING MEARS
SAMUEL W. GROSS	SAMUEL ASHHURST
J. EWING MEARS	WILLIAM HUNT
SAMUEL ASHHURST	JOHN ASHHURST, JR.
WILLIAM HUNT	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 Samuel Hunt to serve with them on distribution of the prize.

1895–1899	1905
J. EWING MEARS	WILLIAM J. TAYLOR
JOHN ASHHURST, JR.	RICHARD H. HARTE
WILLIAM W. KEEN	DEFOREST P. WILLARD
1900–1901	**1910**
WILLIAM W. KEEN	WILLIAM J. TAYLOR
J. EWING MEARS	RICHARD H. HARTE
J. CHALMERS DACOSTA	JOHN H. GIBBON
1902–1904	**1915**
WILLIAM J. TAYLOR	WILLIAM J. TAYLOR
WILLIAM L. RODMAN	JOHN H. JOPSON
JOHN B. ROBERTS	EDWARD B. HODGE
1920	**1925**
WILLIAM J. TAYLOR	WILLIAM J. TAYLOR
JOHN H. JOPSON	JOHN H. JOPSON
EDWARD B. HODGE	EDWARD B. HODGE

ACTIVE FELLOWS OF THE PHILADELPHIA ACADEMY OF SURGERY

1910. Alexander, Emory Graham, M.D., 337 South Eighteenth Street. Assistant Professor of Surgery at University of Pennsylvania; Surgeon to the Episcopal Hospital, St. Christopher's Hospital, Abington Hospital, Philadelphia Hospital for Contagious Diseases.

1905. Allen, Francis Olcott, A.B., M.D., 2216 Walnut Street. Surgeon to the Presbyterian Hospital, Children's Hospital, and the Bryn Mawr Hospital.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., F.A.C.S., 257 South Sixteenth Street. Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases.

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

1922. Beltran, Basil R., A.M., M.D., F.A.C.S., 2115 Chestnut Street. Surgeon to the Misericordia Hospital.

1915. Billings, Arthur E., M.D., 2020 Spruce Street. Attending Surgeon to the Pennsylvania Hospital, and Bryn Mawr Hospital; Assistant Surgeon to the Jefferson Hospital; Demonstrator in Surgery at the Jefferson Medical College.

1898. Boger, John A., A.M., M.D., 2213 N. Broad Street. Surgeon to St. Mary's Hospital; Senior Surgeon to the Stetson Hospital.

1921. Boykin, Irvine M., M.D., Elkins Park. Associate Surgeon to the Episcopal Hospital; Instructor in Surgery, Medical School, University of Pennsylvania; Assistant in Surgery, Graduate School of Medicine, University of Pennsylvania.

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

1919. Brown, Henry P., Jr., B.S., M.D., 2134 Spruce Street. Surgeon to the Pennsylvania Hospital and Children's Hospital; Assistant Surgeon to the Presbyterian Hospital.

1923. BUZBY, B. FRANKLIN, A.B., M.D., F.A.C.S., 1924 Spruce Street. Orthopædist, Cooper Hospital, Camden, N. J.; Assistant Surgeon to the Philadelphia Orthopædic Hospital; Assistant Instructor of Surgery at the University of Pennsylvania.

1907. CARMANY, HARRY S., M.D., 366 Green Lane, Roxborough, Pennsylvania. 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. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to Medico-Chirurgical Hospital, Polyclinic Hospital, Philadelphia General Hospital, American Stomach Hospital and Babies Hospital.

1916. CLARK, JOHN GOODRICH, M.D., 2017 Walnut Street. Professor of Gynæcology, University of Pennsylvania; Gynæcologist-in-Chief, University of Pennsylvania Hospital.

1919. CROSSAN, EDWARD T., M.D., 257 South Sixteenth Street. Associate Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Instructor in Surgery, University of Pennsylvania.

1896. DACOSTA, JOHN CHALMERS, M.D., LL.D., 2045 Walnut Street. Samuel D. Gross, Professor of Surgery, Jefferson Medical College; Surgeon to the Jefferson Medical College Hospital; Consulting Surgeon to St. Joseph's Hospital and to the Misericordia Hospital.

1922. DAVIS, WARREN B., M.D., 135 South Eighteenth Street. Oto-Rhino-Laryngologist, St. Agnes' Hospital; Instructor in Surgery at the Jefferson Medical College; Assistant Surgeon to the Frankford Hospital.

1890. DEAVER, JOHN BLAIR, M.D., Sc.D., LL.D., 1634 Walnut Street. Surgeon-in-Chief to the Lankenau Hospital; Emeritus Professor of Surgery, University of Pennsylvania.

1898. DEAVER, HARRY C., M.D., 337 South Eighteenth Street. Visiting Surgeon to the Episcopal Hospital; Surgeon-in-Chief to the Kensington Hospital for Women; Surgeon to the Children's Hospital of the Mary J. Drexel Home.

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.

1921. ELIASON, ELDRIDGE E., M.D., Sc.D., F.A.C.S., 326 South Nineteenth Street. Professor of Clinical Surgery at the University of Pennsylvania; Associate Professor of Surgery University of Pennsylvania Graduate School of Medicine; Surgeon to the University of Pennsylvania Hospital and the Howard Hospital.

1909. ELMER, WALTER G., B.S., M.D., 1801 Pine Street. Associate Professor of Orthopædics in the Graduate School of Medicine at the University of Pennsylvania; Clinical Professor of Orthopædic Surgery at the Woman's Medical College; Visiting Orthopædic Surgeon to the Philadelphia General Hospital, the Polyclinic Hospital, the Hospital of the Woman's Medical College of Pennsylvania, and the Jewish Hospital; Visiting Surgeon to the Pennsylvania Training School for Feeble-Minded Children at Elwyn.

1898. FRAZIER, CHARLES HARRISON, M.D., Sc.D., 3600 Walnut Street. John Rhea Barton Professor of Surgery in the University of Pennsylvania.

1899. GIBBON, JOHN H., M.D., 1608 Spruce Street. Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania Hospital; Consulting Surgeon to the Bryn Mawr Hospital.

1914. GILL, A. BRUCE, A. B., M.D., The Lenox, Thirteenth and Spruce Streets. 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 Training School for Crippled Children; Consulting Surgeon to St. Edmond's Home for Crippled Children.

1902. GIRVIN, JOHN H., M.D., 2120 Walnut Street. Gynæcologist to the Presbyterian Hospital; Associate Professor of Gynæcology, Graduate School of Medicine, University of Pennsylvania.

1925. GRANT, FRANCIS CLARK, M.D., 3600 Walnut Street. Associate in Surgery, School of Medicine, University of Pennsylvania; Associate in Neuro-Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon, Department of Neurological Surgery, Philadelphia General Hospital.

1913. HEARN, WILLIAM P., M.D., 2119 Spruce Street. Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the Jefferson Hospital.

1922. HERMAN, J. LEON, B.S., M.D., 1521 Pine Street. Associate Surgeon, 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, Graduate School of Medicine, University of Pennsylvania; Professor of Anatomy and Histology, Temple University, Dental School; Surgeon to the Memorial Hospital, Roxborough, Philadelphia, Pennsylvania.

1925. HINTON, DRURY, M.D., F.A.C.S., 4301 Spruce Street. Instructor in Surgery at the University of Pennsylvania; Instructor in First Aid, Summer School, University of Pennsylvania; Instructor in Physical Diagnosis, University of Pennsylvania; Assistant Surgeon to the Howard Hospital; Visiting Surgeon to the American Oncologic Hospital.

1905. HODGE, EDWARD B., A.B., M.D., 2019 Spruce Street. Surgeon to the Presbyterian and Germantown Hospitals; Associate Surgeon to the Pennsylvania Hospital and to the Widener Memorial School.

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., 1503 Medical Arts Building, Sixteenth and Walnut Streets. Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Plastic Surgeon to the Hospitals of the Graduate School of Medicine; Oral Surgeon to the Episcopal Hospital; Consulting Plastic Surgeon to the Children's Hospital; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D. C.

1922. JOHN, RUTHERFORD L., M.D., 248 South Twenty-first Street. Instructor in Surgery at the University of Pennsylvania Medical School, Associate Surgeon, Orthopædic Service, Episcopal Hospital; Assistant Surgeon, Philadelphia Orthopædic Hospital; Orthopædist, Chief of Orthopædic Dispensary, at St. Christopher's Hospital for Children.

1915. JONES, JOHN F. X., B.S., A.M., M.D., 103 South Twenty-first Street. Surgeon to St. Joseph's Hospital, Misericordia Hospital and to St. Agnes' Hospital.

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

1925. KEATING, PETER M., A.B., M.D., 1726 Pine Street.

1914. KEENE, FLOYD D., M.D., Medical Arts Building, Sixteenth and Walnut Streets. Assistant Professor of Gynæcology, University of Pennsylvania; Assistant Gynæcologist, University of Pennsylvania; Assistant Gynæcologist, University of Pennsylvania Hospital; Gynæcologist to Chestnut Hill Hospital and Bryn Mawr Hospital.

1910. KELLY, JAMES A., A.M., M.D., 1815 Spruce Street. Visiting Surgeon to St. Mary's Hospital, St. Joseph's Hospital, and Misericordia Hospital; Associate Professor in Surgery, Post Graduate School, University of Pennsylvania.

1913. KLOPP, EDWARD J., M.D., 1611 Spruce Street. Demonstrator of Surgery, Jefferson Medical College; Surgeon at the Pennsylvania Hospital; Assistant Surgeon, Jefferson Hospital.

1914. LAWS, GEORGE MALCOLM, B.S., M.D., 2033 Locust Street. Associate Gynæcologist, Presbyterian Hospital; Gynæcologist, American Hospital for Diseases of Stomach.

1910. LEE, WALTER ESTELL, M.D., F.A.C.S., 905 Pine Street. Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to Pennsylvania Hospital, Germantown Hospital, Bryn Mawr Hospital, and Children's Hospital; Consulting Surgeon to the Chester County Hospital.

1899. LOUX, HIRAM R., M.D., Medical Arts Building, Sixteenth and Walnut Streets. Professor of Genito-urinary Surgery, Jefferson Medical College; Attending Genito-urinary Surgeon, Jefferson Medical College Hospital; Surgeon to the Philadelphia General Hospital.

1900. MARTIN, EDWARD, A.M. M.D., Ph.D., LL.D., 135 South Eighteenth Street. Professor of Surgical Physiology, University of Pennsylvania.

1917. MENCKE, J. BERNHARD, A.B., M.D., 1816 Spruce Street. Assistant Surgeon to the Lankenau Hospital; Surgeon to the Northwestern General Hospital.

1915. MERRILL, WILLIAM JACKSON, A.B., M.D., 2009 Spruce Street. Orthopædic Surgeon to the Children's Hospital and to the Germantown Hospital.

1904. MITCHELL, CHARLES F., M.D., 2003 Pine Street. Surgeon to the Pennsylvania Hospital, Bryn Mawr Hospital and Germantown Hospital; Consulting Surgeon to the Chestnut Hill Hospital.

1919. MONTGOMERY, EDWARD EMMET, B.S., M.D., Hon. A.M., LL.D., F.A.C.S., 1426 Spruce Street. Gynæcologist to Jefferson Hospital and to St. Joseph's Hospital; Consulting Gynæcologist to the Jewish Hospital.

1906. MULLER, GEORGE P., M.D., B.S., 1930 Spruce Street. Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals.

1921. MURPHY, EUGENE C., M.D., F.A.C.S., 1841 South Broad Street. Surgeon to the 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., F.A.C.S., 1710 Locust Street. Associate Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Medical College Hospital; Surgeon to St. Joseph's Hospital; Chief Surgeon to Frankford Hospital; Surgeon to Girard College; Consulting Surgeon to Pottstown Hospital, Pottstown, Pennsylvania; Consulting Surgeon to Newcomb Hospital, Vineland, New Jersey.

1890. NEILSON, THOMAS R., A.M., M.D., 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., M.D., 1927 Spruce Street. Associate Professor of Gynæcology, Graduate School of Medicine, University of Pennsylvania; Assistant Gynæcologist and Obstetrician Methodist Hospital; Gynæcologist, Abington Hospital, Abington, Pennsylvania.

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, Philadelphia General Hospital; Attending Surgeon, Woman's College Hospital; Assistant Surgeon, Orthopædic Hospital; Chief Surgeon, Bureaus of Police and Fire of Philadelphia.

1912. PFEIFFER, DAMON B., A.B., M.D., 1822 Pine Street. Surgeon, Methodist Episcopal and Abington Hospitals; Associate Surgeon, Presbyterian Hospital; Assistant Surgeon, Lankenau Hospital; Assistant Professor of Surgery, Graduate School, University of Pennsylvania.

1919. PIPER, EDMUND B., B.S., M.D., F.A.C.S., 1936 Spruce Street. Professor of Clinical Obstetrics, University of Pennsylvania; Obstetrician and Gynæcologist to Philadelphia General Hospital; Obstetrician to the Philadelphia Lying-in Hospital; Obstetrician to Chestnut Hill Hospital; Associate Obstetrician to Presbyterian Hospital.

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

1924. RAVDIN, I. S., M.D., 1930 Spruce Street. Assistant Surgeon University Hospital; Associate in Surgery, School of Medicine, University of Pennsylvania.

1898. ROBINSON, J. WEIR, M.D., 326 South Sixteenth Street.

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

1913. RUGH, J. TORRANCE, A.B., M.D., Medical Arts Building, Sixteenth and Walnut Streets. Professor of Orthopædic Surgery, Jefferson Medical College; Clinical Professor of Orthopædic Surgery, Woman's Medical College of Pennsylvania; Orthopædic Surgeon to the Jefferson Hospital, to the Methodist Episcopal Hospital, to the Philadelphia General Hospital, to the Montgomery Hospital of Norristown, and to the North American Sanatorium of Atlantic City, New Jersey; Consulting Orthopædic Surgeon to the Philadelphia Lying-in Charity Hospital, to the West Philadelphia Hospital for Women, to the Pennsylvania State Institution for Epileptic and Feeble-Minded, Spring City, Pennsylvania, to the New Jersey State Institution for Feeble-Minded and Epileptics, Vineland, New Jersey, and to the Pottstown Hospital, Pottstown, Pennsylvania.

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

1903. SITER, E. HOLLINGSWORTH, M.D., 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.

1922. SHALLOW, THOMAS A., M.D., 2045 Walnut Street. Assistant Surgeon to Jefferson Medical College Hospital; Surgeon to the Philadelphia General Hospital.

1924. SMYTH, CALVIN M., JR., M.D., 257 South Twenty-first Street. Associate Surgeon to the Methodist Episcopal Hospital, and the Abington Memorial Hospital, Abington, Pennsylvania; Associate in Surgical Research, Graduate School of Medicine, University of Pennsylvania.

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

1898. SPELLISSY, JOSEPH M., A.M., M.D., 317 South Fifteenth Street. Orthopædic Surgeon to St. Joseph's Hospital and to St. Edmond's Home for Crippled Children.

1911. STELLWAGON, THOMAS C., JR., M.D., 220 South Sixteenth Street. Chief Clinical Assistant in the Out-patient Surgical Department of the Jefferson Medical College Hospital.

1919. SWARTLEY, WILLIAM BLAINE, M.D., 6002 Greene Street, Germantown. Surgeon to Germantown Hospital; Demonstrator of Anatomy Jefferson Medical College.

1908. SWEET, J. E., A.M., M.D., Sc.D., F.A.C.S., 301 St. Marks Square. Professor of Surgical Research, School of Medicine, University of Pennsylvania.

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

1911. THOMAS, B. A., M.D., 1900 Spruce Street. Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon to the Presbyterian Hospital.

1911. THOMAS, T. TURNER, M.D., 2009 Spruce Street. Associate Professor of Applied Anatomy and Associate in Surgery in the University of Pennsylvania.

1915. THOMAS, W. HERSEY, M.D., Medical Arts Building, Sixteenth and Walnut Streets. Professor of Genito-urinary Surgery in Temple University; Genito-urinary Surgeon to the Philadelphia General Hospital, the Samaritan Hospital, and the Garretson Hospital.

1923. WELLS, J. RALSTON, M.D., Medical Arts Building, Sixteenth and Walnut Streets. Associate Professor of Surgery, Woman's Medical College of Pennsylvania; Associate Surgeon of the Visiting Staff, Woman's College Hospital; Assistant Surgeon Bryn Mawr Hospital; Assistant Surgeon Radiological Surgery, Philadelphia General Hospital; Chief Anæsthetist, Philadelphia General Hospital; Assistant Surgeon, Philadelphia General Hospital; Instructor in Surgery, Graduate School, University of Pennsylvania; Assistant Instructor in Surgery, Undergraduate School, University of Pennsylvania.

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

1919. WILLARD, DEFOREST P., B.S., M.D., 1630 Spruce Street. Professor Orthopædics, Graduate School of Medicine, University of Pennsylvania; Orthopædic Surgeon, Polyclinic Hospital; Orthopædic Hospital, and Delaware Hospital and Shrine Hospital.

1898. WOOD, ALFRED CONARD, M.D., 2035 Walnut Street. Assistant Professor of Surgery at the University of Pennsylvania; Surgeon to the Howard Hospital, and Philadelphia General Hospital.

FELLOWS DECEASED SINCE LAST PUBLICATION

DIED

1922. George Gorgas Ross.

1924. James K. Young.

1924. Robert G. LeConte.

1924. Duncan Lee Despard.

1925. Richard H. Harte.

1925. Morris Booth Miller.

1925. John B. Roberts.

1925. Henry Wharton.

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.		
1881.	W. T. Briggs, Nashville, Tennessee	June	13, 1894.
1881.	Christopher Johnson, 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 B. McBurney, New York, N. Y.	November	7, 1913.
1898.	Nicholas Senn, Chicago, Illinois	January	2, 1908.
1898.	Theodore Prewitt, St. Louis, Mo.	October	17, 1904.
1898.	L. McLane Tiffany, Baltimore, Md.	October	23, 1916.
1898.	Nathaniel P. Dandridge, Cincinnati, Ohio.	1911 or 1912.	
1898.	Roswell Park, Buffalo, N. Y.	February	15, 1914.
1898.	Robert F. Weir, New York, N. Y.		
1898.	Frederick S. Dennis, New York, N. Y.		
1900.	W. H. A. Jacobson, London, England.		

ELECTED		DIED
1900.	THEODORE KOCHER, BERNE, Switzerland...	July 27, 1917.
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.	
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 WHARTON, Philadelphia, Pa.	December 1925.

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.

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. Clarke.
1921. J. Torrance Rugh.
1922. George P. Muller.
1923. W. Estell Lee.
1924. Robert Ivy.
1925. John Speese.

CONTENTS

TRANSACTIONS
OF THE
PHILADELPHIA ACADEMY OF SURGERY

Meeting of January 3, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. T. Turner Thomas. — Two cases of operation for intussusception in infancy with recovery.

Case Reports.

Dr. T. Turner Thomas. — 1. Return of inguinal hernia after Halstead's operation.
Discussed by Doctor Owen.
2. Two cases of excision of portion of humeral head for recurrent dislocation of the shoulder, one being bilateral.
Discussed by Doctors Gill, McKnight and Thomas.

Dr. George G. Ross and Dr. K. P. A. Taylor. (By invitation.) — Sliding hernia of the ureter.
Discussed by Doctor Ross.

Dr. E. J. Klopp and Dr. G. A. Ulrich. (By invitation.) — Amniotic hernia.

Dr. E. J. Klopp. — 1. Patent urachus with sarcoma developing in the wall.
2. Acute inflammation of Meckel's diverticulum.

Dr. Arthur E. Billings. — Pyonephrosis with late secondary hemorrhage.
Discussed by Doctor Ross.

Meeting of February 7, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. H. R. Owen.
1. Retro-œsophageal abscess following a foreign body in the œsophagus.
Discussed by Doctors Manges (by invitation) Jopson, Ashhurst and Owen.
2. Two cases of femoral hernia following operation for double inguinal hernia.

Dr. G. M. Dorrance.
Fracture of the jaw.
1. Interdental splint.
2. Intermaxillary wiring.
3. Intermaxillary cast splint.
Discussed by Doctors Ivy, Roberts, Dorrance and Hewson.

Dr. A. Hewson.
Showed a case of interdental splint.

Case Reports.

Dr. A. P. C. Ashhurst.
Showed a patient with a recurrent dislocation of mandible.
Loose cartilage in elbow-joint. Fracture of semilunar cartilage of knee-joint.
Discussed by Doctors T. T. Thomas, Rugh, Skillern, Jopson and Ashhurst.

Dr. H. P. Brown, Jr.
Showed a case of fracture of atlas and axas.
Discussed by Doctors Hewson and Rugh.

Dr. D. B. Pfeiffer.
An undeveloped kidney, presenting a chronic unilateral nephritis with unusual symptoms.
Discussed by Doctors Ross and Ashhurst.

Dr. E. T. Crossan and Dr. Don Lew. (By invitation.)
Acute intestinal obstruction from Meckel's diverticulum complicating case of acute appendicitis.
Discussed by Doctor Crossan.

Exhibition of Specimens.

Dr. A. P. C. Ashhurst and Dr. A. S. Walking. (By invitation.)
Fracture of vertebra.
Fibroma of ovary.
Ununited fracture of neck of femur.
Discussed by Doctors Owen, T. T. Thomas, Ashhurst, Jopson and Owen.

Paper.

Dr. E. C. Murphy and Dr. G. M. Dorrance.
Results of treatment of twenty cases of intracapsular fracture of the femur by the abduction cast method.
Discussed by Doctors Gill, Dorrance, Thomas, Ashhurst, Jopson and Owen.

Meeting of March 7, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Instruments.

Dr. H. R. Owen.
Truesdale's tourniquet.

Exhibition of Patients.

Dr. E. G. Alexander.
Supra-pubic sarcoma.
Discussed by Doctors Bromer and Roberts.

Case Reports.

Dr. John B. Roberts.
Contribution to subcutaneous direct fixation of transverse fractures of the patella.
Discussed by Doctor Rugh.

Dr. George P. Muller.
1. Epiphyseal separation of humerus.
2. Compression fracture of os calcis.
3. Fracture of upper end of tibia.
Discussed by Doctors Thomas, Jopson, Laws, Ashhurst and Rugh.

Annual Oration.

Dr. J. Torrance Rugh.
The Surgery of Infantile Paralysis.

Paper.

Dr. George P. Muller and Dr. K. P. Taylor. (By invitation.) — Tetanus ascendens. Discussed by Doctor Ashhurst.

Meeting of April 4, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. H. P. Brown, Jr.
1. Bullet wound of intestines.
2. Gunshot wound of knee.
Discussed by Doctors William B. Miller, Muller, and Baldwin.

Case Reports.

Dr. E. G. Alexander. — Four cases of fracture of metatarsal bones. Discussed by Doctors Gill, Ashhurst, and Alexander.

Dr. George P. Muller. — Splenectomy for splenic anemia. (In a child.) (In an adult.)

Dr. I. M. Boykin.
1. Rupture of pregnant uterus. Discussed by Doctor Ashhurst.
2. Amputation of both thighs for senile gangrene. Discussed by Doctor Ross.

Dr. C. H. Frazier and Dr. F. C. Grant. (By invitation.) — Auto transfusion. Discussed by Doctor Boykin.

Papers.

Dr. George P. Muller and Dr. I. S. Ravdin. (By invitation.) — Acute perforation of duodenal ulcer without ulcer symptoms. Discussed by Doctors Alexander, McKnight, Ross and Muller.

Dr. Adrian Voegelin. (By invitation.) — Amœbic abscess of liver. Discussed by Doctors Laws and Voegelin.

Meeting of May 2, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. J. S. Rodman.
1. Cranial defect.
2. Jacksonian epilepsy.
Discussed by Doctor Muller.

Case Reports.

Dr. John H. Girvin and Dr. John Speese. — Lymphangioma of omentum. Discussed by Doctor Outerbridge and Doctor Girvin.

Dr. H. A. McKnight.
1. Traumatic intra-articular separation of the pelvic bones.
2. Double longitudinal fractures of the lower end of the tibia.
Discussed by Doctors Ashhurst, Ross and McKnight.

Dr. Verne Burden. (By invitation.) — Case of gynecomastia. Discussed by Doctors Muller and Rodman.

Dr. Leo Reed. (By invitation.) — Case of mesenteric thrombosis. Discussed by Doctors Jopson, Despard and Ross.

Dr. W. F. Belk. (By invitation.)
1. Ostitis, fibrosa, cystica of the femur.
2. Osteo-chondroma of the fibula.

Dr. A. Hewson.
1. Rupture of the lungs without fracture.
2. Removal of bullet from the ventral aspect of the scapula.

Paper.

Dr. John Speese and Dr. H. P. Brown, Jr. — Carcinoma developing from benign goitre.

Exhibition of Dissecting Table.

Dr. A. Hewson.

Meeting of October 3, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Dr. A. P. C. Ashhurst continued the discussion of Dr. H. A. McKnight's case report at the meeting May 2, 1921, of "Double Longitudinal Fractures of the Lower End of the Tibia."

Papers.

Dr. J. E. Sweet and Dr. C. M. Smyth, Jr. Dr. F. L. Hartman. Dr. J. K. W. Wood. (By invitation.)	The results of high ligation of the cystic duct in cholecystectomy. Discussed by Drs. J. E. Sweet, M. M. Alter (by invitation), and A. M. Willis (by invitation).
Dr. A. Murat Willis, Richmond, Va. (By invitation.)	Some problems in gall-bladder surgery. Discussion opened by Dr. John Gibbon, and continued by Doctor B. B. Vincent Lyon (by invitation).
Dr. L. H. Landon and Dr. M. M. Alter. (By invitation.)	Primary carcinomatous papilloma of the renal pelvis. (Lantern Slides.) Discussed by Doctors Thomas, Landon, Farley and Alter (by invitation).

Meeting of November 7, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Cases.

Dr. Hubley R. Owen.	Cases illustrating methods of treating thyroid toxemia.
Dr. A. Bruce Gill.	End-results of operation for Dupuytren's contracture.
Dr. J. Stewart Rodman.	1. Cranial defect; post-operative result. 2. Extradural hemorrhage with paralysis of arm.
Dr. Robert H. Ivy and Dr. John H. Jopson.	Cicatricial ankylosis of the jaw relieved by plastic operation.

Case Reports.

Dr. John H. Jopson and Dr. John Speese.	Obstruction of common bile-duct with complete external biliary fistula. Relief by choledochogastrostomy.
Dr. P. G. Skillern, Jr.	Case of obstructing carcinoma of lower sigmoid in a woman aged seventy-two, operated on by cæcostomy and the two-stage Mikulicz method under local anæsthesia. Recurrence sixteen months later. (Lantern Slides.)

Papers.

Dr. Joseph A. Blake, New York. (By invitation.)	Drainage. Discussed by Drs. J. B. Deaver, G. E. Brewer, Walton Martin, John H. Gibbon and Joseph A. Blake.
Dr. John H. Gibbon and Dr. John B. Flick. (By invitation.)	The present status of epiplopexy.

Meeting of December 5, 1921, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. Roland Phillips. (By invitation.)	Suture of stab wound of heart with recovery. Discussed by Doctors Ashhurst, Mitchell and Owen.
Dr. A. P. C. Ashhurst, Dr. Ralph C. Bromer and Dr. C. Y. White. (By invitation.)	Lantern demonstration showing sarcoma and cysts of bones. Discussed by Doctor Jopson.
Dr. Charles F. Mitchell.	Abscess of omentum caused by foreign body.
Dr. W. Estell Lee.	Multiple fibromata of the ilium causing recurrent intussusception. Discussed by Doctor Allen.

Dr. W. Estell Lee and Dr. J. Ralston Wells. (By invitation.)	Primary sarcoma of the axillary glands.

Papers.

Lieutenant J. W. Ellis, U.S.N. (By invitation.)	The cause of death in high intestinal obstruction. Discussed by Doctor Sweet.

Meeting of January 9, 1922, in Cadwalader Hall, College of Physicians. President, Dr. George G. Ross, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Patients.

Dr. James K. Young.	1. Bone transplantation for tibial cyst. Discussed by Doctors Ashhurst and Young. 2. Disarticulation of the hip-joint for periosteal sarcoma.
Dr. P. G. Skillern, Jr.	Hypertrophic villous synovitis of knee-joint; arthrotomy by z-shaped incision. End result. Discussed by Doctors Owen, Young, Jopson and Skillern. Doctor Owen in discussing Doctor Skillern's case showed three cases of villous synovitis of the knee-joint, upon whom he had operated.
Note. Dr. H. R. Owen.	
Dr. G. M. Dorrance.	1. Case of tuberculosis of the elbow cured by X-ray treatment. Discussed by Doctor Dorrance. 2. Case of avulsion of the skin and subcutaneous tissue of the palm treated by abdominal pedicle graft. Discussed by Doctors Gill, Skillern and Dorrance.
Dr. Morris B. Miller.	Presented a case of "Septic Arthritis of the Knee-joint"—Final result.

Case Reports.

Dr. Henry P. Brown, Jr.	Inguinal hernia of the uterus, tubes and ovaries. Discussed by Doctor Jopson.
Dr. James H. Baldwin.	1. Case of gall stones. Discussed by Doctor Ross. 2. Case of foreign body in the rectum. Discussed by Doctor Billings.

Papers.

Dr. T. Turner Thomas.	Intraperitoneal rupture of the bladder. Discussed by Doctors Crossan and Ross. Discussed by Doctor Lipshutz. (By invitation.)

Demonstration of Instruments.

Dr. G. M. Dorrance.	Demonstration of skin coaptation buttons. Discussed by Doctor Ashhurst.

Meeting of February 6, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. J. K. Holloway. (By invitation.)	Spontaneous lateral ventral hernia.
Dr. E. J. Klopp and Dr. F. S. Borzell. (By invitation.)	Non-rotation of the colon. Discussed by Doctors Pfeiffer, Mitchell, Ashhurst and Klopp.
Dr. W. Estell Lee.	1. Multiple gunshot wounds of the ileum; resection of the bowel. Recovery. Discussed by Doctors Ross and Nassau. 2. Carcinoma of the tongue with metastasis of the cervical glands under treatment with radium and X-ray. Discussed by Doctors Owen and Lee.

Dr. H. R. Owen. — Caseous pyelonephritis. Discussed by Doctors Randall, Speese, Gill, Outerbridge and Owen.

Meeting of March 6, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. Joseph M. Spellissy. — Congenital absence of patellæ. Discussed by Doctor Ashhurst.

Dr. P. G. Skillern, Jr. — Actinomycosis. Discussed by Dr. R. H. Ivy.

Dr. Edward B. Hodge. — 1. Compound luxation of elbow with rupture of brachial vessels. Double phlebitis as a sequel. 2. Backward luxation of the foot on the leg, with fracture of the fibula. Discussed by Doctor Gibbon.

Papers.

Dr. Thomas A. Shallow. (By invitation.) — A modification of the Kader operation of Gastrostomy (with report of thirty cases). Discussed by Doctors Pfeiffer, Ashhurst, Jopson, Wm. Spencer and Shallow.

Dr. Benjamin Lipshutz. (By invitation.) — Malignancy of the undescended testis. Discussed by Doctors Nassau, Randall, Jopson, Gibbon and Lipshutz.

Meeting of April 3, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. DeForest P. Willard. — 1. Results of the Bennett operation for lengthening quadriceps extensor. 2. Fracture of the head of the humerus with injury to the brachial plexus.

Case Reports.

Dr. George P. Muller. — 1. Post-operative tetanus. 2. Carcinoma of rectum treated by palliation.

Exhibition of Instruments.

Dr. Robert H. Ivy. — Jaw dilator as adjunct in the treatment of mandibular ankylosis.

Papers.

Dr. George W. Crile. (By invitation.) — Relation between the brain and liver, with especial reference to the surgery of the liver. (Lantern Slides.) Discussed by Doctors Gibbon, Muller, Sweet and Crile.

Col. W. L. Keller, M.C., U.S.A. (By invitation.) — The surgical treatment of persistent thoracic cavities following empyema, where the usual operations have failed to produce obliteration. Discussed by Doctors LeConte, Butler and Colonel Keller. (Lantern Slides.)

Meeting of May 8, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. H. R. Owen. — 1. Fat transplant for painful stump following amputation of leg.

Dr. Charles Nassau. — Exophthalmic goitre.

Dr. Benjamin Lipshutz. (By invitation.) — Branchial fistula—operation.

Case Reports.

Dr. George P. Muller. — Two cases of gunshot wound of femoral artery. Discussed by Doctors Jopson, Nassau, Owen, Muller, Dorrance and Lee.

Papers.

Dr. E. G. Alexander.	D'Espine operation for prolapse rectum. Discussed by Doctors Roberts, Alexander, Pfeiffer and Nassau.
Dr. B. Franklin Buzby. (By invitation.)	Salivary calculus with report of two cases. Discussed by Doctors Ivy and Roberts.
Dr. J. Stewart Rodman.	Carcinoma of the breast—with especial reference to certain pre-cancerous lesions. Discussed by Doctors Nassau, Roberts, Jopson and Rodman.
Dr. Hans Zenssen, Holland.	Addressed the Academy on "The Length of Muscle Fibres."

Meeting of October 9, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. H. R. Owen.	Traumatic popliteal aneurism. Discussed by Doctor Ashhurst.

Case Reports.

Dr. H. D. McKnight.	1. Recurring perforations of the stomach and duodenum. 2. Stab wound of the kidney. Discussed by Doctor McKnight. Nephrectomy discussed by Doctors Despard and Jopson.
Dr. John F. X. Jones.	Removal of a retention cyst from the liver.

Papers.

Dr. Donald Guthrie, Sayre, Pa. (By invitation.)	Practical hospital psychology. Discussed by Doctors Doane, Mohler, McCarthy and Guthrie, all by invitation.

Meeting of November 6, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. John H. Jopson and Dr. Norman S. Rothschild. (By invitation.)	Chronic empyema—treated by the Keller method. Discussed by Doctor Jopson.
Dr. H. A. McKnight.	1. Unusual fracture of the skull. Discussed by Doctors Rodman and Jopson. 2. Multiple fractures of the pelvis.
Dr. William Jackson Merrill.	Stabilization of wrist-joint by bone graft in spastic paralysis. Discussed by Doctor Hodge.
Dr. John H. Jopson.	Supra-condyloid fracture of humerus. Discussed by Doctors Ashhurst and Jopson.

Case Reports.

Dr. John B. Wolfe, Wilkes Barre, Pa. (By invitation.)	Fractured dislocation of third and fourth lumbar vertebræ with very marked deformity and complete paraplegia. Recovery under conservative treatment. Discussed by Doctors Rodman and Ashhurst.
Dr. G. M. Laws and Dr. William Bates. (By invitation.)	Hæmolytic jaundice—treated by repeated blood transfusions. Discussed by Doctor O. H. P. Pepper (by invitation), and Doctors Dorrance, Despard and Laws.
Dr. Henry P. Brown, Jr.	Report of two hundred fifty cases of empyema treated at the Children's Hospital. Discussed by Doctors Jopson, Ashhurst, Hodge, Thomas, Rodman, Despard, Roberts and Brown.

Meeting of December 4, 1922, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Bruce Gill. — Cases illustrating heliotheraphy for tuberculosis of bones and joints. Discussed by Doctors Dorrance, Ivy, Jopson, Ashhurst and Gill.

Case Reports.

Dr. Robert B. Ivy. — A case of actinomycosis treated by parenchymatous injections of copper sulphate.

Dr. Damon B. Pfeiffer. — A case of perforated Meckel's diverticulum requiring resection of the intestine. Discussed by Doctors Jopson and Pfeiffer.

Dr. E. L. Eliason. — Sub-acute massive proctitis. Discussed by Doctors Sweet, Pfeiffer, Muller and Eliason.

Papers.

Dr. George P. Muller. — Annual Oration. Some surgical relations of the sympathetic nervous system.

Meeting of January 8, 1923, in Thompson Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Astley P. C. Ashhurst. — Result of operation for rupture of the extensor longus pollicis ("Drummer's Paralysis"). Discussed by Doctor Roberts.

Dr. G. M. Dorrance. — Two cases of plastic repair of the cheek.

Dr. J. Stewart Rodman. — Neuritis and perineuritis of the arm. Injection of alcohol into peripheral nerves. Discussed by Doctors Hodge, Taylor and Rodman.

Case Reports.

Dr. George L. Carrington. (By invitation.)
Dr. J. Howard Cloud. (By invitation.)
Dr. Walter Estell Lee. — A fatal anaphylactic reaction following blood transfusion in a case of primary anæmia. Discussed by Doctors Lee, Pfeiffer and Carrington.

Dr. J. Rallston Wells. (By invitation.)
Dr. Walter Estell Lee. — Perforation in utero of a gastric ulcer. Discussed by Doctors Rodman, Lee and Wells.

Dr. A. P. C. Ashhurst. — Reported by title a case of recurrent unilateral subluxation of the mandible.

Dr. J. Stewart Rodman. — Acute intestinal obstruction caused by Meckel's diverticulum. Discussed by Doctor Taylor.

Papers.

Dr. A. Gonzales. (By invitation.) — Pleural epilepsy. Discussed by Doctors Rodman and Owen and Gonzales.

Meeting of February 5, 1923, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. J. W. Bransfield. — Bullet wound of the pregnant uterus. Discussed by Doctor Jopson.

Dr. Alfred Hand. (By invitation.) — An œsophageal, pleural, cutaneous fistula.
Dr. J. Rallston Wells. (By invitation.) — Discussed by Doctor Ashhurst.

Dr. Damon B. Pfeiffer. — Gunshot wound of the thigh. Discussed by Doctor Jopson.

Papers.

Dr. Thomas A. Shallow. — Radical treatment of fractures of the skull.
Discussed by Doctors Roberts, Shallow, Nassau, Rodman and Bernard Neubauer (by invitation).

Dr. George M. Dorrance. — Immediate plastic operations in injuries involving tendons or joints.
Discussed by Doctors Willard, Ashhurst and Dorrance.

Meeting of March 5, 1923, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. James A. Kelly.
1. Intestinal obstruction due to impacted ascarides lumbricoides.
2. Incised wound of the neck, involving the right fourth and fifth cervical nerves.
3. Bullet wound of thigh, involving the femoral artery.
Discussed by Doctors Ashhurst and Owen.

Dr. H. R. Owen. — End-result of operation for aneurismal varix of popliteal artery.

Case Reports.

Dr. D. L. Despard.
1. Splenectomy for advanced splenic anemia.
2. Repair of the musculo-spiral nerve.
Discussed by Doctors Stetten, Ashhurst, Jopson, Brown and Despard.

Dr. DeWitt Stetten, New York City. (By invitation.) — Further observation on a modified inguinal hernio-plasty technique, with complete utilization of the aponeurosis of the external oblique.
Discussed by Doctors Owen, Ashhurst, Despard, Jopson and Stetten.

Meeting of April 2, 1923, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Specimens.

Dr. A. Hewson.
1. A Lane plate, healed and in place.
2. A healed mastoid.
3. A double vagina, double uterus, and double Douglas' pouch.
4. Outline of a combined hydrocele and spermatocele with a spermatolyth.
Discussed by Doctor Herman.

Exhibition of Patients.

Dr. Chevalier Jackson. — Thoraco-puncture for removal of penetrating foreign body from lung.
Discussed by Doctors Spear, Shallow, Owen and Jopson.

Dr. Robert H. Ivy.
1. Arthroplasty for mandibular ankylosis.
2. Rhinoplasty.
Discussed by Doctors Roberts, Gill, Dorrance, Elmer, Jopson, Rugh and Ivy.

Case Reports.

Dr. John Speese.
1. Forward dislocation of the astragalus.
2. Results of operation for spina bifida occulta.

Dr. E. L. Eliason. — Septic mesenteric emboli (postoperative).

Papers.

Dr. G. M. Dorrance. — The treatment of webbed fingers, congenital and acquired.
Discussed by Doctors Jopson, Gill, Rugh, Ivy and Dorrance.

Meeting of May 7, 1923, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Patients.

Dr. Duncan L. Despard. — Removal of foreign body from bronchus.
Discussed by Dr. J. B. Roberts.

Case Reports.

Dr. Alexander Randall.	1. A case of torsion of the testicle. 2. An unusual vesical calculus.
Dr. Leon Herman.	1. Calculus in persistent urachus. 2. Foreign body (bullet) in bladder. 3. Septum in bladder with calculus in posterior compartment. Discussed by Doctor Mitchell.
Dr. E. G. Alexander.	1. Report of an unusual case of splenomegaly. Discussed by Dr. George P. Muller.

Papers.

Dr. J. Stewart Rodman and Dr. B. B. Neubauer. (By invitation.)	The management of cranial injuries. Discussed by Doctors Frazier, Roberts, Francis Grant, Muller and Rodman.
Dr. Walter M. Boothby, Rochester, Minn. (By invitation.)	The diagnosis of thyroid disease. Discussed by Doctors Frazier, Muller and Boothby.

Meeting of October 1, 1923, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Specimen.

Dr. Edward J. Klopp.	Carcinoma of the sigmoid with perforation of the cæcum.

Exhibition of Patients.

Dr. Astley P. C. Ashhurst.	1. Result of operation for cervical rib. 2. Callus from spontaneous (?) fracture of clavicle, mistaken for tumor. 3. Cervical adenitis mistaken for sarcoma. Discussed by Doctor Nassau. 4. Keloids of both ears. Discussed by Doctor Bromer (by invitation) and Doctor Dorrance. 5. Brachial plexus anæsthesia for amputation of forearm in a patient with advanced phthisis. Discussed by Doctor Nassau.

Dr. E. L. Eliason.	Rapid regeneration following facial nerve suture. Discussed by Doctor Frazier.

Case Reports.

Dr. T. Turner Thomas.	1. Hydronephrosis from kink in ureter. Accidental detachment of ureter during operation and anastomosis of ureter into dilated kidney pelvis. 2. Chorioepithelioma with exhibition of specimen. Discussed by Doctor Ashhurst.

Papers.

Dr. Charles H. Frazier.	Operative relief for paralysis of the recurrent laryngeal nerve. Preliminary note.

Meeting of November 5, 1923, Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Hubley R. Owen.	1. Dislocation of ulnar nerve (six cases). Discussed by Doctors Jopson and Brown. 2. Treatment of acute traumatic synovitis (hæmarthrosis and hydarthrosis) by repeated aspiration and immediate active mobilization without splinting (eleven cases).

Case Reports.

Dr. J. W. Bransfield.	Suppurative pericarditis. Discussed by Doctors Roberts, Elmer, Jopson and Despard.
Dr. Damon B. Pfeiffer.	Intra-abdominal hemorrhage due to ruptured corpus luteum follicle. Discussed by Doctors Muller and Speese.

Papers.

Dr. George P. Muller.	Pseudo-myxoma peritonei.
Dr. Henry P. Brown, Jr. Dr. Henry A. Strecker. (By invitation.)	Some observation on the treatment of fractures of the skull. (Report of one hundred cases from the Pennsylvania Hospital.) Discussed by Doctor Rodman.

Meeting of December 3, 1923, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. E. G. Alexander.	1. Fracture of lower end of humerus (two cases). Discussed by Doctor Ashhurst. 2. Gunshot wound of abdomen. 3. Incomplete rupture of ileum. 4. End-result. Sympathectomy for Buerger's disease. Discussed by Doctors Brown, Muller, Billings and Dorrance.

Case Reports.

Dr. George M. Dorrance.	Temporo-mandibular arthroplasty.

Papers.

Dr. W. Estell Lee. Annual Oration.	Post-operative pulmonary complications.

Meeting of January 7, 1924, in Cadwalader Hall, College of Physicians. President, Dr. John H. Jopson, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. E. R. Murphy. (By invitation.) Dr. E. B. Hodge.	Lung abscess and pyopneumothorax following tonsillectomy. Discussed by Doctors George Fetterolf, and Herbert Fox (by invitation), and Doctor J. B. Roberts.
Dr. Sharnoff. (By invitation.)	A case of intestinal obstruction (hernia through foramen of Winslow). Discussed by Doctor Deaver.

Dr. John B. Deaver.	1. Fracture-dislocation upper end of humerus (two cases). Discussed by Doctor Pfeiffer. 2. An interesting abdominal case. Discussed by Doctors Muller and Deaver.
Captain Spear. (By invitation.)	A case of acromegaly.

Case Reports.

Dr. George P. Muller.	Two cases of periarterial sympathectomy. (a) Scleroderma. (b) Atrophic ulcer.

Papers.

Dr. Benjamin Lipshutz. (By invitation.)	A contribution to the study of congenital fistulæ and cysts of the neck. Discussed by Doctors Ashhurst, Jopson and Lipshutz.

Meeting of February 4, 1924, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. G. M. Dorrance.	Multilocular cyst of jaw.
Dr. A. P. C. Ashhurst.	Three cases showing end-result of operation for ankylosis of elbow, and two cases for ankylosis of hip-joint.

Case Reports.

Dr. J. Torrance Rugh.	1. A case of Kuemmel's disease.
Dr. J. Torrance Rugh and Dr. George Wagoner. (By invitation.)	2. A case of fragilitas ossium. Discussed by Doctors Baer and Rugh.
Dr. A. Bruce Gill.	Case reports on reconstruction of the hip-joint, with lantern slides.

Papers.

Dr. Wm. S. Baer, Johns Hopkins University, Baltimore. (By invitation.)	Production of motion in ankylosed joints. Discussed by Doctors A. P. C. Ashhurst, DeForest Willard, Robert Ivy and Doctor Baer.

Annual Conjoint Meeting with the New York Surgical Society, March 12, 1924, at the University Hospital. Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Dr. Thomas C. Stellwagon.	Two cases of carcinoma of the kidney, and one case of bilateral varicocele. Discussed by Dr. C. L. Gibson.
Dr. John H. Gibbon.	A case of hypernephroma in a child of three years. Discussed by Dr. C. L. Gibson.
Dr. George P. Muller.	Cases of lung abscess. Discussed by Dr. Walton Martin.
Dr. Chevalier Jackson.	Chalk talk on bronchoscopy in diseases of the lungs. Discussed by Dr. Willy Meyer.
Dr. Gabriel Tucker.	Moving picture demonstration of bronchoscopic treatment. Discussed by Doctors Willy Meyer, Lillienthal and Auchincloss.
Dr. Edwin Sweet.	Parietal sacculi of the bile-ducts. Discussed by Dr. A. O. Whipple.
Dr. John G. Clark.	The present status of radium in pelvic carcinoma. Discussed by Dr. Burton J. Lee.
Dr. John H. Jopson.	A case of fracture-dislocation of the radius and ulna. A case of separation of the lower epiphysis of the femur and fracture of both bones of the leg. Discussed by Drs. H. H. M. Lyle. Morris K. Smith, John Gurster and John Gibbon.

Dr. Astley P. C. Ashhurst.	Excision of a brachial fistula. Exenteration of the nasal passages and sinuses for adenocarcinoma. Discussed by Drs. Ellsworth Eliot, Jr., John Douglas, Lillienthal and Ivy.
Dr. John Speese.	Insulin as an adjunct to surgery. Discussed by Doctors Whipple, LeConte, and Lilienthal.
Dr. W. Estell Lee.	Jejunostomy in intestinal obstruction. Discussed by Drs. Seward Erdman and Frederick T. Van Beuren.
Dr. John B. Deaver.	Remarks on fecal fistula. Discussed by Drs. Walton Martin, Gibson, and Deaver

Meeting of April 7, 1924, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Thomas A. Shallow.	1. Pulsating exophthalmus following fracture of base of skull. 2. Arteriovenous aneurism of internal jugular vein and vertebral artery following stab wound. Discussed by Doctor Ashhurst.
Dr. E. L. Eliason.	1. Fracture of tibia and astragalus with dislocation of the latter. 2. Bilateral fracture of the astragalus, tibia and fibula.

Case Reports.

Dr. K. Kornblum. (By invitation.)	Echinococcus cyst of the omentum.

Papers.

Dr. Robert G. LeConte. Dr. Walter Estell Lee. Dr. McKean Downs. (By invitation.)	Chronic partial intestinal obstruction, caused by a congenital malformation of the colon. Discussed by Doctor Sweet.

Dr. Temple Fay. (By invitation.)	The diagnostic value and interpretation of cerebro-spinal determinations: With moving picture demonstration.

Meeting of May 5, 1924, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Robert H. Ivy.	Two cases of plastic surgery of the face. Discussed by Doctor Dorrance.

Case Reports.

Dr. Walter Estell Lee. Dr. T. McKean Downs. (By invitation.)	Resuscitation of an arrested heart by cardiac massage. Discussed by Doctors Brown and Lee.
Dr. Drury Hinton. (By invitation.)	1. Compound fracture-dislocation at the wrist. 2. Compound fracture-dislocation at the elbow. Discussed by Doctor Jopson.
Dr. Thomas J. Ryan. (By invitation.)	Pneumothorax following traumatic injury to the lung. Illustrated.

Papers.

Dr. Thomas A. Shallow. Dr. Louis H. Clerf. (By invitation.)	Surgical treatment of bronchiectatic cavities. Discussed by Doctors Clerf and Hodge.
Dr. James H. Baldwin. Dr. W. R. Gilmour. (By invitation.)	A study of one hundred and thirty cases of gall-bladder surgery. Discussed by Doctor Baldwin.
Dr. Damon B. Pfeiffer. Dr. Calvin M. Smyth, Jr. (By invitation.)	Late results of splenectomy for traumatic rupture of the spleen. Discussed by Doctors Pfeiffer, Sweet, Hodge and Brown.

Dr. Wilbur H. Haines. (By invitation.)	Caudal anæsthesia in urology; a method for locating the sacral hiatus. Illustrated. Discussed by Doctors Thomas J. Ryan, Wells, Pfeiffer, Mumey and Haines.

Dr. W. Vickers of Australia was introduced to the Academy. Dr. Vickers gave a very interesting talk on Sympathectomy for Spastic Paraplegia, Parkinson's Disease, and Paralysis Agitans.

Meeting of October 6, 1924, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Hubley R. Owen.	Compound fracture of the olecranon associated with Colles' fracture of the same arm.

Case Reports.

Dr. Alexander Randall.	Two unusual cases of urinary calculus. Discussed by Doctor Ashhurst.
Dr. Hubley R. Owen.	A severe electric burn complicated by tetanus. Discussed by Doctor Dorrance.

Paper.

Dr. Benjamin Lipshutz. (By invitation.)	"Perirenal hydronephrosis; with comments on the technique of abdominal extraperitoneal nephrectomy." Discussed by Doctors Randall, Herman and Lipshutz.

Meeting of November 3, 1924, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. E. L. Eliason.	Spontaneous rupture of gangrenous urinary bladder.

Papers.

Dr. A. P. C. Ashhurst.	Removal of screws and plates after insertion in bone. Discussed by Doctor Jopson.
Dr. Francis C. Grant. (By invitation.)	The relief of pain in carcinoma of the face. Discussed by Doctor Ashhurst.
Dr. I. S. Ravdin.	The treatment of superficial burns. Discussed by Doctors Ashhurst, Baldwin, Dorrance, Owen, T. T. Thomas and Ravdin.

Meeting of December 1, 1924, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Case Reports.

Dr. E. J. Klopp.	Two cases of exophthalmic goitre with large thymus. Discussed by Doctor Sweet.
Dr. James H. Baldwin.	Case of ruptured spleen. Discussed by Doctor Smyth.

Papers.

Dr. Henry P. Brown, Jr.	Acute intussusception in children. Discussed by Doctors Bleckensmith (by invitation), Lee and Sweet.

Annual Oration.

Dr. Robert H. Ivy.	Swellings of the submaxillary region.

Meeting of January 5, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. H. R. Owen.	Rupture of biceps muscle, result of direct violence.

Case Reports.

Dr. B. Franklin Buzby.	A case of bilateral congenital dislocation of the patellæ. Discussed by Doctor Owen.

Papers.

Dr. John B. Deaver.	Cholecystectomy; external and internal cholecystostomy. Discussed by Doctors Sweet, Deaver, Jopson, Nassau, Ashhurst, Hodge and Deaver.
Dr. Thomas A. Shallow.	Mesenteric cysts. Discussed by Dr. H. C. Deaver.
Dr. Ralph Goldsmith. (By invitation.)	Kienbach's disease of the semilunar bone with report of three cases. Lantern Slides. Discussed by Doctor Jopson.

Meeting of February 2, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Francis C. Grant. (By invitation.)	Endothelioma of brain, with involvement of skull. Discussed by Doctors Ashhurst and Grant.
Dr. A P. C. Ashhurst.	A case of adeno-carcinoma of the nasal passages, with invasion of cervical lymph-nodes; radical extirpation; röntgentheraphy; plastic operations; no recurrence twenty-seven months later.

Case Reports.

Dr. A. E. Billings.	Report of a case of aberrant malignant thyroid.
Dr. Irvin E. M. Boykin.	Plastic cases and skin grafting. Discussed by Doctors Ivy, Muller, Ashhurst and Boykin.
Dr. A. P. C. Ashhurst.	A case of carcinoma in an aberrant (lingual) thyroid. Discussed by Doctors Muller and Billings.

Papers.

Dr. E. L. Eliason.	Surgical emergencies of the abdomen.
Dr. I. S. Ravdin.	The surgical service at the University Hospital. Discussed by Doctor Frazier.

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 Wednesday, February 25, 1925, at 2.30 P.M.

SCIENTIFIC PROGRAM

Dr. Charles A. Elsberg and Dr. Alfred Taylor.	Symptoms and late results in neoplasms of the spinal cord. Discussed by Drs. Charles Frazier, and J. Stewart Rodman.
Dr. Joseph A. Blake. Dr. George B. Brewer. Dr. Charles H. Peck.	Methods and late results of resection of the rectum for carcinoma. Discussed by Drs. John H. Gibbon, Damon B. Pfeiffer and Daniel F. Jones of Boston. (By invitation.)
Dr. Carl Eggers. Dr. Franz Torek.	Late results of resection of the œsophagus for carcinoma.

Meeting of March 2, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Hubley R. Owen.	Fracture of fibula. Dislocation through epiphysis of tibia. Discussed by Doctor Ashhurst.

Case Reports.

Dr. George P. Muller.	Bone graft of the tibia following removal of a benign cyst. Discussed by Doctors Willard, Ashhurst and Muller.
Dr. I. S. Ravdin.	Bone-graft of the tibia following removal of a giant cell tumor. Discussed by Doctor Muller.
Dr. E. J. Klopp.	Aneurism of renal artery, diagnosed calculus.

Papers.

Dr. Leon Herman and Dr. Jay Carp. (By invitation.)	Prostatic abscess—an analysis of nineteen cases treated by extra-urethral perineal prostatotomy. Discussed by Doctors Randall and Pfeiffer.
Dr. J. Bernhard Mencke.	Post-operative complications. Discussed by Doctor Owen.

Meeting of April 6, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. John H. Jopson and Dr. Norman Rothschild. (By invitation.) Dr. R. F. Farrell. (By invitation.)	1. Gunshot wound of heart or aorta. migration of bullet by way of arterial circulation to superficial femoral artery. Discussed by Doctor Brown. 2. Acute dilatation of the stomach and tetanoid convulsions following operation for large inguinal hernia. Discussed by Doctors Ashhurst and Jopson. 3. Open operation in an adult for irreducible fracture-dislocation of the hip. Discussed by Doctors Willard, Ashhurst, Farrell and Jopson. 4. Fecal fistula recurring after many operations. Treated by ileocæcal resection and ileocolostomy.
Dr. G. M. Dorrance.	Second stage of rhinoplasty by the peak roof method. Discussed by Doctor Ivy.

Case Reports.

Dr. E. L. Eliason.	Case of pylephlebitis and liver abscess following acute appendicitis with recovery. Discussed by Doctors Ashhurst, Nassau and Eliason.
Dr. Albert E. Bothe. (By invitation.)	Hypernephroma of the forearm. Discussed by Doctor Herman.

Papers.

Dr. G. M. Dorrance. — Cleft palate with a method of lengthening the soft palate. Discussed by Dr. W. B. Davis.

Dr. Norman S. Rothschild. (By invitation.) — Diverticula of the jejunum.

Meeting of May 18, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Presentation of Specimen.

Dr. Damon B. Pfeiffer. — Carcinoma of the recto-sigmoid. Discussed by Doctor Rodman.

Exhibition of Patients.

Dr. Damon B. Pfeiffer. — Repair of hernia by living sutures. Discussed by Doctors Jopson, Gill, Ivy and Pfeiffer.

Dr. Robert H. Ivy. — Total rhinoplasty by forehead flap.

Dr. Jessie W. Pryor. (By invitation.) — Case of thyro-glossal cyst. Discussed by Doctors Rodman and Owen.

Case Reports.

Dr. J. Rallston Wells. — Melena. Discussed by Doctors Smyth, Pfeiffer and Wells.

Dr. J. Stewart Rodman. — Depressed fracture of skull involving speech centre.

Meeting of October 5, 1926, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. Astley P. C. Ashhurst.
1. End-result fourteen years after astragalectomy for fracture-dislocation.
2. End-result of excision of the ankle, by the late Dr. John Ashhurst, Jr., in 1887.
3. Case of divergent dislocation of the metatarsus.
4. Two cases of arthrodesis for flail elbow.

Discussed by Doctors Buzby, Rugh, Elmer and Ashhurst.

Dr. Edward T. Crossan. — Two cases of dislocation of the astragalus in the same patient, one antero-lateral, the other upward (Nelaton's dislocation) discussed by Doctors Ashhurst and Herman.

Dr. E. L. Eliason.
1. Echinococcus cyst of the liver complicated by gall-stones.
2. Sarcoma of the stomach.

Discussed by Doctor Pfeiffer.

Presentation of Specimen.

Dr. John Speese. — Rupture of the aorta with hemopericardium.

Case Reports.

Dr. Edward T. Crossan. — Four cases of hypernephroma. Discussed by Doctors Ashhurst and Herman.

Dr. John Speese. — The occurrence of a fecal fistula in a large ventral hernia.

Dr. DeForest P. Willard.
1. Multiple enchondromata localized in the hand.
2. End-result following malposition of transverse supracondylar fracture of the humerus in a child.

Meeting of November 2, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Instruments.

Dr. B. A. Thomas.
1. Improved suprapubic drainage cup apparatus.
2. New prostatectomy lobe forceps.
3. Prostatic punch.

Discussed by Doctor Stellwagon.

Exhibition of Patients.

Dr. Thomas C. Stellwagon. — Two cases of complete extirpation of penis. Discussed by Drs. B. A. Thomas, Wells and Stellwagon.

Case Reports.

Dr. Henry P. Brown, Jr. — 1. Partial hydronephrosis caused by pressure from double ureter. 2. Two cases of echinococcus cyst of liver. Discussed by Doctors Hodge, Randall, Laws, Ashhurst, Herman, Stellwagon, Ivy and Brown.

Dr. George M. Laws. — Solitary cyst of the kidney.

Dr. Alexander Randall. — 1. Abscess of the urachus. 2. Tumor of the urachus.

Paper.

Dr. Leon Herman. — Practical aspects of pyelography. Discussed by Doctor Laws.

Meeting of December 7, 1925, in Cadwalader Hall, College of Physicians. President, Dr. Edward B. Hodge, in the Chair.

SCIENTIFIC PROGRAM

Exhibition of Patients.

Dr. A. P. C. Ashhurst. — Three cases of bone tumors.

Dr. John B. Carnett. — One case of bone tumor.

Dr. Damon B. Pfeiffer. — Two cases of osteitis fibrosa.

Paper.

Dr. Ernest Amory Codman, Boston, Mass. (By invitation.) — The registry of bone sarcoma as an illustration of the end-result idea in hospital organization.

Discussions.

Drs. A. P. C. Ashhurst, John B. Carnett, Robert H. Ivy, George P. Muller, Henry K. Pancoast and Damon B. Pfeiffer. The discussions consisted of the presentation of patients with bone sarcoma.

ANNUAL ORATION, MARCH 7, 1921

THE SURGERY OF INFANTILE PARALYSIS

By J. Torrance Rugh, M.D.
of Philadelphia, Pa.

It is an indisputable fact that, with the exception of the modern ill-fitting shoes, no other factor causes so large and so varied a number of deformities as infantile paralysis. This has not always been so, but with the frequently recurring epidemics in the past twenty years, the number of cases which come under the care of the orthopædic surgeon has increased tremendously. It is only when one considers that, in addition to the vast number which have been left with some form of paralysis, an equal or even greater number have recovered without any stigma of the disease, that one fully realizes the ravages and severity of this condition.

The surgical problems of the various stages of infantile paralysis have always proved of the greatest interest to the orthopædic and general surgeons. However, the very great increase in the number of cases following the widespread epidemic of 1916 has not only called forth tremendous surgical efforts from the orthopædic surgeons during the past two years, but has also proved an active stimulus to the creative genius of the surgeon, along mechanical lines, to more effectively deal with the problems that are constantly being presented. The time is not long since the surgery of flaccid paralysis was limited to a very small number of procedures. The treatment of this condition consisted entirely in the application of some form of brace, of massage, manipulation and more frequently of incantations until the introduction of subcutaneous tenotomy by Stromeyer, of Hanover, Germany, in 1831. This simple operation was the first great surgical procedure for the easy correction of many of the malformations associated with paralysis and formed an epoch in the surgery of deformities. It was introduced into America in 1837 by Dr. Wm. Detmold, and its use rapidly became general. The *Association Medical Journal,* London, June, 1855, says, "Never was any great operation involving a vital principal of surgery, spread in less time over the globe and with less resistance than tenotomy." No further advances were made in the surgery of this condition until in 1878, when arthrodesis for the stabilization of certain joints was performed and advocated by Albert of Vienna. The next epochal advance in the surgery of these deformities was brought out by Nicoladoni, who, in 1881, first transplanted one of the peroneal tendons into the tendo Achillis in a case of calcaneus, and this was the beginning of the development of that branch of work which has held the attention of orthopædic surgeons to the present day. In these earlier cases transplantation was

always performed by inserting tendon into tendon, and the results of the work in a large percentage of the cases proved disappointing because of the failure of the attachment to become permanent.

The next forward step was the work of Glück, who in 1892 first used silkworm gut sutures to bridge the gap in tendons. These were promptly substituted by strands of silk, and in 1896 Kümmel, in the examination of a case several years after operation, "found that the strands of silk had become incorporated in and had changed into fibrous tissues." Willard in 1891, in a paper before the American Orthopædic Association, reported the use of tenotomies and resection of joints for the correction of paralytic deformities. In 1897, Goldthwait first reported the direct transplantation of muscles in the treatment of paralytic deformities, and about the same time Fritz Lange employed long silk cords for the transmission of power from a live muscle to a paralyzed part. Thus it will be seen that up to the beginning of the present century the correction of paralytic deformities by modern surgical methods was extremely elementary, and yet the field for the work was full of promise and the prospects afforded a tremendous stimulus to the creative efforts of the mechanical surgeon.

The surgery of paralysis of this era has now become obsolete. The insertion of tendon into tendon has been found entirely insufficient because of the stretching of the parts. The use of silk ligaments for the stabilization of a joint or the maintenance of a part in a given position or for the preservation of the continuity of a tendon for the transmission of muscle power, which at first offered such promising results and appeared to be the method which would afford the greatest relief and benefit in the treatment of these conditions, proved totally unreliable after trial. A silk insert will remain in place almost indefinitely and will act as a check to support a certain strain, but under continued tension and use it will became separated from the surrounding scar tissue and will gradually be extruded by a process of non-infectious suppuration. It has not been my experience that a foreign substance such as silk will become a part of the tissue which surrounds it, even after a period of years, and almost all orthopædic surgeons have found that active use or strain upon such an insert will be followed by the loss of the insert through a process as above mentioned.

The active surgery of infantile paralysis is usually classed under the headings of corrective and reconstructive. The first includes those operations done during the stage of recovery from the attack of the disease and before the resultant or permanent paralysis has become established. The second includes the operative work after all possible recovery has obtained and the need is present for the building up and restoration of as much essential function as possible. I have always felt that there should be three divisions for this, the first to include corrective measures for those cases of deformity which occur during the first one or two years. In this period, tendon transference, tenodesis, arthrodesis, and such types of operation should not be done, as regeneration of nerve cells and partially paralyzed muscles has not yet been completed, and they may greatly interfere with the natural processes of recovery. The most useful procedure at this time is tenotomy, either partial or complete, and it is always to be supplemented with efficient mechanical restraint or support, developmental and corrective gymnastics, physio-therapy and training in the proper use of parts. Occasionally an osteotomy may be indicated, as in those cases with weakness or loss of the internal hamstrings and active function in the external which results in a knock-knee with a tendency to an external rotation or partial luxation of the tibia at the knee as well as a strain upon the foot and ankle structures which may result in deformity. It is almost impossible to hold such a leg by a brace, and yet it is essential to the proper functions of the foot that the leg be maintained straight. No destructive operation should be considered at this time. (By destructive operation is meant one which sacrifices certain functions in order that the most essential function of the part shall be conserved, such as arthrodesis, tenodesis, resection of bone, etc.) Thus it will be seen that during the first two years of the disease, surgery must be of the most elementary type, and much greater reliance must be placed upon mechanical and restorative means of treatment, such as physio-therapy, hydrotherapy, etc. The tendency on the part of many operators is to do too much and too radical surgery in the earlier years of the disease, and it not infrequently happens that they thus destroy the chances of recovery in certain parts that would be of the greatest benefit to the patient. Prevention of deformity and correction by the simplest procedures are the keynote of the surgery during the stage of active regeneration and recovery from the attack.

After this period has elapsed and further regeneration has begun to be more slow and retarded, we may consider the second type of surgery, viz., constructive. The aim of this is to preserve as many of the functions in the part as possible rather than the establishment of one or two essentials. The total loss of power in or the weakness of one muscle or of a group allows the onset of unbalanced and faulty positions which, if permitted to persist, will result in deformity. If, now, a complete loss of power and degeneration of the muscles can be demonstrated, surgery is to be considered "to utilize what is ill-directed voluntary movement and to restore the balance of power so far as possible in the affected part" (Jones and Tubby).

There are certain principles concerned in the adaptation of surgical methods in these cases. In the first place, there is the mechanical principle of the condition itself. This must be carefully studied both as to the function of the part and as to the proper and best means of preserving it. In many instances, mechanical measures will suffice to maintain proper function, and in those cases in which there is a certain amount of power remaining, mechanical correction will relieve these parts of strain and will permit of a fairly rapid return of power and function in them. When, however, the balance is dis-

turbed by overaction on the part of certain muscles or groups of muscles, surgical measures must be used to again restore it.

In the next place, the mechanics and the mechanical relations of related parts must be considered because of their effect upon the function of the part in question. This is well illustrated in a case where the foot is in a position of adduction and the knee in valgum. The one tends very greatly to exaggerate the other and neither one can be corrected and maintained without the complete correction of the other.

In the third place, one must consider the mechanics of the surgical procedure to be employed in the correction of a given case. These must be as simple as possible and must be accurately adapted to the needs of the individual case. It is well recognized that in many of these conditions there are two types of operative procedures in the muscle and tendon structures, viz., those which concern structures having analogous functions and those having opposing functions. It is also conceded that those of the first group more readily adapt themselves both surgically and functionally to the betterment of the condition than those of the second group. When tendon transplantation first began to be generally considered in these cases, the question arose as to whether there would be difficulty in reëducating the part or the muscle in its changed position. This is still a debatable proposition in that it concerns direct and indirect cerebration. There is no doubt that in the great majority of individuals, the impulse to actuate a certain movement is inseparably connected with the movement of the part and not with the action of the muscle which moves the part. The impulse, therefore, expresses itself to the mind in terms of motion of the part and not in the contraction of the muscle itself. This is so because in most persons movement is automatic and is not concerned with the direct function of a muscle or group of muscles. One fully realizes this in the attempts at muscle training for the purpose of corrective work in certain deformities. The very small proportion of individuals who possess voluntary control over muscle action is startling. Great patience and labor are frequently necessary to the proper development of muscle intelligence on the part of patients and some do not possess the ability to develop it at all. The attempt, therefore, to convert a flexor into an extensor or vice versa, or an adductor into an abductor or the contrary, will meet with failure in a large percentage of cases because of the lack of muscle intelligence or muscle sense in so many patients. Again, there must be an actual reversal of the thought image in the mind of the patient and the impulse must be transformed into its opposite until it can become automatic with the individual. Fortunately, however, for the purpose of the work in many cases of infantile paralysis, this becomes extremely easy and the reversal of the impulse of action entirely unnecessary, in fact impossible, because the opposing impulse cannot be delivered on account of the loss of its motor centres, and all that is necessary is that the same automatic contraction of the active muscles shall continue in order that the part may be moved in the proper direction.

By far the best results in the transplantation of live tendons are obtained where a tendon having a similar function to the paralyzed one can be substituted for it. However, there are certain factors which must be taken into consideration in these procedures. In the first place, it is useless to transplant a weak tendon where a strong one is needed. In the earlier days of this work, probably more failures resulted from disregard of this principle than from any other factor. For instance, the posterior tibial tendon or the peroneal tendons cannot possibly replace the tendo Achillis and the tibialis anticus cannot possibly perform all the functions of the dorsal flexors of the foot and toes. But in either case they may aid tremendously in the reëstablishment of the preservation of functions when additional measures for support and stabilization are employed. It has been very succinctly stated clinically that a pale muscle should not be transplanted at all, a pink one may be used but a red one is best (Soutter). Again, the function of the transplanted tendon should approximate as nearly as possible that of the paralyzed one and its pull must be made in the same direction. For instance, where an anterior tibial is used to aid in dorsi-flexion of the foot, it must be attached as far down on the dorsum of the foot as possible so that it may have and may exercise a greater leverage upon the part as nearly equal to the common extensors as possible. This likewise holds true in all other cases of substitution and is an extremely important principle of operation. Again, it is now quite well established that if the sheath of the tendon can be transplanted with the tendon or, failing in this, if the transplanted tendon can be conducted through the sheath of the tendon whose function it is intended to replace, there will be much less impairment in the excursion of the tendon and hence better function in the part to be actuated. Again, the attachment of a tendon should be made either directly to or into a bone or subperiosteally. There has been a large percentage of failures from the attempted union of tendon with tendon because of the very slow repair in this tissue and because of the type of union which occurs. The other method of attachment gives much greater assurance of success and should be utilized wherever possible. Union between the end of the tendon and the bone or periosteum can be made much more certain if, on the removal of the tendon from its original attachment, a small layer of bone is included and this is placed in contact with bone or periosteum. By this means bony union is obtained instead of the fibrous union which occurs otherwise. The operation of tendon transplantation, both direct and indirect, has proven a very great advance in the surgery of infantile paralysis, but it is gradually reaching its level of efficiency and success, and it is safe to say that the next ten years will see a much smaller number of cases operated upon by this method than by some of the others. Conservatism in this field is becoming more and more pronounced and the limitations of tendon transplantation are being much more accurately defined. It is now generally conceded that it is practically useless to do it prior to ten or twelve years of age. This is true by reason of the fact that prior to this age

it is almost impossible to secure the coöperation of the patient for the development of the special functions in the transplanted tendons. Of course, this dies not obtain in that class where skilled aids can be employed over a long period of time and where the patient can be given a special training that is so essential to success, but unfortunately so many of these cases cannot afford such special training and care, and in such patients the procedure must be left until a later period. The social status, therefore, of the patient becomes of the utmost importance in determining what shall be done for the correction or the elimination of a deformity.

For the resultant paralysis (that which persists after five or six years have passed), when the probability of any further regeneration of muscle and nerve tissue or of nerve cells is entirely gone, another type of surgery can be considered, and its most useful field is in the correction of many of these permanently crippled and deformed. This is really the third type of surgery which can be well termed. "reconstructive surgery," because it has for its purpose the rebuilding of a part or of parts for the purpose of establishing one or two essential functions in the part even though at the expense of all other functions. Under this heading are included those operations generally of a destructive character, such as arthrodesis, resection, cuneiform osteotomy, tenodesis, bone transplantation and the construction of ligaments out of tendons, fascia and other structures. It is not my purpose to detail all of the operative procedures under the constructive and reconstructive headings which have proven themselves of value in these paralytic cases, as the time required would be entirely too long. A few, however, stand out rather prominently in their worth and these we will mention.

Before proceeding to the consideration of these, however, it is pertinent to speak of a procedure from which the greatest possible benefit was at first hoped, viz., that of nerve transplantation and nerve grafting. On theoretical grounds these operations should have proven the solution of the surgery of infantile paralysis, but unfortunately human tissues and structures will not always follow functionally and anatomically the surgeon's theories. The direct transplantation of a live nerve into the distal end of a severed paralyzed one has always resulted in failure; likewise, the anastomosis of a live with a paralyzed nerve has availed nothing. The direct implantation of a live nerve into a paralyzed muscle has resulted in a slight growth of the neuraxes into the muscle tissue, but there has been no regeneration or functioning of the muscle tissue as a result. Live muscles have been carefully anastomosed over a large surface with paralyzed ones in the hope that neurotization of these would occur but without results. It has often occurred to the writer that if it were possible to determine very early in the onset of the paralysis which muscles would be permanently paralyzed, a transplantation of a live nerve into those muscles before degeneration had taken place or perhaps into the nerve trunks before they underwent degeneration, might preserve the functions in these parts. However, on account of the nature of the infection in the spinal

cord and of the involvement of the motor cells, such a determination is absolutely impossible.

Experiments on animals have been recorded wherein a motor nerve has been resected and a portion of another motor nerve has been inserted into the distal part; after six weeks or so, a return of power was noticed in the muscles supplied by the resected nerve, which gradually increased and in due time approximated the normal, and it was concluded that there had been an establishment of function through the transplanted nerve. Careful dissection of the part, however, showed that the original nerve which had been resected had reëstablished its connections with the distal portion and that the motor function took place through this and not through the transplanted nerve (Steindler). This field of surgery, however, is so intensely interesting and theoretically so promising in results that investigations will be continued with these structures until it shall have been proven either an assured possibility or a total failure.

About 1910, Putti of Bologna, Italy, acting upon a suggestion of his predecessor, Codivilla, began the manufacture of ligaments out of the tendons of paralyzed muscles for the purpose of stabilizing joints and maintaining a part in a given position. His experience with the silk ligaments and other foreign substances had been similar to that of other operators, and it was felt that if normal tissues or structures could be utilized to maintain a part in balance, the danger of loss of the restraining ligament would be obviated. Codivilla had first buried the tendon under the periosteum in order to fix the proximal end for purposes of holding a part, and this plan has been amplified by Gallie of Toronto and has been extensively followed by many orthopædic surgeons. Putti, however, separated the tendon from the muscle and passed it through a hole drilled in the bone and then back between the periosteum and the bone and securely fastened it to itself and the periosteum. This plan of operation has given better results in my hands than any other procedure, though many operators have reported failures in its use. There are several factors essential to successful tenodesis after both methods. In the first place, it has been stated by many men that the tendons of these paralyzed muscles stretch under the effects of strain. My own opinion is that it is not the tendon which stretches but rather the old degenerated muscle to which the tendon is attached. If the tendon has been detached from this structure and fixed into a bone and recurrence of the deformity has occurred, the recurrence has been due not to the stretching of the tendon but to a slipping of its attachment, which is fibrous in all cases and which proved in the earlier days of transplantation entirely unreliable. Then too, if, in the fixation of the tendon, complete removal of all peritendinous tissue or sheath has not been accomplished, this tissue will unite with the bone and periosteum, and under strain the tendon will slip through and allow recurrence of the deformity. Histologically, tendons are but very slightly elastic and the amount of stretching which takes place under tension is extremely limited. Another factor in

the recurrence of deformity after this operation is that between the two attachments of the tendon now made into a ligament, there are usually several joints and the flexion or bending of these joints permits of movement of the part without any lengthening of the tendon structure. This is well seen in the front or on the side of the foot where a ligament has been formed from a neighboring tendon. There is still much to be desired from tenodesis in these cases, but there is no doubt that this method of restraint offers better and more lasting results than any other yet devised. Where the pull on this ligament is a direct one as from the posterior part of the tibia to the os calcis when the tendo Achillis or a portion of it is fixed into the tibia, the part will stand quite a degree of strain after firm union has been secured.

Peckham, of Providence, has utilized for the same purpose a strip of fascia dissected from the side of the leg and passed downward through a tunnel under the skin and attached to the foot below. This forms a dense scar of connective tissue which aids in maintaining a part in a given position. However, the great tendency of scar tissue to stretch, limits the amount of strain which such a structure will carry. A strip of fascia from the side of the thigh or other part may also be used as a free transplant to form a ligament, though the free flap transplant is more liable to become infected and discharged than is one with a pedicle and hence is not so safe a procedure.

Another operation which has proven of very great value in stabilizing a foot in which there is very free lateral motion, especially between the astragalus and os calcis, is one originated by the late Dr. G. G. Davis and is known as *sub-astragalar arthrodesis.* The real purpose of the fixation of the articulation between the astragalus and os calcis is the fixation of the heel. It is well recognized that if the heel of the foot can be maintained in a proper position, the rest of the foot will likewise remain balanced. The destruction of the joint is accomplished through a lateral incision below each malleolus, the tendons in each side are displaced, and with an osteotome or chisel the entire articulation is thoroughly destroyed, the pieces of cartilage and bone being left *in situ.* The lateral incisions are closed and the foot is held in plaster of Paris for two or three months until firm union between these bones obtains. In some cases, it is well to combine with this a destruction of the astragaloscaphoid joint as well. These operations are extremely simple, free from danger and have proven most satisfactory in cases of lateral instability of the foot. Tendon transplantations and tenodesis may be combined with this operation.

Where the ankle itself is flail, and there is no prospect of any further regeneration of muscle, many operators have performed an arthrodesis of the tibio-astragalar articulation to secure stabilization of the foot. When this result obtains, the walk becomes extremely awkward, though, of course, in standing the position is excellent. However, the ultimate results of a large number of cases over a long period of years have practically determined its abandonment by most operators. Greater reliance is now placed upon the

use of the Whitman or the Davis operation combined with tenodeses and ligament construction.

Another form of deformity which has proven extremely troublesome is that of calcaneus due to paralysis of the gastrocnemius and soleus muscles. Whitman, of New York, many years ago originated a plan of treatment which has proven most satisfactory in these cases. The mechanical principle involved is that of placing the weight of the body nearer the centre of the foot, and he accomplished this by removing the astragalus and then dislocating the foot backward after having made a new seat for the malleoli as far forward as possible. Valgus or varus may be present in these cases and either of these malpositions must be considered in the after placement of the foot. Whitman also transplants certain of the posterior muscles into the os calcis in order to assist in maintaining the foot in equinus. When plaster of Paris is applied, the foot is placed in the position of equinus and recovery takes place in this position, which not only eliminates the former deformity but also compensates for the shortening caused by the removal of the astragalus. In the severe cases of calcaneus, there is no other surgical procedure which will give as good results as astragalectomy and as in all operative procedures in paralytic cases, the after treatment must be most carefully carried out to safeguard against relapse. In the milder cases of calcaneus, where there is a less degree of contraction of the plantar fascia and the os calcis is not tilted forward so far, Davis secured the same mechanical result as Whitman by performing what he called a *transverse horizontal section* and dislocating the foot backwards. This operation is accomplished through an incision on one or both sides of the foot, and then with an osteotome the body of the astragalus is cut through horizontally just below the malleoli and the incision is carried in the same plane forward through the tarsal bones; the periosteum is then freely separated from the tarsal bones below the incision in front and on each side, and the tissues on the posterior portion above the incision are dissected up sufficiently to allow the foot to be dislocated backwards a half inch or more. The skin incisions are closed, the foot is pushed backward as far as possible and is preferably held in plaster in the position of equinus for from eight to ten weeks. Tendon transplantation and tenodesis may be combined with this procedure. This method is extremely efficient in the milder cases and possesses a great advantage over astragalectomy in that there is no shortening, furthermore, the operation is not at all serious and is not mutilating.

At the knee-joint a flail condition in an adult is best controlled by an arthrodesis and the patella may be advantageously ankylosed to the femur and tibia at the same time to provide greater stability. Where there is weakness or paralysis of the quadriceps extensor muscle, much good may be accomplished by the transplantation of a live biceps, external hamstring or sartorius into the patella. Where this attachment holds, a brace at the knee can usually be dispensed with. For faulty lateral positions of the leg at the knee, an osteotomy gives the most satisfactory results.

A flail hip proves one of the most difficult conditions with which the surgeon has to contend. If there is ilio-psoas power, a brace can be applied which will enable the patient to walk, but if there is total paralysis, arthrodesis of the hip-joint is the only procedure which will be of any benefit to the patient. In all of these cases where ankylosis is attempted, especially in the larger joints, the patient should be above ten years of age and better results are obtained in the older cases than in the younger ones. Firm fibrous ankylosis is preferable to flaccidity, and in some of the tarsal joints of the foot an arthrodesis may be done under ten years to advantage.

Another distressing condition in the paralytic cases is that of a scoliosis which develops with involvement of the spinal and abdominal muscles. One of the most difficult mechanical problems is the fitting of a support which will maintain the body of one of these paralytics in a straight position. The skin will not stand the pressure that is necessary for the support of the upper trunk and extremities. A plastic operation on the spine offers better results in the treatment of these cases of permanent paralysis than any other procedure. This operation may be that of a bone graft taken from the tibia or of a plastic character as advocated by Hibbs of New York and Forbes of Montreal. The spine is fixed at the point of greatest curvature, but before the operation is done an attempt should always be made to secure as much correction of the curve as possible. External fixation by plaster, jacket or brace is maintained for one or two years until bony ankylosis is complete.

The arm paralyses offer greater difficulties to surgical improvement than do those of the lower extremity. The most difficult with which to deal and in which to secure functional benefits are those of the shoulder. Muscle transplantations about this part have been performed but the degree of improvement which has been obtained is not of the most encouraging character. The trapezius has been used to supplant the deltoid, as has also the pectoralis major, but the leverage of the arm at the shoulder-joint is so disadvantageous that the results from these operations are not brilliant. I have obtained better results in the deltoid form of paralysis by an arthrodesis of the joint with the humerus at an angle of about 60 degrees. This posture enables rather free use of the arm through the action of the scapular and other chest muscles, and the function may be improved at least 50 per cent. Where there is loss of the biceps with inability to flex the arm, Sir Robert Jones advocates the removal of a diamond-shaped flap of skin from the front of the elbow and the closure of the edges of this denuded area to obtain a permanently flexed position of the arm. Where there is good power in the wrist and hand this operation aids very much in certain functions of the arm. In paralysis of the flexors or extensors of the fingers the transplantation of the carpi radialis or ulnaris of either the flexor or extensor group may be utilized to very great advantage. Almost any of these specialized tendons lend themselves well to transplantation, and attachment of tendon to tendon may be made in these parts with advantage because of the greatly lessened strain upon the point

of union as compared with that of the foot and leg where the element of body-weight proves of the greatest possible disadvantage.

While the surgery of infantile paralysis has been proven to be one of the most beneficial agents in correcting deformities and in reconstructing parts for the development and maintenance of function, there are two aids which may ever be lost sight of in the quest for success. These two aids are proper and efficient mechanical support until sufficient power has developed to take care of the condition and developmental exercises and training to improve and increase the power which may remain in any of the muscle structures. Coördinate and coöperative efforts are absolutely essential to this work. Patience, perseverance and an unlimited fund of optimism are indispensable to the securing of the ultimate results in these cases. One may never relax his efforts for a moment until the final purposes of the work are assured, even though the efforts may require years for their accomplishment. When the surgeon can examine one of these deformed paralytics which is commonly considered hopeless by the physician and the friends, and can visualize the end results of treatment which may require a period of years for their accomplishment and can then carry out the plan of treatment to a successful termination, he experiences a sense of satisfaction in the improved or finished product of his labors which can come from no other source.

ANNUAL ORATION, DECEMBER 4, 1922

SURGICAL RELATIONS OF THE SYMPATHETIC NERVOUS SYSTEM

By George P. Muller, M.D.

of Philadelphia, Pa.

Much might be written upon the relations of the sympathetic nervous system to abdominal diseases and their symptoms. The greater part of the sympathetic ganglia are massed in the abdomen, the most important actions of the vagus nerve, except its influence on the heart, are exerted there, and in the pelvis we have the sacral autonomic. Beginning a number of years ago with the appearance of the monograph of Eppinger and Hess and continuing to the present day, attempts have been made to rewrite physiology and pathology upon the basis of vagotonia and sympatheticotonia. Something has been gained, but as Carlson [1] well says, "The uncritical use of these terms in the sense of established etiology of well-known disease complexes retards medical progress." Alvarez [2] believes that while the theories of vagotonia and sympatheticotonia may eventually prove useful, he thinks that the foundations are so shaky that some day the whole edifice is going to go. As Alvarez puts it, we should learn from Gaskell and others the lesson that the sympathetics are not separate and distinct brain systems, but are there to conduct and not to exercise faculties requiring almost human intelligence. "There are times when the animal as a whole needs to communicate with its digestive tract; there are times also when one end of the tract must communicate with the other; and on all these occasions the extrinsic nerves come into play. The vagi carry feelings of hunger and of satiety from the stomach to the brain; they help in adjusting the tone of the stomach wall to the food coming down the œsophagus; and they carry the stimuli that give rise to the psychic secretion of gastric juice. If the food must be rejected by vomiting, they carry the impulses which bring the abdominal muscles to the aid of the stomach. Moreover, they probably carry messages from the digestive tract which make the animal feel comfortable and sleepy. The splanchnics serve largely to quiet the tract and to stop digestion when the body is distressed or injured. The extrinsic nerves probably have much to do with the digestive upsets with disease elsewhere in the body, but these changes can be accounted for also by actual damage to the gastro-intestinal muscle."

Some years ago, enthusiastic over Eppinger and Hess, I tried to work out a complex which would distinguish a free and supposedly chronically

diseased appendix from one adherent to the parietal peritoneum or cæcum, based on purely vago-phenomena of the former, but my analysis was not fruitful. I am not sure that the vagus control stops at the end of the ileum and furthermore an irritated ascending colon will produce spasm of the ileo-cæcal valve and imitate the vagotonic group of symptoms.

Abdominal pain and discomfort constitute the keynote of the group of symptoms characteristic of surgical abdominal disease, and it is a commonplace for us to say that in the area supplied by the sympathetic network there is no pain, that the liver, intestines, stomach or other viscera may be touched, cut, pinched or burned and yet not feel pain. Head,[3] in a most interesting paper published recently, calls our attention to the fact that so long as the internal organs are discharging their functions normally the afferent impulses which they may originate do not enter consciousness. Internal surfaces are unable to respond to such artificial stimuli to which they have never been exposed during the life of the individual or race. But the hollow viscera, such as the stomach and the bladder, react briskly to changes in tension, which are their natural mode of stimulation. Hyperacute or long-continued visceral irritations may overcome the resistance of the higher centres and sensation follows. Head applies this reasoning to appendicitis. "In the early stages before perforation has taken place, widespread pains may be present in the abdomen corresponding to the afferent supply of the upper parts of the digestive tract. Such pains are due to abnormal movements of the stomach and intestine and express the reaction of normal parts to a lesion situated in some allied physiological system." When perforation occurs these phenomena are replaced by the local manifestations.

Spasm is the most important physiological phenomenon resulting from intra-abdominal irritation and the familiar pylorospasm is the most conspicuous example. Surgical treatment aims to remove such irritation and relieve spasm, as after appendicectomy, gall-stone operations or gastro-enterostomy. A more direct method of attack has been proposed by Braun,[4] who advocates division of the gastro-hepatic and gastro-colic omenta in the region of the pylorus to alleviate painful stomach spasm. Enterospasm may occur and I have operated on at least two patients suffering from acute abdominal pain in which a contracted segment of the small intestine several feet in length was the only finding. In one case it relaxed and dilated while I was handling it. A third patient had contraction of nearly the whole length of small intestine following a gunshot wound with a perforation. The relation which such conditions bear to the vagus and sympathetic, the action of the pelvic sympathetic on the bladder and uterus and so on, are of interest and importance, but I must pass over this vast chapter. I have had two cases of automatic bladder after complete destruction of the spinal cord in the dorsal region. The patients are able periodically to empty the bladder, although voluntary control is lost. That this does occur often is due to the coincident cystitis interfering with bladder action.

From the standpoint of operative surgery little is done directly to the abdominal sympathetics. A new method of local anæsthesia has been introduced by Kappis and others whereby the splanchnics are blocked by injecting the semilunar ganglia. Let us next consider the peripheral sympathetics.

Resection of the Cervico-thoracic Nerve.—This operation, advocated principally by Jonnesco, was at first performed for the treatment of epilepsy, but was then applied to the cure of exophthalmic goiter, migraine, trifacial neuralgia, glaucoma, and recently to angina pectoris. As done by Jonnesco the entire nerve is removed except in glaucoma, where only the superior ganglion is resected.

Epilepsy.—Cervical sympathectomy proved a failure as a treatment for epilepsy. The anatomical basis for the operation rested on the fact that the superior cervical ganglion is connected by post-ganglionic fibres with the blood-vessels of the brain, the constrictor fibres probably following the course of the internal carotid artery. A number of deaths attended the operation not only in epilepsy, but in goiter and other diseases, and a probable cause has been brought out recently by Orr and Sturrak,[6] who found distinct lesions consisting of a paralysis of vessels in the cerebral cortex, Ammon's horn, amygdaloid nucleus and pyriform lobe after section of the cervical sympathetic. A new operation recently suggested by Fisher [6] probably is destined also to the limbo of useless procedures. He advanced the theory that a reduction of the adrenal substance in the body would reduce the tendency to convulsions. Accordingly, adrenalectomy has been performed about thirty times by four or five German surgeons, but the results were disappointing. More important than epilepsy is the relation of the sympathetics to goiter.

Goiter.—It has long been known that the sympathetic nerve supply of the thyroid gland follows the superior thyroid artery, and Drobnik [7] believes that nerves are given off from the second cervical ganglion uniting with the first cardiac nerves and then sending branches along the inferior thyroid arteries. Jaboulay, Jonnesco and others some years ago advocated the removal of the cervical sympathetics for exophthalmic goiter but the operation has never met with support. Ligation of the superior vessels is regarded by many surgeons as not only favoring anæmia, but also as a means of cutting off the sympathetic supply. Crile [8] states that "the greater part of the benefit from ligation is the result of a break in the nerve supply of the thyroid since the principal sympathetic nerves run in the walls of the superior thyroid arteries." Recently, Odermatt [9] reports the results of a study of the sensations experienced during ligation of the thyroid vessels. If the artery was dissected bare, ligation was painless. If periarterial tissue remained, the ligation pain was constant in the anterior branch of the superior thyroid artery but inconstant in the posterior branch of the superior thyroid and in the inferior thyroid. Leriche [10] practised superior thyroid sympathectomy on one side in a case of goiter and noted a remarkable regression in its size. He also [11] speaks of supplementing this operation in toxic cases with tachycardia by resection of the superior cardiac nerves, and of the control of the exophthalmos by pericarotid sympathectomy. Some years ago I performed bilateral removal of the superior cervical ganglion for persisting exophthalmos, but while some improvement was noted the patient did not consider the result as satisfactory.

Glaucoma.—I also, in one case, removed the upper ganglion for acute glaucoma, but the operation failed to save the eye. A number of years ago this was thought justifiable and Wilder [12] collected sixty-eight cases with good results in the majority, especially in the chronic form. The operation seems to be rarely done at present and De Schweinitz [13] does not advocate it.

Optic Atrophy.—Sympathectomy has been performed for this condition by Abadie,[14] who resected one centimetre of the carotid sheath to interrupt the sympathetic innervation. The atrophy in his case was characterized by a restriction of the visual field on the nasal side. The immediate results were brilliant, the visual field becoming normal in approximately two weeks, but two months after the operation the condition retrogressed until about the same as before operation. Ligation of the carotid was followed by similar transient improvement.

Parotid Fistula.—In 1914, Leriche [15] proposed resection of the auriculo-temporal nerve to suppress parotid secretion, based on Bernard's observation that this nerve is the secretory nerve for the parotid gland. The operation has been endorsed by Dieulafe,[16] Weitz,[17] Tromp [18] and others, but Cole [19] condemns the operation by comparing it to the plumber who would repair a leaky pipe by cutting off the water supply of the house.

Angina Pectoris.—Without attempting any argument of the somewhat obscure etiology of this disease, I can recount the attempts at its cure, or rather amelioration, by surgical measures. In 1916, Jonnesco [20] removed the middle cervical ganglion, the sympathetic trunk, the plexuses about the inferior thyroid and vertebral arteries, the inferior cervical and first thoracic ganglia of the left side in a case of typical angina pectoris. Four years later he examined the patient and reported the case. During the subsequent four years the patient had no attack of angina and follows his occupation as a clerk. The cardiac action, pulse and respiration were found normal. X-ray examination showed a slight dilatation of the aorta with thickening and a broadening of the heart shadow. This report was followed by the report of another case. The second patient operated on by Jonnesco [21] was fifty-four years old and for eight years had suffered from attacks of suffocation and from paroxysms of pain radiating to the cervical region and left arm. The left cervico-thoracic nerve was resected and marked improvement was noted during the ensuing four months. Jonnesco apparently believes that the phenomena of angina pectoris are due to irritation of the terminal filaments of the nerves of the sympathetic plexus in the walls of a chronically inflamed aorta. He only resected the left side because the patient refused to go further, and was surprised at the completeness and permanence of the relief.

Jonnesco's operation has brought to light another case, namely that of Renon, who had a woman patient with aneurism of the aorta which was dis-

creet and fusiform. The case was referred to Tuffier,[22] who exposed the aorta by a transverse division of the sternum and wrapped the length of the aneurism with a strip of fascia lata. Renon concluded that there was some amelioration of symptoms and the general state better, but I would judge that the operation actually accomplished little. However, in the discussion of this case Delorme pointed out that the act of freeing the aneurism caused the division or removal of the sympathetic plexuses, which really supplied the reason for the relief of pain. In a later paper Delorme[23] suggests that sympathectomy would be worth while as an effort to relieve the pains of aortitis.

Periarterial Sympathectomy.—In 1913, Leriche published his first communication and since then about twenty others have appeared from this surgeon alone. The technic of the operation is well known and consists in the removal of 8 or 10 cm. of the adventitia of the artery. While the stripping is in progress a marked contraction of the artery is noted, followed by a peripheral dilation which becomes attenuated in five or six days and disappears after three or four weeks. This vasodilation, according to Leriche, is the therapeutic effect of the operation, but Bruening and Stahl[24] believe that the hyperæmia is not the only factor at work in producing beneficial results from sympathectomy. There is a transposition of the entire vascular function as a result of the interruption of the normal sympathetic stimulation. The autonomous nature of the vascular nerve apparatus explains the return to normal after a brief period of vasodilation. Handley[25] very recently has published a substitute method for sympathectomy as performed by Leriche. After exposing the artery he injects four minims of alcohol at each of four equidistant points around the circumference of the vessel, the needle being introduced obliquely into the tunica adventitia. Two cases were injected, both of actual gangrene. In the first there was recession of the line of gangrene and the formation of new lines of demarcation around the toes. In the second, an unfavorable case, the operation brought but slight benefit but definitely accentuated the vasodilation already present. Handley believes his method superior to the original one because it is simpler to perform, does not produce the initial stage of vasoconstriction and attains immediately the vasodilator results aimed at by the operation. As usual we have a German claim for priority in the discovery of this operation in the person of Heinrich Highier.[6]

In his article in the ANNALS OF SURGERY last year, Leriche[27] reports that he has performed sympathectomy sixty-four times for various affections, sometimes with remarkable success and sometimes with complete failure. Many other cases are recorded in the literature and I myself have had the opportunity to do this operation thirteen times on eleven patients. The anatomical aspects of the operation are rather interesting. The arterial sympathetics are probably remains of the primitive nerve nets of the low scale animal and perhaps act as "booster" stations for the vasomotor impulses coming in from the somatic nerves. How much autonomy they may have is

not clear. They are situated in the adventitious tunic of the artery. The vasomotor innervation of the arteries of the extremities is made possible by the re-entry into the spinal roots of sympathetic fibres by way of the gray rami communicantes; they intermingle with other afferent and efferent fibres and pursue a straight course to the periphery. Along the course of the peripheral nerves twigs are given off from time to time which connect with or form the sympathetic net on the blood-vessels. But little information is given in the text-books regarding these twigs and our knowledge is rather scanty.

The Nerves to the Arteries.—In 1914, some observations from the Western Reserve University were published which sum up and add to the existing knowledge. Kramer and Todd[28] investigated the nerve supply to the arteries of the arm and found that the subclavian and proximal part of the axillary arteries received a nerve supply directly from the sympathetic chain, between or including the middle and inferior cervical ganglia which reached the artery in the interval between the scalenus anticus and the bone. The portion of the subclavian artery immediately adjacent to its origin was supplied by a varying number of twigs from the musculo-cutaneous nerve, the radial from the superficial ramus of the radial nerve and the ulnar from the ulnar nerve in the forearm. Todd and Kramer point out that, "the more distal arteries are supplied by sympathetic fibres which have travelled to their distribution along special nerve-trunks and not along main vessels. These twigs are distributed to the vessels from the nerve-trunks at intervals; the intervals growing shorter as the more distal portions of the limb are reached, as though a greater wealth of nerves was needed in these parts. Possibly the diminishing size of the member and consequently the greater need for constant regulation in size of vessels may be associated with this fact. Again the distribution of nerves to vessels corresponds pretty closely with the distribution of nerves to the skin and musculature of the same area." The clinical significance of the nerve supply to the blood-vessels of the upper extremity is well illustrated in cervical rib. The symptoms of the lesion may be motor, sensory, vascular, or sympathetic. In the latter case it is not necessary that the sympathetic net on the subclavian artery must be involved because pressure on the brachial plexus may involve the sympathetic fibres reaching into the median ulnar or radial and thence to the arterial sympathetic. Todd[29] reported a case in 1912 with no palpable pulse, operation revealing no compression of the vessel, and in which he believes the vascular phenomena were sympathetic in origin. I think I have seen an exactly comparable case in which vascular symptoms began at the periphery and there was no palpable pulse. Sympathectomy was followed by relief of pain and the hand became warm. Later, cervical rib resection was followed by cure.

Another interesting point has been brought out by study of the arm sympathetics. Tournay[30] has shown that section of the sympathetic produces a reinforcement of sensation. Regard[31] reports a case where after suture of an ulnar nerve, sensation was restored almost immediately and vasomotor

disturbances disappeared. Motion was not affected even after eight months. He explains the phenomenon by the fact that the dissection removed fibres of the sympathetic about the ulnar which permitted sensation to be transferred by the median nerve.

The distribution of nerves to the arteries of the lower extremity has been investigated by Potts.[32] He found that the femoral artery received branches, after its bifurcation into the superficial and deep femorals, from some independent twigs and from muscular branches of the femoral nerve; the lower part may receive a twig from the saphenous nerve. The popliteal artery is supplied by the tibial and in many cases also by the azygos nerve. The posterior tibial is richly supplied from the tibial nerve and from the nerve to the flexor hallucis longus muscle, the perineal artery is reached by the muscular branches to the popliteus and flexor longus hallucis. The anterior tibial and the dorsalis pedis receive twigs from the peroneus profundus.

Leriche divides the phenomena resulting from injury of the periarterial sympathetic plexus into two groups: In the first group the characteristic physiologic reaction is pure, with two striking aspects, painful ischæmia and consecutive vasodilation; in the second, reaction is disturbed and gives various troubles.

In the first, Leriche classes "stupeur arterielle" and Raynaud's disease. Sudden arterial spasm may occur and be so intense as to lead to gangrene. It may occur after trauma, such as fracture or in war wounds where concussion of the artery has occurred. Reichle[33] has noted two cases of segmental spasmodic contraction of a large vessel after trauma. If not recognized unnecessary amputations may be done. Another phase of this gangrene has been described by Oppel,[34] writing on spontaneous gangrene, who believes that the adrenal plays a part in its production through overaction, the increased amount of adrenin causing an ischæmia and a disturbance in the nutrition of the arterial walls. We might theorize that if this is so and if the effect of the emotions, particularly fear, is to increase the flow of adrenin, then we have a reasonable explanation of the etiology of Buerger's disease in the Russian Jew.

Raynaud's Disease.—First described by Raynaud in 1862, this affection is distinctly a disturbance of the vasomotor mechanism. The local syncope and the asphyxial attacks are constrictor in nature. Rarely the dilator phenomena of hyperæmia are observed. Halpert[35] has described in detail the capillary changes in a patient with typical Raynaud's syndrome. She found increased tortuosity of the capillaries, as well as groups of capillaries from three to five times larger than normal. The blood flow was slow. During an attack the giant capillaries became fuller, especially in their venous portion, and exhibited changes in contour, such as projections and strictures. The blood appeared to be pushed through the vessel by a peristaltic-like wave. In a severe attack the blood became completely stagnant and blue. These observations correspond strikingly to the explanation originally offered by Raynaud. Gangrene is a terminal phenomenon and is usually characteristic.

The essential features of this disease are well known and need not be repeated here. Recently Buchanan[36] has presented a study of the cases seen in the Mayo Clinic, sixty-seven in number.

Judging generally by the reported cases the results of treatment in this disease have not been very successful. If we agree that vasoconstriction is the predominant influence then sympathectomy causing dilation is worth while. Leriche has twice done this operation in Raynaud's disease with good results. I have performed sympathectomy on both brachials in a case of Raynaud's disease occurring in a man seventy years old. He had the characteristic "dead fingers" with other symptoms and early gangrenous patches on the skin of the hands. He was practically cured. Perhaps we are justified in adding to this group a condition known as acrocyanosis.

Acrocyanosis.—Many of us have no doubt noted the occurrence of cold and cyanotic hands in certain persons, particularly those of an asthenic type, and Cordier[37] believes that a localized arterial hypertension, especially of the hands and feet, will occur more frequently in the future as the result of "the intoxications, commotions, fatigues and latent infections" of the War. Sufferers from the so-called "irritable heart" of soldiers often exhibit acrocyanosis to a marked degree. Boas[38] studied twelve cases of acrocyanosis and found that "when the hands are cold and cyanotic, the capillary blood-pressure is low and the flow sluggish. This cannot be due to a constriction of the venules, but must depend on a constriction of the arterioles or a marked dilatation of the capillaries. It is significant, too, that the capillaries become fuller when the hands are warm. If the venules were constricted, the capillaries would be engorged during the period of cyanosis."

Warmth will usually accelerate the blood flow in the capillaries, but in intractable cases I would suggest the performance of sympathectomy as an experiment to note the permanency of the resulting vasodilation.

In the second group described by Leriche are placed a miscellaneous number of affections characterized by a disturbance of physiological reaction from contracture of too long duration or abnormally persisting dilatation. The only pathology noted is an adhesion of the vessels to the common sheath, or an increase of the vascularization of the adventitia. Sometimes nothing is seen. Leriche no doubt is over-enthusiastic when he ascribes disturbance of the sympathetic innervation as the cause of trophic ulcers following nerve section, but there is some ground for the opinion that it plays a part. Stopford[39] believes "there is strong reason to conclude that irritative nerve lesions can produce changes in the walls of the arteries supplied by the affected nerves. These changes seriously reduce the calibre of the vessel and must inevitably diminish the blood supply to the muscles, bones, joints and skin." It has been pointed out also that injury in regions remote from the large blood-vessels may be accompanied by pain or trophic disturbances, but Leriche counters by stating that with injury in a richly furnished zone of sensory innervation the vasomotor disturbances may be due to ortho- or antidromic reflexes, starting from the injured point, and referred back along

the periarterial sympathetics. Much was written during the War about the effects of nerve ischæmia in wounds of arteries, and trophic, sensory, and motor abnormalities were noted. The reaction was thought to be similar to that occurring in the optic nerve after thrombosis or embolism of the central retinal artery. Ischæmic myositis after blood-vessel injuries has been discussed also by Stewart [40] and ischæmic paralysis well presented by Burrows.[41]

Physiologic research is needed to further elucidate the importance of the sympathetics in these conditions, but all in all there is much to support the claim of Leriche.

Clinically, successes have been claimed in (1) causalgia after war wounds; (2) certain painful crises preceding gangrene caused by obliterative endarteritis; (3) vasomotor trophic neuroses with contractures; (4) painful stump; (5) trophic ulcerations of stumps and extremities; (6) trophic œdema; (7) ischæmic paralysis of the forearm, etc.

In my own experience the most gratifying results, aside from the cases of Raynaud's and cervical rib already mentioned, have been in (1) a painful stump from amputation eight years previously and with almost continuous pain during that time. He had been operated on without relief six months previously. Following sympathectomy, complete relief was experienced. (2) A case of threatened or beginning gangrene of the toes with calcareous tibial arteries as shown by X-ray and marked pain, was not only cured of pain but the gangrenous areas cleared up in a most remarkable manner. I saw this man seven months later and his foot is perfectly well, except for cyanosis in the dependent position. (3) A case of tropho-neuroses with contractures and pain in the foot. Marked relief of both attended sympathectomy. One of both attended sympathectomy. One case of painful stump was a failure, two cases of Buerger's disease were slightly improved, a case of gangrene of the fingers with bilateral sympathectomy was distinctly improved. I had one fatality in an elderly woman with acrocyanosis and arteriosclerosis, following infection of the wound, hemorrhage and death after ligation of the femoral artery. Matona [42] reports a similar occurrence. Recently I assisted Doctor Frazier perform sympathectomy in a case of tropho-neuroses of unknown origin with burning pain and œdema of the foot, the patient being completely relieved by the operation.

It will be noted that six of the patients were cured and in one case gangrene was probably stayed; the other four must be classed as failures.

Causalgia.—This is a painful vasomotor neurosis resulting from irritation of a mixed nerve. It was first described by Weir Mitchell and he coined the name. From the standpoint of this paper the following from Mitchell [43] is of interest: "Further study led us to suspect that the irritation of a nerve at the point of the wound might give rise to changes in the circulation and nutrition of the parts in its distribution, and that these alterations might be of themselves of a pain-producing nature." Many articles on causalgia have appeared since the War, mostly from French sources, the best article in English being, I believe, that by Carter.[44]

While the pain is essentially a peripheral reflection along somatic nerves, usually the sciatic or median, set up by peripheral irritation, yet the sympathetic plays a part in several ways. After division of one of these nerves pain may continue, the impulse travelling along anastomosing sympathetic fibres in a centripetal direction and thence reflecting along centrifugal fibres. Leriche believes that causalgia is due to a neuritis of the periarterial sympathetic system and not to the direct injury to the nerve trunk. But there is much evidence on record to show that causalgia may occur when no injury to the main vessel could have occurred. Potts, from his anatomical study referred to above, states that "local damage to a large artery will injure the vascular plexus at the point of damage only, but will not account for changes produced in the vessel at a distance from the injured site. If absolute proof can be obtained of the relation between damage to the sympathetic supply of an artery and morphological changes in the vessel itself of more than focal character, then the nerve damage must occur at some distance from the arterial tree, and not simply to the sympathetic plexus as it lies on the vessel."

Nevertheless theory sometimes must give way to facts. Leriche states that in causalgia after war wounds sympathectomy gave him in nine cases two complete failures, two satisfying improvements, and five excellent results. Platon [45] reports excellent results in eighteen cases, in sixteen the pain stopping at once and in two more gradually. The motor nerves were involved in all but two of the cases. Girou [46] differentiates causalgia from sympathetic irritation pain, and diagnoses the latter by the occurrence of flexion contraction of the hand, indicating a vessel lesion involving the sympathetics. You will remember that Lewis [47] following the suggestion of Sicard [48] reported three cases of causalgia treated by intraneural injection of 60 per cent. alcohol in which the patients experienced almost instant relief. He prefers this method to sympathectomy because of its simplicity.

REFERENCES

[1] Carlson: Journal A. M. A., 1922, vol. lxxix, p. 1101.
[2] Alvarez: Mechanics of the Digestive Tract. Hoeber, 1922.
[3] Head: British Medical Journal, 1922, vol. i, p. 1.
[4] Braun: Zentralbl. f. Chir., 1921, vol. xlviii, p. 267.
[5] Orr and Sturrak: Lancet, 1922, vol. i, p. 267.
[6] Fisher: Ztsch. f. d. ger. Neurologie u. Psychiatrie, 1920, p. 56.
[7] Drobnik: Quoted by Hertzler, Diseases of Thyroid Gland, Mosby, 1922.
[8] Crile: The Thyroid Gland, Saunders, 1922.
[9] Odermatt: Beitr. z. klin. Chir., 1922, vol. i, p. 127.
[10] Leriche: Lyon Chir., 1920, vol. xvii, p. 109.
[11] Leriche: Presse Méd., 1920, vol. xxviii, p. 301.
[12] Wilder: Journal A. M. A., 1904, vol. xlii, p. 350.
[13] De Schweinitz: Personal communication.
[14] Abadie: Presse Méd., 1920, vol. xxviii, p. 606.
[15] Leriche: Lyon Chir., 1914, vol. xi, p. 256.
[16] Dieulafe: Presse Méd., 1917, vol. xxxiii, p. 392.

[37] Weitz: Deutsche Zeitschr. f. Chir., 1919, vol. cxlix, p. 419.
[38] Tromp: Zentralbl. f. Chir. 1917.
[39] Cole: Lancet, 1919, vol. i, p. 971.
[40] Jonnesco: Bull. de l'Acad. de Méd., 1920, vol. lxxxiv, p. 93.
[41] Jonnesco: Bull. de l'Acad. de Méd., 1921, vol. lxxxvi, p. 208.
[42] Tuffier: Bull et Me. della Soc., 1920, p. 166.
[43] Delorme: Bull de l'Acad. de Méd., 1921, vol. lxxxv, p. 582.
[44] Bruening and Stahl: Berlin. klin. Wchnschr., 1922, vol. i, p. 1402.
[45] Handley: 1922, vol. ii, p. 173.
[46] Heinrich Highier: Berlin. klin. Wchnschr., 1922, vol. i, p. 1208.
[47] Leriche: Annals of Surgery, 1921, vol. lxxiv, p. 385.
[48] Kramer and Todd: Anat. Rec., 1914, vol. viii, p. 243.
[49] Todd: Lancet, 1922, vol. ii, p. 362.
[50] Tournay: Comptes Rendus de l'Acad. des Sciences, 1921, vol. clxxiii, p. 939.
[51] Regard: Bull. et mem. Soc. de Chir. de Paris, 1922, vol. xlviii, p. 619.
[52] Potts: Anat. Ariz., 1914, vol. xlvii, p. 138.
[53] Reichle: Beit. z. klin. Chir., 1921, vol. cxxiv, p. 650.
[54] Oppel: Lancet, 1922, vol. ii, p. 116.
[55] Halpert: Quoted by Boas.
[56] Buchanan: Amer. J. Med. Sc., 1922, vol. clxiv, p. 14.
[57] Cordier: Archiv. des Mal du Coeur. 1920, vol. xiii, p. 241.
[58] Boas: Jour. A. M. A., 1922, vol. lxxix, p. 1404.
[59] Stopford: Lancet, 1918, vol. i, p. 465.
[60] Stewart: Brit. Med. J., 1918, vol. i, p. 199.
[61] Burrows: Brit. Med. J., 1918, vol. i, p. 199.
[62] Matons: Semana Med., 1922, vol. ii, p. 98.
[63] Mitchell.
[64] Carter: Journal Neurology and Psych., 1922, vol. iii, p. 1.
[65] Platon: Archiv. Franco-Belg. de Chir., 1921, vol. xxv, p. 226
[66] Girou: Presse Méd., 1918, vol. xxvi, p. 584.
[67] Lewis: Journal A. M. A., 1920, vol. lxxiv, p. 1.
[68] Sicard: Lancet, 1918, vol. i, p. 213.

ANNUAL ORATION, 1923

POST-OPERATIVE PULMONARY COMPLICATIONS

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A review of the previous Annual Orations before this Academy finds the subjects following two definite lines, (1) the philosophical and (2) the technical. In choosing a subject for this occasion we promptly eliminated the philosophical. Your intimate knowledge of our work and limited years of experience would embarrass any attempt upon our part at the retrospective or perspective. The technical subjects presented have been based upon original research or have visioned new fields in surgery. Again we were forced to admit our limitations. Our final decision has been to discuss such a commonplace subject as post-operative pulmonary complications and we hope you will bear with us in the presentation of one of our hobbies.

Our interest in post-operative pulmonary complication began when in charge of a department of anæsthesia. This was at a time when all such complications were considered the direct result of the anæsthetic and incidentally the skill of the anæsthetist. The resentment of youth may have been the real incentive which stimulated an investigation of this problem at that time. Although the work was never published it served to convince us, and a few of the staff surgeons, that there were many etiological factors other than anæsthesia concerned in these embarrassing complications. Curiously anæsthetists are still passively accepting this easy explanation. Cutler [1] states that it is unfortunate that anæsthetists and anæsthesia should bear the blame of pulmonary complications following anæsthesia and operative procedures, when the facts seem to exonerate both in the majority of cases. This certainly is in accord with the experience of many surgeons.

The literature of the last few years has shown a very rapid increase in the incidence of these complications, and the present figures are such as to demand a serious consideration of the problem by both the surgeons and the anæsthetists. Undoubtedly this increase in the morbidity is largely, if not entirely, due to more careful physical examinations and better records so that the following figures more nearly represent the situation than any of our older statistics. Cutler in two of his reports, which were three years apart, shows a marked increase in his later group. It now seems well established from many sources that 1 in every 50 patients operated upon develops a pulmonary complication and one in every 150 to 175 developing such complications die, a morbidity of between 3 and 4 per cent. and a mortality

of about 0.6 per cent. Pepper,[3] McKesson,[4] Cutler and Hunt,[2] Norris.[5] With such figures the value of our generally accepted anæsthetic risks, ether 1–16,000, chloroform 1–3000, ethyl chloride 1–12,000, nitrous oxide 1–300,000, appear useless in estimating operative risks.

We can no longer regard all post-operative pulmonary complications as post-anæsthetic sequelæ, nor assume that the only risk of post-operative pulmonary complication arises in the anæsthesia. Long before the days of the routine use of general anæsthesia we find Norman Cheevers[6] writing in Guy's Hospital Reports, "that pneumonia is the most frequent cause of death after surgical procedures." The literature contains many references to the effect that the incidence of these complications is as great, and many claim greater, with local anæsthesia as with general, though the mortality when following general anæsthesia is slightly higher than in local. Mandl,[7] Gottstein,[8] Mikulicz,[9] Henle,[10] Sauerbrock.[11]

Instead of the anæsthetic being considered the most important factor (and the only one by many) in these complications, its greatest effect can only be contributory, and it should be considered with such other contributing factors as infection (either pre- or post-operative), preëxisting lung disease, old age and debility and the chilling of the body, all of which have been so carefully studied by Whipple.

It is now generally accepted that the site of the operation and the character of the procedure are the constant and all-important factors. The relation of the operative field to the diaphragm bears a direct relation to post-operative pulmonary complications and statistics show this relationship is the most constant of all etiological factors. Cutler and Hunt[2] give perhaps the highest figures. In a group of 63 cases of post-operative pulmonary complications 43, or 68 per cent., followed laparotomies. Mandl[12] reports a general morbidity of 8 per cent. following operations upon other parts of the body and 14.5 per cent. after abdominal operations. Norris[5] a general morbidity of 1.1 per cent. and 4 per cent. after laparotomy. Pasteur[13] a morbidity of 1.8 per cent. after operations upon the urinary bladder, while there was 13.4 per cent. after operations upon the stomach and 11.0 per cent. following operations upon the liver and gall-bladder.

In addition to the necessity of abandoning our complacent acceptance of anæsthesia as the sole cause of post-operative pulmonary complications, the work of Cutler also makes it necessary for us to abandon the all-inclusive diagnosis of "pneumonia" for these complications. With the more accurate physical examinations his work has stimulated there is a surprising decrease in this diagnosis of post-anæsthetic pneumonia. In the same group of sixty-three cases, previously referred to, Cutler demonstrated thirty-two as being caused by pulmonary embolism and infarction. Rupp[15] found at autopsy in 13,000 post-operative cases, 5 per cent. having demonstrable emboli and infarctions in the lungs. In our immature work years ago, embolism and infarction were found very frequently post-mortem, but their significance was not appreciated at that time. To Cutler belongs the credit of demonstrating

this condition and calling attention to it. The onset is usually abrupt, the physical signs are characteristic and febrile changes are sudden, except when the emboli occur in an aseptic field. In septic emboli the clinical picture may simulate pneumonia, or lung abscess may result. When the clot is sterile the resulting changes are characteristic of minor pulmonary infarcts. "From the second to the fourth day there is usually sudden pain on respiration followed by expectoration in about one-half the cases. The sputum is often blood-stained." "Preceding the onset of this symptom there is usually a rise in pulse, temperature and respiration, and with the pain these may increase." "Immediate auscultation of the chest reveals one or more small areas covered with fine râles over which there is some impairment of breath sounds and, if the focus is sufficiently large, some change in fremitus." "When pain is present a friction rub may be the most distinct sign." "It must be remembered that a friction rub results only when the area of the lesion reaches the periphery of a lobe." It also must be understood if we are to recognize all these lesions that some in the smaller thrombi do not cause sufficient pathology to give those physical signs. Cutler and Hunt advise Röntgen-ray studies as of the greatest value. Invariably they appear as small flurries of consolidation, which from time to time will take the form of a cone-shaped shadow with its base out. Röntgen-ray studies, moreover, should be made immediately, since these lesions chiefly represent merely a change in blood distribution and soon clear up. A definite resolution is complete as a rule within six to seven days. Of course, this process will vary according to whether the emboli are aseptic or septic and also upon the size of the embolus and the vessel in which it lodges. Thus you sometimes will have definite massive areas of infarction and in the lantern slide being shown there was pulmonary infarction which was distinctly demonstrable in the X-ray. In conclusion Cutler and Hunt with many others now believe that embolism from the operative field is the primary factor in all post-operative lung lesions and that all others are secondary and contributing factors only. Embolism is used in the sense of the transfer of small particles, which may or may not be sterile, from the operative field to the lungs, by either the lymphatic or blood channels.

It is our belief that pulmonary embolism and infarction will be found in a much larger proportion of post-operative pulmonary complications than has been reported up to the present time (Cutler 50.7 per cent.) as the character of our physical examinations improves. The importance of this phenomenon has been admirably presented by Cutler from whose reports we have freely quoted, and need take no more of our time.

There is, however, another post-operative pulmonary complication whose incidence is probably constant and may be as great as embolism and infarction, to which we wish to devote the rest of the time. *Post-operative Massive Collapse of the Lungs.*—Attention to this phenomenon was first called by Pasteur in a paper entitled "The Respiratory Paralysis after Diphtheria as

a Cause of Pulmonary Complications." [16] In 1910,[17] he stated that there was a close connection between the mechanism producing collapse of the lower lobes of the lung in post-diphtheritic paralysis and that underlying the collapsive attacks following operative procedures. In 1914,[18] he records a group of 201 post-operative lung complications, in which he recognized 12 cases of massive collapse or a proportion of 6 per cent. Since this report 28 cases have been recorded in literature to which we add 2, making a total of 42. We feel that a general recognition by surgeons of this possibility and a systematic search will greatly increase this incidence of 6 per cent. suggested by Pasteur. That in the last year we have encountered two definite cases and a possible third would suggest this. In several of the clinics in this country, routine X-ray examinations of the lungs are being made at the present time after all major operative procedures.

Undoubtedly varying degrees of pulmonary collapse occur and Briscoe [18] states his belief that the large majority of post-operative pulmonary symptoms are entirely due to varying degrees of pulmonary collapse. Pasteur [13] takes exception to this definition of the condition and would have us confine the term collapse to the condition of massive collapse, in which the lung is completely deprived of its air. When not completely airless, he suggests the term partial deflation. This seems to us unnecessarily confusing. Further, it would also exclude its constant presence as a modifying and often a determining factor in such other conditions as pulmonary embolism and infarction. To us its importance as a post-operative factor lies not in the occasional massive collapse we encounter, but that it always occurs in varying degrees after operative procedures, trauma and other conditions of which we will speak later. However, only massive collapse of one or more lobes has been recognized up to the present time. A brief recital of our own cases will probably present the phenomena in the clearest way and make possible a detailed discussion. The usual phenomenon is as follows: A few hours to as long as seven days after a surgical operation, usually abdominal, the patient suddenly presents the symptoms of a catastrophe. It is impossible at first to localize the condition, the thorax after a short time engages one's attention. Acute dilatation of the heart, coronary embolus, pulmonary embolism or pulmonary infarction are the common preliminary diagnoses. A more careful examination may suggest pneumothorax. There is usually only a moderate febrile reaction unless there is coincident infection. There may or may not be an increase in respiratory rate, sometimes reaching 30 or 40. A pulse rate and a respiratory rate directly related to the febrile reaction are to be expected, but otherwise they are remarkably undisturbed. The physical signs of the chest are perhaps the most characteristic findings. Upon inspection there is diminished or even absent respiratory movements of the chest wall over the affected area. The intercostal spaces apparently are hollow and very much narrower than upon the normal side. The cardiac impulse is seen displaced toward the affected side (just the opposite to that one finds in

a pneumothorax or effusion). The apex has a tendency to tilt outward and upwards, so that the apex of the impulse of the heart beat may often be felt in the axilla (this is particularly true when it occurs on the left side). In one of our cases, right-sided, it reached the right anterior axillary line and was first diagnosed by the house officer as a case of dextrocardia (a very frequent preliminary diagnosis). The dome of the diaphragm on the affected side is abnormally high and immobile. The high diaphragm is readily detected by percussion in the left-sided cases. On the right side, however, percussion is not so reliable but X-ray examination yields definite evidence both as to its position and immobility. These symptoms are common to all cases, in other words, physical symptoms which indicate a falling into the pleural space of the surrounding structures, namely, those of the mediastinum and diaphragm. A further study of physical signs divides the cases into two distinct groups. In both, dulness on percussion is present over the affected side and may extend as high as the clavicle; this is usually posterior but may be anterior. It corresponds to the area of the collapsed lung. The pleural space unoccupied by the collapsed lung is hyperresonant and may be tympanitic. In one group the vocal fremitus is diminished or absent, while in the other it is increased. In the group where the vocal fremitus is diminished or absent, the breath sounds are also diminished or absent, but when increased the breath sounds are loudly tubular or amphoric in character and bronchophony and pectoriloquy are also extremely well marked. This difference in the physical signs is probably dependent upon the patency of the bronchi. When there is a large proportion of air in them there is an increase in breath sounds which are loud, tubular, amphoric in character and bronchophony and pectoriloquy are present. One will readily see that the physical signs in the lungs are those commonly attributed to pneumonic consolidation but, if anything, the signs are even more marked, especially the tubular and amphoric character of the breath sounds. One of the main reasons why massive collapse of the lungs is so frequently overlooked, is that the tubular breathing is so extraordinarily well developed that its mere presence is at once regarded as conclusive evidence of the existence of pneumonic consolidation. Due regard to the other signs, namely, cardiac displacement, should make the diagnosis clear. In the type of case where there is dulness on percussion and diminished or absent vocal breath sounds the diagnosis is more difficult, unless adequate stress is laid upon the displaced position of the cardiac impulse. Broadly speaking, this type of case in which the bronchi are not patent is usually found in the early stages of the condition, while the patent bronchi are found in the later stages of expansion. In one of our cases the breath sounds and the transmitted voice when heard through the stethoscope were almost deafening. Râles and adventitious sounds may be present but are often absent throughout the entire process. Again they may be abundant, especially in the latter stages of the disease when the lung is reëxpanding. When, in rare cases, inflammatory complications develop in the collapsed lung, the presence of adventitious sounds will coin-

cide with the lesion developed, but they are not the essential signs of massive collapse. The cardiac displacement is the most characteristic physical sign and the condition cannot be diagnosed with certainty unless this sign is present. This marked displacement of the heart is rarely if ever accompanied by cardiac murmurs. Though the displacement is mainly lateral, in cases where the whole lobe or the upper lobe is involved the displacement is also upward, so that the maximum impulse may be felt in the third interspace or behind the rib. The Röntgen-ray corroborates all these physical signs and will be of the greatest aid when the lesion is on the right side. The lung shadows on the affected side will be more or less opaque and suggest a purulent pleural effusion in its degree of density. The extent and density of this shadow will, of course, vary with the amount of lung involved and the degree of airlessness. As the air returns this opacity gradually disappears, the opposite to a pleural effusion. Instead of an increased pleural pressure, as in pleural effusion, pushing away the heart and diaphragm, there is a negative one and the heart and diaphragm encroach upon or are drawn into the pleural space. The displacement of the heart toward the affected side is usually very marked and the dome of the diaphragm ascends to an unusual degree. The X-ray interpretation in one of our cases was a subdiaphragmatic abscess. Rose-Bradford [19] says, "that its being a complication of other diseases and injuries probably explains its being so frequently overlooked, its physical signs usually being attributed to other causes." There is a surprising dearth of references to this condition in the literature of general medicine. Norris and Landis [20] speak of massive collapse of the lung as a complication of pneumonia. Rose-Bradford [23] refers to it as a possibility in pneumonia and reports an autopsy in one case where there was a collapse of the lower lobe and pneumonia in the upper lobe. Tidy [21] reports an undoubted case of a massive collapse of the entire right lung in a healthy man twenty-nine years of age who was suddenly taken without any previous illnesses or premonitions with pain in the right chest. Upon entering the hospital forty-eight hours later he had the typical symptoms of a massive collapse, which disappeared entirely at the end of six weeks. The only etiological factor which Tidy could find was the immobilization of the lower right chest and the right diaphragm. This inhibition of the respiratory muscles Tidy felt was due to the pain probably caused by a pleurisy and the inhibition or arrest of respiration was followed by a collapse of the lung. Collapse of the lung in the newborn and in infancy has received considerable attention. Reynolds [22] speaks of airlessness in the lungs of newborn children and calls the condition apneumotosis. He was convinced at this early date, 1871, that the distribution of affected lobules was in direct relation to bronchial tubes, lobules supplied by one particular bronchial tube often presenting characteristic lesions, while lobules supplied by closely adjacent bronchial tubes may be perfectly healthy. In his mind this precluded the possibility of it resulting from an infection spreading by simple continuity. But literature contains the greatest number of references

to pulmonary collapse after diphtheria as described by Pasteur.[13] The military surgeons supply the next largest group of cases in those of traumatic origin, following unilateral wounds of the chest (penetrating or non-penetrating) non-penetrating wounds of the abdominal wall and occasionally wounds of the buttocks, pelvis and thigh. There has been an increasing interest of late in its relation to operative procedures.

Varieties.—As the clinical forms of massive collapse are quite similar irrespective of the variety, it will be possible to consider the subject with reference to the varieties merely from an etiological standpoint. Thus the clinical forms irrespective of their etiology may be (1) lobular, (2) lobar, or (3) total in distribution. In the lobular or partial type the upper or middle third of one or both lower lobes is the part most frequently affected. In the lobar type one or both lower lobes are usually affected. In the total variety the whole lobe is in collapse. Massive collapse not only varies in the extent of the area involved but also in the degree of airlessness, and it is this variation in the amount of air which accounts for weak or absent breath sounds at times and at others loud tubular or amphoric breathing. The similarity of the phenomena of collapse of the lung in post-diphtheritic paralysis to that found so frequently in the misnamed post-anæsthetic pneumonia was first called attention to by Pasteur [13] in 1914, and has aroused considerable interest; the English literature contains excellent clinical and experimental observations. Rose-Bradford [23] gave the first exhaustive discussion of the phenomena which he had encountered so frequently as a result of gunshot wounds of the chest. That it was not fully recognized by him until after very extensive experience with chest cases probably means that it is really much more frequent than he found. He reports his belief that it occurs in fully 10 per cent. of all non-penetrating injuries of the thoracic wall. The most readily recognized and certainly the best for study are those cases which are associated with non-penetrating wounds of the chest wall and especially those which curiously occur on the side opposite to that injured. He did not have the opportunity of seeing patients who had wounds of other portions of the body, his work being confined to those of the chest, but as we have elsewhere stated he had knowledge of its occurrence following abdominal wounds, wounds of the pelvis, buttocks and lower extremities, but no cases with wounds of the head or upper extremities. The varieties encountered in thoracic wounds he divides into homolateral, contralateral and bilateral, all of which may be lobular, lobar or total. The contralateral variety of massive collapse involving the whole of one lung is a very remarkable condition, more especially as in many cases the wound on the opposite side is not only non-penetrating but most trivial in character, causing no fracture nor indeed any extensive injury of the chest wall. Personal communications from a number of American, English and French medical officers in the late war has given evidence that this phenomena was frequently recognized but unexplained. In war, of course, the determination of its earliest establish-

ment after the receipt of the wound was quite impossible, but Rose-Bradford reports that he saw a case that was completely established with total massive collapse of one lobe, fourteen hours after the receipt of the injury. Although all this has a definite surgical bearing, the part which is germain to the present discussion is its association with operative procedures.

Etiology.—For a condition which we have seen may develop as a congenital abnormality as in the apneumotosis, or atelectasis of the newborn; which may develop spontaneously, apparently being caused by an acute pleurisy; which follows post-diphtheritic paralysis of the respiratory muscles; which follows infection of the lung and of the bronchi themselves, as pneumonitis and purulent bronchitis; which follows non-penetrating traumatic injuries of the chest and of the adjacent abdominal wall, buttocks, pelvis and lower extremities, and that has an approximate incidence of about at least 6 per cent. in abdominal operations; for such a condition it would seem difficult to find a common etiological factor. This is apparently true, for in the discussion of its etiology there is at the present time no definite consensus of opinion. Various theories, of course, have been offered. That it can be caused entirely by paralysis of the diaphragm or respiratory thoracic muscles is proven by Pasteur in his post-diphtheritic phrenic paralysis. Pasteur in his article [17] quotes experiments of Martin and Hare in which lungs were found collapsed in cases of death occurring in animals as the result of section of both phrenic nerves. Briscoe,[18] experimenting with normal rabbits, divided the phrenic nerve on one side of the neck and was able to obtain varying degrees and location of pulmonary collapse following this procedure. Curiously the deflation was not limited to the same side as the paralyzed half of the diaphragm. The opposite lung was affected in almost the same area and frequently to a greater degree. He also was unable to obtain, which is rather important in view of some of the theories, any evidence of a reflex paralysis or arrest of one-half of the diaphragm as the result of intra-abdominal irritation. Of course, as he said, these conditions were all tried upon normal animals and not ill ones as in the cases of Pasteur. He reports three observations upon cases of spinal paralysis, due to injury and paresis, in which there was a complete paralysis of the cord high up. In these cases he found complete deflation of the pulmonary lobes. Schroeder and Green [24] state as the result of clinical and experimental work with animals and birds: (1) That the diaphragm is not an essential muscle of respiration. (2) That the nerve supply is practically entirely dependent upon the phrenic nerves. (3) That after section of the phrenic nerve the intercostal nerve supply is sufficient to carry on the action of the diaphragm. (4) That section of one phrenic nerve produces collapse of the lower lobe of the lung on the affected side. This, of course, was not in agreement with the work of Briscoe. (5) The destruction of one phrenic nerve in man, is not necessarily fatal. Pearson-Irvine [25] report a case of diphtheritic paralysis of the thoracic muscles (auxiliary muscles of respiration) with an overacting of

the diaphragm. In this case there was a definite collapse of the upper lobe of the lung. He is perhaps the first to suggest that this collapse of the lung is not only due to lack of movement of the thoracic cage but also to some extent to a paralysis of the muscles of the bronchial tree. Lictheim [26] produced a definite collapse in the lung tributary to bronchi in which he had placed laminaria plugs. These experiments were performed with rabbits. This theory of bronchial obstruction is one which has appealed to many men. Dingley and Elliott [27] suggest that in man consequent to immobilization of the thoracic wall and diaphragm, irrespective of its cause, secretion collects in the bronchioles and even in the larger bronchi sufficient to prevent the egress of air and leads to a gradual absorption of the alveolar air by the pulmonary circulation and ultimate collapse and airlessness of the lung tissue. We have been able to confirm this by autopsy in one of our cases, a case of a strangulated femoral hernia which was operated upon under local anæsthesia. The collapse apparently occurred on the third day following the operation. This was demonstrated by X-ray. On the fifth day he had definite signs of pneumonic consolidation (lobar) of the upper right lung and he died on the tenth day. Autopsy showed a definite collapse of the lower right lobe with a purulent pneumonia in the two upper lobes. In tracing the bronchus of the lower lobe a definite plug of purulent mucus was encountered which blocked the tissue tributary to it. Grailey and Hewitt [22] suggest the curious explanation, that the tapering funnel-like character of the bronchial tree would necessarily have an action upon the obstructing plug similar to that of a ball valve. The effect of inspiration being to propel the plug towards the alveolar tract, and jam it when it arrives at a bronchiole whose calibre is less than that which it originally occupied and during expiration it would be dislodged, allowing the air to escape from the alveoli. Rose-Bradford is inclined to feel that obstruction does not play an important rôle. He emphasizes the fact that it is well-known that insufficient expansion of the chest, however produced, is capable of causing collapse of various degrees in the underlying lung. In some instances a constrained posture or a prolonged recumbency is sufficient to cause quite extensive collapse, involving, for instance, one lobe of the lung. Briscoe [18] agrees in part with this statement, and after his experimental work with animals and more or less analytical study of posture and respiratory movements of various individuals, says that massive collapse of the lower lobes of the lung is a natural sequence of prolonged quiet breathing in the supine position in such people as do not use the abdominal muscles to fix their chest. He suggests that it is circulatory in its actual beginning, that as a result of the inhibition of the respiratory muscles an œdema of the pulmonary tissues develops following which collapse takes place. Rose-Bradford [19] also feels that in some unexplainable way this condition is brought about by reflex action, particularly when it follows injury on the opposite side of the chest, upper abdomen and lower extremities, though, as has been stated, Briscoe was absolutely unable to demonstrate this experimentally. Cymble [28] naïvely

suggests that a man with a unilateral chest wound would usually lie upon the unwounded side and the consequent immobilization of respiratory movements are the real cause in the production of contralateral collapse.

To recapitulate the explanation probably lies in more than one factor. First, to bronchial obstruction due to mucus plugs or foreign bodies or possibly to some paralysis or bronchial spasm due to a reflex irritation from other parts of the body. Secondly, to arrest of the respiratory muscles either of the chest walls or of the diaphragm itself, such arrest being caused by direct nervous influence but in a large proportion of cases by posture.

Progress.—In discussing the progress of the condition, resolution usually requires from ten to twenty-one days for return to normal. The patients, at least those on record, practically never die from this condition unless it be bilateral massive collapse.

Complications.—It is the complications which produce the mortality. If inflammation supervenes, râles may appear and a friction sound be heard over the accompanying pleuritis. Expectoration rarely appears until pneumonitis is established, and it is rarely bloody until this stage is reached. Effusion has been known to occur. When the condition occurs as a complication of bronchitis, according to Norris and Landis,[20] or bronchial pneumonia, or whooping cough, it is usually attended by a blocking of the smaller bronchi. Its presence is inferred largely by an increase in the severity of the symptoms rather than by any other sign. Purulent bronchitis undoubtedly occurs as a complication, as is evidenced in our case. Pleurisy is not uncommon as a late complication. It is usually the dry variety giving rise to a friction rub, but effusion may occur later. Pneumonia, according to Rose-Bradford, is usually limited to the collapsed lobe.

Differential diagnosis must be made from acute dilatation of the heart, pulmonary embolus, pulmonary infarct, pleuritis, with or without effusion, and pneumothorax. If one bears in mind that the affected side is retracted and immobile; that the diaphragmatic and cardiac encroachment on the affected side is extreme; that the general symptoms are invariably less severe than with pneumonia, embolism and infarction; and the marked hyperresonance and increase of breath sounds with loud transmitted spoken voice sounds, the diagnosis should be made. To this should be added the high level of the diaphragm and the question of the displaced cardiac impulse. Such errors as subphrenic collection of gas and fluid have been made to account for the upward displacement of the heart and diaphragm on the side of the collapsed lung. Pneumothorax is also a frequent mistaken diagnosis. Cardiac dilatation has been an explanation of the misplaced cardiac area. The fact that even in cases of marked cardiac displacement the pulmonary physical signs may be comparatively slight, often gives rise to a mistaken diagnosis, such as dextrocardia, but after a lapse of a few hours or days there is usually a development of the pulmonary signs and, which is more conclusive, the return of the heart to its normal position. The upward displacement

of the diaphragm is a sign of the greatest importance and it is detected on the left side by physical examination and on the right by X-ray. It should also be remembered that when the entire lung or the upper lobes are collapsed the displacement of the cardiac impulse is oblique. For diagnostic purposes Rose-Bradford divides the physical signs into three periods. In the first the signs are retraction and immobility of the affected side, together with the weakness or absence of breath sounds and displacement of the heart which is often extreme. In the second weakness of the breath sounds has been replaced by loud tubular or amphoric breathing together with increased vocal fremitus, loud bronchophony, pectoriloquy and transmitted spoken voice. In the third period, the stage when the lung is expanding, abundant râles may be present over the area where the tubular breathing is marked. In both the second and third stages the heart is still displaced, but as we have previously mentioned, the lung signs may sometimes persist over a small area, at a time when the heart has returned to its normal position.

Prognosis and Mortality.—It is impossible to estimate the mortality at the present time because of the general lack of recognition of the condition. We believe we have had two fatal cases during the last six months and obtained an autopsy in one.

Our purpose in presenting this subject before such a body is to obtain the real incidence of post-operative massive pulmonary collapse. We are convinced it is far greater than Pasteur's 6 per cent., which he reported in 1914. We offer the suggestion that collapse of the lung in varying degrees always follows any operative procedure, and any traumatic or inflammatory injury of the chest and trunk which may cause inhibition of normal respiratory movement by pain or posture. We also believe that it is a constant factor in all post-operative pulmonary complications.

CONCLUSIONS

We suggest that the phenomena of pulmonary collapse of varying degrees, together with pulmonary embolism and infarction, are the real etiological factors in post-operative pulmonary complications. That all other factors, such as anæsthesia, infection (either pre- or post-operative), preëxisting lung disease, old age and debility, and the chilling of the body are contributory only.

We wish to express our obligations to the following contributors to this subject for the use of and many quotations from their writings.

Since the reading of this paper three more cases have been recognized and demonstrated by Röntgen.

REFERENCES

[1] Cutler: Surgical Clinic of North America, vol. ii, pp. 935, 945, Aug., 1922.
[2] Cutler and Hunt: Arch. Intern. Med., vol. xxix, pp. 449–481, April, 1922.
[3] Pepper: Med. Clin. North Amer., 1921, vol. v, pp. 737–749.
[4] McKesson: Amer. Jour. Surg., vol. xxxii, p. 16, 1918, Quar. Sup. Anæsthesia.

[5] Norris: Ill. Med. Jour., Oct., 1921.
[6] Chevers: Guy's Hospital Reports, 1876.
[7] Mandl: Deutsch Zeit. f. Chir., July, 1921, p. 67.
[8] Gottstein: Klin. Arch. f. klin. Chir., vol. lvii, p. 409, 1898.
[9] Mikulicz: Zerhandl. d. deutsch. Gesellschr. f. Chir., vol. xxx, p. 560, 1901.
[10] Henle: Zerhandl. d. Deutsch. Gesellschr. f. Chir., vol. xxx, p. 560, 1901.
[11] Sauerbrock: Zeitr. d. klin. Chir., vol. cxxii, p. 234, 1921.
[12] Mandl: Wien. klin. Wnschr., vol. xxxiv, p. 214, May 5, 1921.
[13] Pasteur: Amer. Jour. Med. Sciences, 1890.
[14] Bibergeil: Koerter's Clinic, Berlin, 1906.
[15] Rupp: Arch. f. klin. Chir., Berlin, Mar. 21, 1921, vol. cxv, p. 672.
[16] Pasteur: British Jour. Surgery, 1914, vol. I, p. 587.
[17] Pasteur: Middlesex Hospital Reports, 1910, Lancet, London, 1910.
[18] Briscoe: Quarterly Jour. Med., 1919–1920.
[19] Rose-Bradford: Oxford Loose Leaf System Med., vol. ii, pp. 127–137.
[20] Norris-Landis: Diseases of Chest, 1922, p. 487.
[21] Tidy: Lancet, London, May 2, 1914, p. 1245.
[22] Grailey and Hewitt: Reynolds System of Med., 1871, p. 865.
[23] Rose-Bradford: Quarterly Jour. Med., vol. xii, 1918–19.
[24] Schroeder-Green: Amer. Jour. Med. Sc., New Series 123, p. 196.
[25] Pearson-Irvine: Clin. Transact. Lon., vol. ix, 1876.
[26] Lictheim: Arch. Exper. Path. u. Pharm., vol. x, p. 54.
[27] Elliott-Dingley: Lancet, London, May, 1914.
[28] Cymble: Brit. Jour. Surg., vol. v, p. 363, Jan., 1918.
[29] Pasteur: Bradshaw Lecture, London, 1908.
[30] Scrimger: Amer. Jour. Surg., vol. xxxvi, pp. 53–60, April, 1922.
[31] Herb: J. A. M. A., vol. lxxix, pp. 339–342, July 29, 1922.
[32] Hirschbroeck: Amer. Jour. Med. Sc., vol. clxiv, pp. 268–274, Aug., 1922.
[33] Scrimger: Surg., Gyn. and Obst., No. 6, p. 32.
[34] Mortimer: Medical Press and Circular, vol. cviii, 1919, pp. 507–09.
[35] Schlotz: Med. Jour. of South Africa, Jan., 1921, vol. xvi, p. 202.
[36] St. John:
[37] Hampton and Wharton: Johns Hop. Med. Bull., Apr., 1920, vol. xxxi, p. 95.
[38] Hall: Lancet, London, Jan. 15, 1922.
[39] Barling and Levestre: Manual of War Surgery, p. 191.
[40] Elwyn-Girsdansky: J. A. M. A., Aug. 26, 1922, No. 9, vol. lxxix.

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SWELLINGS OF THE SUBMAXILLARY REGION

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Immediately beneath the lower border of the mandible lies a region peculiarly related to the structures of the mouth in that it is frequently the seat of secondary manifestations of disease arising within the mouth and lower jaw. The boundaries of the submaxillary triangle are: Above, the lower border of the mandible and a line drawn from the angle of the mandible to the tip of the mastoid process; below, the anterior and posterior bellies of the digastric muscle. The coverings of the triangle are: The skin and the superficial cervical fascia, the platysma myoides and the deep cervical fascia. The superficial fascia and the platysma myoides form an inseparable layer attached above to the lower border of the mandible and blending imperceptibly into the superficial fascia and muscles of expression of the lower lip and chin. Beneath the platysma lie the submaxillary and submental lymph-nodes, which receive the lymphatics draining from the lower gums, floor of the mouth and tongue. Posterior to these and somewhat deeper, just beneath the angle of the jaw is the deep cervical node receiving drainage from the fauces and tonsil. Beneath the deep fascia is the submaxillary salivary gland. The floor of the submaxillary triangle is formed by the hyoglossus and mylohyoid muscles, the latter separating it from the mouth. The duct of Wharton leaves the under surface of the submaxillary gland, passes around the posterior edge of the mylohyoid muscle, then runs forward on the upper surface of this muscle beneath the mucous membrane of the floor of the mouth, and has its outlet in a papilla at the side of the frenum of the tongue. Other important structures lying within the submaxillary triangle are, the facial (external maxillary) and lingual arteries, the facial (anterior facial) vein, the branches of the facial nerve to the platysma and muscles of the lower lip, the hypoglossal and glossopharyngeal nerves. The facial artery is given off from the external carotid and passes upward and forward through the submaxillary gland to the facial notch which is a depression that can be felt in the border of the mandible about 2 cm. in front of the angle. Two branches of this artery, the submaxillary and submental, are given off in the submaxillary gland. The facial vein is also intimately connected with the submaxillary gland. The facial nerve branches to the platysma and muscles of the lower lip lie in the superficial fascia. The hypoglossal and glossopharyngeal nerves lie deeper and are not concerned in this paper.

There appears to be misapprehension about several conditions manifested by swelling in the submaxillary region. Sébileau called attention to this con-

fusion in an admirable paper in the Presse Médicale, March 16, 1921. He points out the common error of regarding the acute inflammatory swelling in the submaxillary region following dental abscess as a lymphadenitis instead of a cellulitis by direct extension from the periosteum of the mandible. The course of an acute dental abscess beginning in the lower jaw depends entirely upon the place at which the pus perforates the bone. (The quotations following are from Sébileau's article.) "The abscess is at first intra-alveolar, then intraosseous; it then becomes subperiosteal, and finally extraperiosteal, provoking around it a more or less extensive cellulitis, which resembles lymphadenitis. If the perforation takes place at the level of the alveolar process of the jaw, there is formed a swelling in the vestibule of the mouth with a buccal opening or an opening on the skin of the face; if, on the contrary, the perforation occurs at the level of the body of the bone, a true submaxillary phlegmon is formed." The location of the swelling differs somewhat according to whether the outer plate, the inner plate, or the lower border of the mandible is perforated. That this process is one of osteoperiostitis and not lymphadenitis is shown by several facts: (1) The submaxillary swelling communicates with the alveolus. This can be demonstrated by pressure over the swelling causing pus to be discharged from the socket of the tooth. (2) If the submaxillary swelling is incised through the skin, with a probe a more or less extensive surface of denuded bone can always be felt. (3) All cases, and more especially those involving the posterior part of the mandible are accompanied by trismus or limited opening of the jaws. "The early onset and extent of the trismus bear a close relation to the nearness of the lesion to the wisdom tooth and the angle of the jaw. It would be an error to believe that trismus is entirely and constantly absent in lymphadenitis of the submaxillary and retroangular region, but here it is never so intense." The trismus is due to a fusion of the jaw bone with the inflammatory mass, and is the most important sign in diagnosis of osteoperiostitis. "Except for a few cases of diffuse osteomyelitis in children (and even this is open to discussion), mandibular osteomyelitis should practically always be regarded as of dental origin, either the consequence of dental caries, pericementitis, maleruption of the third molar, or retained teeth." Even those cases following fracture or other trauma are nearly always due to dental disturbances. Consequently, in every case manifesting an inflammatory swelling of the submaxillary region, accompanied by trismus, dental pathology should be suspected. Owing to the trismus, proper clinical examination of the inside of the mouth may be impossible, and much dependence must be placed on the X-ray. It is not denied that acute submaxillary lymphadenitis can exist, but very seldom as a complication of acute dento-alveolar infection. Lymphadenitis in this region is nearly always due to ulcerations of the oral soft tissues, the gums, vestibule and floor of the mouth and tongue. In tonsillitis and inflammations about the fauces the lymph-node beneath the angle of the jaw is involved. These lymphatic swellings are almost never accompanied by trismus. They are generally more circumscribed in the beginning than the osteoperiostitis cases. The

submaxillary phlegmon complicating osteoperiostitis of dental origin usually requires drainage by incision beneath the lower border of the jaw. By planning the incision according to the point at which the pus approaches the skin either in front of or behind the facial artery, annoying hemorrhage from cutting this artery will be avoided. The tooth or teeth from which the trouble originates should be removed at the time the external incision is made or soon after, otherwise a sinus may persist or the condition will recur, or the trismus may develop into ankylosis. Even though a considerable portion of the surface of the mandible be denuded of periosteum, prompt incision and drainage with removal of the cause may result in healing without necrosis.

A not uncommon condition is that of an acute or subacute swelling in the submaxillary region, fairly well circumscribed and quite tender to pressure. In the acute cases the pain may be severe. The patient will often state that this painful enlargement comes and goes every time he catches cold. There is no limitation in opening of the mouth. This condition also is usually diagnosed as an inflammatory enlargement of the submaxillary lymph-nodes, and attributed to drainage from infected teeth or tonsils. As mentioned above, it is quite the rule for a tonsillar infection to be accompanied by a lymph-node enlargement, the tonsillar node being situated rather at the angle of the mandible than in the submaxillary triangle itself. Acute inflammation of the gums and other soft tissues of the mouth, as in Vincent's gingivitis, are also characterized by lymph-node enlargement, this time in the submaxillary and submental region. But it has been shown that an entirely different picture characterizes the swelling from infection arising in the teeth and mandible itself. Here we have direct extension of the inflammation to the submaxillary soft tissues from a periostitis, with marked trismus. In the absence of an inflamed tonsil or acute stomatitis, what other conditions may cause the symptoms mentioned, *viz.*, acute or subacute circumscribed tender swelling in the submaxillary region? We must not overlook the presence in this region of the submaxillary salivary gland and the possibility of its enlargement from obstruction of Wharton's duct by a salivary calculus or by inflammation without calculus. It is this condition that is most commonly mistaken for a lymphadenitis due to infection from teeth or tonsils. In most of the cases that have come to our notice the patients have been told that the trouble was due to infection from the teeth, and have had one or more teeth extracted without relief. In typical acute inflammatory obstruction of the duct of Wharton by a calculus no difficulty should be encountered in diagnosis, yet even here the mistaken diagnosis of dental abscess is often made. The patient will generally give a history of several previous attacks, with increase of pain and swelling especially during meals (salivary colic). In addition to the tender circumscribed swelling in the submaxillary region, there will be painful œdematous swelling under the tongue and difficulty in swallowing. The outlet of the duct behind the incisor teeth may be reddened and pus may be expressed from it. An extremely tender nodule—the calculus—may be felt somewhere along the course of the duct in the floor of the mouth by combined intraoral and extra-

oral palpation. Occasionally, these symptoms and signs will exist from acute inflammatory obstruction of the duct without calculus. The diagnosis is easy also when the stone is seen to be spontaneously extruded from the orifice of the duct. It is in the milder or subacute recurrent cases that there is more excuse for overlooking the true condition present. Here, the only symptoms may be more or less mild recurrent attacks of circumscribed tender swelling in the submaxillary region, with no particular complaint in the floor of the mouth. The likelihood of a stone should, however, always be thought of under these circumstances and careful palpation of the floor of the mouth will frequently reveal a point of tenderness or a hard nodule. The diagnosis will be confirmed by X-ray examination. A No. 2 film (2½ x 3¼ in.) is placed horizontally between the upper and lower teeth as far back in the mouth as possible with the sensitized side down, and the rays directed from beneath the chin. A calculus in the anterior three-fourths of Wharton's duct will cast a clear shadow on the lingual side of the teeth and jaw. If the stone is farther back, near the beginning of the duct, a lateral extraoral film may be required to show it. The extraoral method of examination for a small calculus in the anterior part of Wharton's duct is often unsatisfactory, as the stone shadow may be covered by that of the mandible.

The treatment of obstructive enlargement of the submaxillary gland by calculus in Wharton's duct is primarily removal of the calculus. If the calculus is in the anterior two-thirds of the duct this can be accomplished by an incision through the mucous membrane of the floor of the mouth under local anæsthesia. The cases vary greatly in difficulty. No doubt most of us have picked out with forceps a calculus impacted in the orifice of the duct. Where an incision becomes necessary, anæsthesia is best attained by injecting the lingual nerve as in the mandibular injection for extraction of teeth. A fine lacrymal probe passed into Wharton's duct often proves a valuable guide. The mucous membrane is incised in the direction of the duct, and in case of a large calculus the latter can then be readily felt with the finger, the duct incised and the stone liberated. In case of a small stone with little or no surrounding inflammatory reaction, the duct can be first isolated with the probe as a guide, and an incision made in it over the stone. In non-suppurative cases, the mucous membrane incision can be closed without drainage. If much acute inflammatory reaction or suppuration be present, a small wick of gauze or strip of rubber dam should be left in the incision. There is usually considerable reaction following the trauma of this operation, lasting for a few days, which may be partially controlled by hot mouth washes and the application of ice externally. Pain may call for a sedative. If the calculus lies near the point at which Wharton's duct is given off from the gland, removal by external incision is indicated. In cases of long standing, the gland undergoes degenerative changes from chronic inflammation, and even removal of the stone does not effect a return to normal. Here, it is advisable to remove the gland as well as the stone. In this operation, the usual skin incision runs about an inch below and parallel with the lower border of the mandible from just behind the symphysis to the angle. The platysma is divided along the same line and turned up as a separate layer. In dissecting out the gland the facial artery and vein are divided and tied. In completing the operation the platysma and skin are sutured in separate layers. A small rubber dam drain is usually inserted for twenty-four to forty-eight hours. Very frequently some of the cervical branches of the facial nerve are cut during the operation, causing a characteristic inability to depress the corner of the mouth due to paralysis of the triangularis menti muscle. Improvement may occur with time. This accident is difficult to avoid, but there is less chance of its occurrence if the incision be made well below the border of the jaw.

Case I.—J. D., male, aged forty, reported that two years previously he began to have attacks of soreness in the floor of the mouth and difficulty in swallowing associated with inflammatory swelling in the right submaxillary region. The pain and swelling increased at meal times. The attacks occurred every two or three months, would be very severe for a few days and then gradually subside. On two occasions during attacks he had supposedly abscessed teeth in the right lower jaw extracted, without preventing recurrence. When seen on February 10, 1924, patient had been suffering from severe pain in the right submaxillary region and back part of the floor of the mouth, and had been unable to swallow anything but liquids for several days. There was a large, tender deep-seated swelling in the right submaxillary region about the size of a small hen's egg, surrounded by redness and œdema. The right side of the floor of the mouth was red, tender and swollen, the orifice of Wharton's duct being distinctly inflamed. No definite nodule could be felt in the floor of the mouth on bimanual palpation. There was practically no limitation of opening the mouth. An intraoral X-ray film showed no calculus in the anterior two-thirds of Wharton's duct. A lateral extraoral film, however, showed a large calculus between the posterior part of the lower border of the mandible and the hyoid bone. February 12, 1924, under ether, through an incision one inch and parallel to the right lower border of the mandible, the submaxillary gland and the calculus were removed. In order to get at the stone, which was embedded in a dilatation of Wharton's duct beneath the gland, it was necessary to dissect the latter entirely free. The wound was closed in two layers, interrupted catgut for the platysma and fascia and dermal suture for the skin. A small rubber dam drain was left in place for forty-eight hours. There was considerable purulent discharge for four or five days, but the patient recovered completely in two weeks. Pathological examination revealed chronic inflammatory changes in the gland.

Cases of chronic inflammatory enlargement of the submaxillary gland with recurrent acute attacks are also met with in the absence of calculus. These are due to obstructive inflammation of the duct, and give rise to the same symptoms as those of obstruction due to calculus. If persistent these also demand excision of the gland. Case II is an example of this condition:

Case II.—P. R., physician, aged fifty, presented a rather interesting history of obstruction of several ducts. Eight years ago he was operated upon for obstruction of the bile duct, and two days later for an abscess of the right parotid gland. About two years ago, while eating, he first noticed a soft swelling in the right submaxillary region, which disappeared after a few days. Since that time he has had many attacks, varying in severity, of painful swelling in the right submaxillary region, with inflammation beneath the tongue and difficulty in swallowing. When first seen early in September, 1924, there was a hard, tender swelling about the size of a walnut in the right submaxillary region. The floor of the mouth on the right side was red, tender and somewhat swollen. No nodule could be felt, and a lacrymal probe could be passed well back in Wharton's duct

without encountering a stone. Intraoral and extraoral X-ray films were negative for calculus. Within the next few days improvement occurred, but then the symptoms suddenly grew worse. September 13, 1924, the right submaxillary gland was removed. After a few days of muco-purulent discharge the wound healed. There is slight paralysis of the right corner of the mouth, which is gradually becoming less apparent. Examination of the gland after operation failed to reveal any calculi but showed chronic inflammatory changes.

Other Conditions Causing Swelling in the Submaxillary Region.—Carcinoma beginning in the mucous membrane of the cheek, gums, floor of the mouth or tongue is usually accompanied in its late stages by metastatic deposits in the submaxillary lymph-nodes. The diagnosis of submaxillary enlargement from this source generally presents no difficulties because of the presence of the primary lesion within the mouth. Rarely, a carcinomatous involvement of the lymph-nodes in this region occurs in which it is difficult to locate the primary source of the disease. Recently a patient was seen with metastatic carcinoma of the submaxillary lymph-nodes, in whom the primary lesion was in the ethmoid cells. The ethmoid disease was not discovered until several months after appearance of the submaxillary swelling.

There are several other important conditions which give rise to swelling in the submaxillary region, most of them involving the lymph-nodes, which will not be taken up in detail here. Among these are syphilis, tuberculosis, certain forms of leukemia, dermoid cysts and ranula. In conclusion, I wish to emphasize the differentiation between the three most common acute inflammatory swellings appearing beneath the border of the mandible:

(a) Infection from the teeth and alveolar process causes a periostitis with extraperiosteal cellulitis and submaxillary phlegmon, not through lymphatic channels but by direct extension into the soft tissues from the periosteum. It is characterized by marked trismus, or limited opening of the mouth, particularly when the molar teeth are implicated.

(b) Infection from the gums, mucous membrane of the floor of the mouth, tongue or tonsillar region, causes submaxillary lymphadenitis, the swelling here being unaccompanied by trismus of any consequence.

(c) Obstruction of Wharton's duct by calculus or by inflammation without calculus may cause acute inflammatory enlargement of the submaxillary salivary gland. There is nearly always evidence of inflammation beneath the tongue, swelling and pain increased on eating, X-ray may show stone, or the latter may be palpated. Trismus is not a prominent feature.

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THE SURGICAL ASPECT OF BLOOD DYSCRASIAS ASSOCIATED WITH SPLENOMEGALY

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Any discussion of blood dyscrasias associated with splenomegaly must take into consideration a review of the function of the spleen and its action in health or disease on the blood and the blood making organs. Without going into the subject of splenic enlargements in general, I shall discuss certain forms of splenomegalies which are accompanied by disease of the blood, and which are becoming of increasing importance because of the attempt to cure or arrest the diseased process by removal of the spleen. I shall review briefly the principal facts brought out by the intensive investigations which have been carried out in this field of research and those facts proven by clinical experience.

It is an accepted fact that the spleen plays a definite rôle in the formation of red blood cells in embryonic life but this function is lost either shortly before or after birth, but is resumed in certain anæmic states in which the spleen reverting to the embryonic type, produces red cells. During infancy and in early adult life, when the germinal centres of the spleen are most active, lymphocytes are found and gain entrance to the circulation by contractions of the spleen. Other varieties of leucocytes, such as the polymorphonuclear and myelocytes, do not occur normally in the spleen, although they may appear in certain forms of anæmia and in myelogenic leukemia.

A more important function of the spleen is that which is concerned in the destruction of worn out red cells. These cells are taken up in the spleen by phagocytes which aid in their fragmentation, then carried to the liver or elsewhere in the reticulo-endothelial system for final destruction and the hæmoglobin changed into bilirubin. The function of red blood cell destruction is carried on after splenectomy by the bone-marrow or lymph-nodes which undergo hypertrophy and assume the splenic function of red cell destruction. In this manner is explained the fact that removal of the spleen is not followed by any alteration in the formation of bile pigment nor is there any accumulation of pigment in the circulation as the red blood cells are phagocytosed anywhere in the body and carried to the liver. When red cell destruction is so pronounced that the hæmoglobin is more than the normal amount which can be taken care of by the liver and transformed into bilirubin, the excess of pigment accumulates in the blood and jaundice occurs.

The rôle which the spleen plays in digestion is not known, although some inter-relationship between it and the stomach has been supposed to exist from the earliest times. Inlow's [1] experiments on this subject and a critical review of the literature have led him to conclude that a definite pepsinogenic function

of the spleen has not been demonstrated and that the relation of the spleen to gastric secretion is probably vascular, the diminution in the amount of gastric juice secreted after splenectomy being attributed to decreased gastric blood supply from injury to the gastro-splenic circulation.

Other observers have thought that the spleen may have an internal secretion which functionally influences some portion of the digestive apparatus by way of the blood stream or of activating one or more of the digestive enzymes by means of this secretion. Krumbhaar [2] believes that a specific splenic hormone is activated by passage through the liver. Mayo on the other hand asserts that the spleen does not possess an internal secretion of importance, not only because removal of the normal organ does not disturb metabolism, but also because of its extremely limited sympathetic nerve supply, as the organs concerned in internal secretion act through the sympathetic nervous system.

On purely hypothetical grounds, Kahn [3] thinks it probable that the spleen develops certain enzymes which are important to its function but it is equally evident that the function of the spleen is shared by other lymphoid or adenoid tissues of the body and that on removal of the organ, its function is continued by these other collaborating structures. After splenectomy the nodes along the greater curvature of the stomach and omentum hypertrophy and become red and new ones develop in the region of the extirpated spleen. Hyperplasia of the lymphatics develops, first in the vicinity of the portal vessels and then inside the liver lobes. This compensatory hyperplasia explains the liver enlargement common after removal of the spleen.

The spleen has been regarded as an important structure in filtering from the circulation various microörganisms and thus acts as a defensive mechanism against disease. In all probability its function as a filter has been overestimated. That it plays a rôle in resisting infection seems to be proven from the vast amount of experimental evidence which indicates that the spleen assumes an active part in the development of resistance against various acute infections, and this function after splenectomy may be assumed vicariously by other body structures.

The effects of splenectomy have been studied extensively in animals, and also in man in those cases in which the organ has been removed for rupture. We should bear in mind, however, that these observations have been noted in healthy individuals, and far different ones may be observed when the spleen is removed for definite pathological processes. In the normal splenectomized animal a secondary anæmia develops either immediately or after a short interval and persists for two or three months. Sometimes the whole reaction is delayed according to Kettle,[4] and occasionally the blood count becomes higher than before splenectomy. There is an immediate increase in the number of leucocytes in which the polymorphonuclears may increase three or four times their normal number, the count falling to normal in three to four months. The increase in the resistance of the red blood cells to hæmolysis after splenectomy is probably due to certain changes in the red cells themselves. This resistance rapidly follows removal of the spleen and lasts for months

and even years. There is a lessened tendency to jaundice which Pearce [2] explains as being due to diminution in the concentration of the products of red cell disintegration as they reach the liver. After removal of the spleen, red cell fragmentation is carried on largely by the bone-marrow located at such a distance from the liver, that the products reach the liver so diluted that jaundice is unlikely.

Following splenectomy there is an increase in the excretion of iron, a decreased output of vital fat in the feces and an increase in the fat and cholesterin in the blood. The output of uric acid and urobilin in the urine and feces is diminished. The thyroid may enlarge after the spleen is removed, which supports somewhat the view that there is some inter-relationship between the two organs. It has been shown in animals that the red cells and hæmoglobin decrease, and regeneration of red cells is retarded by thyroidectomy. The administration of thyroid gland in normal animals may increase the red cells up to 15 per cent. These experiments Mackenzie [5] points out, show under controlled conditions that the thyroid hormone exerts a certain influence on hæmatopoiesis, and that in hypothyroidism the blood picture often suggests a severe anæmia, resembling either chlorosis or primary anæmia without proclaiming itself by the usual signs and symptoms of myxœdema.

The connection which exists between the spleen and the bone-marrow has been investigated extensively by Pierce [2] and his associates who state that the divergent results obtained are characteristic of all phases of experimental work on the spleen and doubtless are to be explained by the fact that removing the spleen takes away only one organ of a system composed of liver, spleen, lymph-nodes and bone-marrow, and that the interrelations of this system may cause compensations of great importance in determining the degree of blood distribution or regeneration and hence the degree of change in the bone-marrow. The relation, therefore, is rather a matter of changes which take place in the storage and utilization of the iron of the body than a specific hormone action. Bone-marrow becomes red after splenectomy because it begins to take on the function of storing iron, for there is such a great deficiency of the iron content of the blood that red cells cannot be produced, as a high iron content is necessary for this function.

That the spleen has some stimulating influence on the blood-forming organs is shown by the fact that splenectomized animals recover from the anæmia produced by hemorrhage or poisons less readily than do animals with a similar degree of anæmia in which the spleen is intact. Stradomsky claims for the spleen a two-fold hormone action on the bone-marrow, an inhibiting one on the over-production of red cells in the narrow, and a second one controlling under-production of the cells. Normally these two influences balance each other, but when the splenic hormone is abnormal or lacking, the bone-marrow produces unlimited quantities of red cells of inferior quality which die off rapidly and this increased erythrocytosis sets up an automatic vicious circle. The findings indicate that the bone-marrow may function to excess after having been released from the controlling influence of the splenic hor-

mone. After splenectomy, the administration of splenic extract is liable to give results confirming this rôle of the spleen as a regulator of blood destruction.

A more detailed description of the investigations on splenic function can be found in the admirable reviews of Pool and Stillman;[6] and Pearce, Krumbhaar and Frazier.[2]

Pernicious Anæmia.—In endeavoring to find some means of checking the blood destruction in primary or pernicious anæmia, splenectomy was suggested by those who thought the cause of the anæmia was to be found in the spleen. We are still in the dark concerning the cause of this disease, and the most that can be admitted at present is that the anæmia is the reaction of the body and particularly the blood system to some toxin of unknown origin which greatly affects the bone marrow, causing on the one hand stimulation and over-production at a time when the marrow is attempting to overcome the action of the toxin.

Kahn and Torrey[7] have studied thirty-three cases of anæmia, in all of which the Welch bacillus was found in the duodenum, associated with symptoms characteristic of pernicious anæmia. On the administration of hydrochloric acid the organisms disappeared and the blood picture improved rapidly. The significance of these results must be determined by future observations. However, the subsequent events in the development of pernicious anæmia, as described by Moynihan,[8] include so marked an effort of the bone-marrow to meet the demand made upon it that the parent cells of the erythrocyte are liberated before their offspring daughter and granddaughter cells are created. The greater the demand made upon the bone-marrow the earlier is the type of cell liberated and set adrift in the circulation, and the character of the nucleated cell thus found may afford an index to the gravity of the disease. Death ensues because of the persistence of the increased destruction of red cells at a time when the efforts of the medulla to form blood break down under the ceaseless strain imposed upon it.

I shall not discuss the pathology and symptomatology of pernicious anæmia in any detail. The general degenerative changes and blood picture are too well known to warrant repetition. The spleen itself is but slightly enlarged in most cases, a factor of some importance in considering its removal in the treatment of the condition. The bone-marrow is red and contains numerous nucleated cells and some hyperplasia of the myeloid tissue is present.

Attention may be called to the insidious character of the onset of pernicious anæmia, beginning with a slow and gradual loss of strength and the development of pallor. Other important clinical manifestations are an achlorhydria, which is almost constant, and if not present, seriously jeopardizes the diagnosis of pernicious anæmia. The nutrition of the patient generally remains good, although loss of weight may occur when glossitis develops with a resulting disinclination to eat. In no other form of anæmia is glossitis so common, and in some cases it is an early and most annoying symptom, and occasionally it is the first symptom complained of and the one for which relief is sought. Spinal cord degeneration in both the lateral and posterior columns may occur

fairly early in the disease and produce tingling and numbness of the extremities, or more severe neuritic pains and symptoms resembling tabes.

As infection undoubtedly plays a rôle in the etiology of pernicious anæmia it is necessary first in treating the disease to remove any possible foci of infection, whether arising in the mouth, gastro-intestinal tract, genito-urinary tract or elsewhere. For the anæmia itself the various therapeutic agents used to stimulate red cell formation may be tried, and some transitory benefit may be secured. Sooner or later transfusion must be employed. The use of small transfusions, frequently repeated, seems to be the method of choice, and so far as I have been able to judge in a large number of patients so treated, the results are equally good with citrated or whole blood. The benefit thus secured is due to stimulation of the bone-marrow with an increased production of red cells. By transfusion the patient may be brought to a "remission stage," remain fairly comfortable for many months, but hæmolysis soon reoccurs and the improvement secured by transfusion is lost and the patient lapses into a stage of profound anæmia. For these apparently hopeless cases, Walterhofer and Schramm[9] have improvised a new method of treatment in which the marrow cavity of the long bones was irrigated through small holes drilled in the cortex. The improvement which followed was so striking and occurred so soon after the irrigation that the authors were led to believe that it could not be a mere coincidence and are hopeful that the method may prove to be of permanent value in the treatment of pernicious anæmia.

Splenectomy has been advocated by many investigators in an attempt to cure pernicious anæmia and a large number of spleens have been removed in the past twelve years with results at first believed to be gratifying and hopeful. Some of the more recent accounts of this operation, however, are not so encouraging and many surgeons have given up splenectomy. The cases which seem to be most favorable are those in which hæmolysis is most active, with symptoms less characteristic of the disease. The prospect of benefit is better in early than in late cases, and only temporary relief of symptoms can be expected when the process has produced such changes in the bone-marrow that its power of cell reproduction is lost.

In spite of splenectomy Carslow[10] believes that the condition goes on to a final result no more satisfactory than that obtained by medical measures, and he condemns splenectomy as unsatisfactory in pernicious anæmia, especially if the spleen is small. It is possible he concedes, that there are several types of pernicious anæmia and that operation may prove satisfactory in one and not in another variety.

There is no doubt that splenectomy was overdone for many years and Krumbhaar[11] thinks the operation now is being neglected and not employed as frequently as it should be. He has collected 208 cases of splenectomy done in patients suffering from pernicious anæmia, there were 35 deaths within one month, the post-operative mortality was 16.8 per cent., 26 were unimproved and 144 improved.

The study of end-results at the Mayo Clinic shows that the results of

splenectomy were far better than anticipated at a time when the operation was discontinued. It was found that 21.3 per cent. of the patients survived the operation three years or more, living two and a half times as long as the average in a similar group of non-splenectomized patients at the same stage of the disease, and that 10.6 per cent. are alive after more than five years.

These results clearly indicate in at least one-third of the cases that the average life of patients with pernicious anæmia is greatly prolonged, and in about 10 per cent. the prolongation is sufficient to lead to the hope that cures may result in some cases. While transfusion gives temporary benefit, the improvement following splenectomy has been far greater than that obtained by transfusion or any other palliative method of treatment. The changes brought about by splenectomy are manifested by improvement in the condition of the blood and amelioration of the nervous symptoms although the nerve structure itself is unchanged. The progression of degenerative changes in the cord are delayed to some extent and the relapses, so commonly seen in other methods of treatment, become less severe and less frequent. The achlorhydria persists even after splenectomy, and the glossitis seems to be but slightly benefited. These various manifestations, Mayo states, indicate that the same agent which destroys the bone-marrow, which injures the spinal cord, which causes achlorhydria and glossitis, also affects the spleen, and that by removing the latter a vicious circle is interrupted.

Hæmolytic Jaundice.—A very important blood dyscrasia, the result of splenomegaly is that of hæmolytic jaundice. The cause of this condition is unknown but as Elliott and Kanavel [12] pointed out the enlarged spleen is the active agent in the destruction of the erythrocytes and in the production of the resulting anæmia. The jaundice is of the non-obstructive type in that we find bile in the stools and not in the urine. This disease, seen most frequently in the second and third decades of life, occurs in two forms, the congenital or Chauffard-Minkowski type and the acquired or Hayem and Widal type.

The congenital form often affects several members of the same family, does not cause such severe symptoms and the patients are often more icteric than sick. The life expectancy is greater and the patients sometimes live to the fifth or sixth decades. The acquired type is more serious, and usually begins with severe symptoms, the anæmia becomes grave and the course is more rapid. This type is more frequently a disease of the adolescent period.

The chief characteristics of hæmolytic jaundice are splenic enlargement, usually moderate; icteric tinge of the skin and sclera, absence of bile in the urine and the presence of bile in the stools. One of the outstanding diagnostic findings is the increased fragility of the red blood cells as is shown in the fragility test in which their resistance to hypotonic salt solution is determined. In the congenital type we find increased fragility of the red blood cells in several members of the family, which Giffin asserts may be an aid in establishing the diagnosis. The increase of the urobilin in the duodenal contents and the presence of urobilinogen in the urine are probably due to the increased

blood destruction. We find also the greatest number of reticulated red blood cells in this condition.

In the course of the disease the patients have definite crises, during which there is an elevation of temperature, chills, increase in size of the spleen, increase in the jaundice and general malaise. The crisis may last from a few to a number of days when the symptoms subside. However, the jaundice, while appreciably diminishing, does not completely disappear. In the acquired type the crises are usually more severe.

It is not uncommon for patients with hæmolytic jaundice also to have gall-stones, which producing an obstructive type of jaundice, makes the diagnosis more difficult. Giffin [22] reports that 58 per cent. of the cases operated on at the Mayo Clinic for hæmolytic jaundice had gall-stones, and Moynihan found that 60 per cent. of his cases were so affected.

The pathological changes in the spleen are by no means characteristic and resemble the changes found in splenic anæmia. The capsule and trabeculæ are thickened, the Malpighian bodies are few in number and atrophic; there is a pronounced fibrosis throughout with atrophy of the splenic pulp. Increase in blood pigment as determined by the presence of hæmosiderin, is not marked.

The results of splenectomy in this condition are most striking and gratifying. The jaundice diminishes in the first twenty-four hours after operation, and in a few days the skin often has a normal color for the first time in many years. The anæmia rapidly disappears, the patient's health is improved and they remain well, showing that the spleen was the active agent in the destruction of the red cells. The fragility of the red cell decreases but does not return to normal. At the Mayo Clinic [13] fifty-one splenectomies have been performed for hæmolytic jaundice with only three deaths. Temporary relapse several months after operation has been noted by Giffin, while Elliott and Kanavel report a case in which a crisis occurred before the patient left the hospital; both patients, however, ultimately recovered and have remained free of symptoms.

Banti's Disease.—Banti's disease and splenic anæmia are now regarded as one disease, although there are some observers who believe that the symptoms of Banti's disease are the result of a terminal stage of splenic anæmia.

Three stages of splenic anæmia are described: (1) A preascitic stage, in which splenic enlargement is present with or without anæmia, the patient undergoing gradually increasing weakness. (2) The transitional stage in which the most prominent symptom is diarrhœa; the anæmia and blood changes are pronounced; the liver is somewhat enlarged and jaundice may occur; (3) The ascitic stage or Banti's disease proper.

The disease is characterized by a clinical course in which there is a progressive increase in the severity of the symptoms, this stage may last for many years. Probably the first symptom noted is enlargement of the spleen, which at first is gradual, then rapid in the late stage of the disease when the spleen may reach huge proportions, although not becoming so large as the splenomegaly seen in Gaucher's disease.

The anæmia met with is of the secondary type, but may become more severe in those cases in which frequent hemorrhages from the gastro-intestinal tract are met with.

The cause of the disease is unknown, although its cure by splenectomy naturally has led many to suppose that the pathogenesis of the affection is to be found as a primary disease of the spleen itself. However, no one has succeeded thus far in demonstrating any single factor which could be regarded as the cause of splenic anæmia.

The chief pathological condition found in the spleen in splenic anæmia is a generalized fibrosis, compression atrophy of the Malpighian corpuscles and endophlebitis. Chaney [14] from the pathological and clinical study of sixty-nine cases states that no changes were found in the splenic tissue that would enable the pathologist to positively diagnose splenic anæmia, yet the abnormality was as characteristic of this disease as in others producing splenomegaly. The average weight of the spleen was found to be 1015 grams and the average age thirty-three years. The disease affected both sexes equally with apparently no familial tendency.

It is believed by some that splenic anæmia is a clinical entity and that fibrotic splenomegaly produces anæmia, irrespective of the initial cause of the splenic enlargement. Mayo [15] favors this view and believes that a patient with chronic splenomegaly who presents characteristics of chronic anæmia, but who is not relieved by treatment is probably a sufferer from splenic anæmia, and will probably be cured by splenectomy without regard to the cause of the disease. In support of this theory we have the clinical evidence of improvement or cures following splenectomy in intractable cases of splenomegaly with anæmia, occurring in syphilis and chronic malaria.

While ascites may be present in splenic anæmia without fibrosis of the liver existing, it is possible that improvement following splenectomy may be due partially to diversion of some of the blood going to the liver, thus relieving it of overwork or toxins from the spleen, which in time may result in those fibrotic changes which lead on to portal obstruction, ascites, the formation of varicosities, rupture of which may cause severe and serious hemorrhages from the œsophagus and stomach. These hemorrhages, so common in splenic anæmia, and the cause of the more advanced degrees of anæmia, may cease entirely after splenectomy in early cases. In cases in which the hemorrhagic tendency has existed for a long time splenectomy may benefit but not stop the bleeding. In some instances the hemorrhages are probably the result of toxins formed in the liver which produce erosion of the gastric mucosa, and in this type of case, relief is less apt to occur after the spleen is removed.

The treatment of splenic anæmia is essentially surgical and consists in splenectomy. Preliminary to operation medical treatment may be tried to improve the general condition of the patient, although but little can be hoped for from these measures. Transfusion, both before and after operation, particularly if blood has been lost from hemorrhages from the mucous membranes, is beneficial but has only a temporary effect on the anæmia. It is highly

important to operate in the earlier stages of the disease, before dense adhesions form between the spleen and surrounding parts, particularly the diaphragm; before pronounced anæmia, liver fibrosis and ascites develop. Unfortunately the early manifestations of splenic anæmia are not sufficiently characteristic to make an early diagnosis likely and the surgeon too often is asked to remove a spleen in the terminal stages, at a time when the risk is great and the mortality correspondingly high. The adhesions between spleen and diaphragm may be so dense that removal of the spleen is impossible on account of the danger of hemorrhage, and the surgeon in such cases may be compelled to abandon the idea of splenectomy. The mortality in the early cases is not over 10 per cent., whereas, in the late stages of the disease it is about 25 per cent.

While the mortality is lower in the earlier manifestations of the disease, many cases recover after splenectomy performed in the terminal stages with ascites and liver cirrhosis present. With such secondary degenerative changes existing, cure of the disease cannot occur, whereas permanent relief can be said to have been obtained in the majority of the operations done in the early stages. It should be remembered, however, that not a few of the patients have recurrence of symptoms even after the lapse of many years.

Krumbhaar's [11] compilation is the most comprehensive collection of the results obtained by splenectomy in splenic anæmia. Of 239 cases collected, the post-operative mortality was 13.6 per cent., if the cases are subdivided into those occurring in recent years, a reduction to 11 per cent. is obtained, and it seems entirely likely that even this comparatively low mortality will be lessened with an increasingly improved technic and a more proper selection of cases. This is demonstrated by Mayo's [15] report of 10 per cent. of deaths in 82 splenectomies for splenic anæmia. Most of the deaths occurred in patients operated on in a late stage of the disease, in which there was a high degree of anæmia, ascites, and cardiorenal degeneration.

Gaucher's Disease.—This disease, first described by Gaucher in 1882, and regarded by him as a malignant process, has received much attention from the pathological standpoint, but less consideration has been given to it clinically because of its infrequency.

We are indebted to Brill, Mandlebaum and Libman for much of our earlier knowledge of the disease. The recent study of Cushing and Stout [16] who have analyzed forty-four cases, serves to bring more forcibly to our attention many of the clinical manifestations of the disease. They found that the affection ordinarily appears in childhood and runs a more acute course than in adults, in whom it is more chronic in nature. Females are more commonly affected than males (67 per cent.). Splenic enlargement which is noted in all cases, is progressive until almost the entire abdomen is occupied by the smooth splenic tumor. The bronze or yellowish discoloration of the skin, face, neck and hands is one of the striking features of the disease. The eyes show a peculiar wedge-shaped thickening of the conjunctiva which is present in most cases. No marked disturbance in general health may be noted for a long period when a secondary anæmia may appear and the patient

develops a tendency to bleed from the mucous membranes or into the skin. The blood picture shows a consistent leukopenia, the red cells may be decreased to a marked extent in the cases which bleed freely. The disease has a family tendency. Ascites and jaundice are rare, although enlargement of the liver is noted in 73 per cent. of the cases. Pain over the spleen is often complained of and pain in the region of the long bones may be a warning symptom of bone destruction, caused by the action of the Gaucher cells on the bone-marrow, and leading on to fracture.

Gaucher's disease, pathologically, is restricted to those cases in which the characteristic large vesicular cells with small eccentric nuclei, are found engorging the sinuses of the spleen, lymph-nodes and bone-marrow, or are crowded about the liver lobules.

In general the treatment of Gaucher's disease by internal medication, transfusion or radiotherapy has met with slight transitory improvement of the patient or no success at all. Splenectomy has been done in twenty-nine cases, with six deaths, an operative mortality of 20 per cent., of twenty-three patients who survived the operation, sixteen have gained in weight and strength and the hemorrhages have ceased. The operation seems to have lengthened the life of these patients to some extent. It can be concluded that splenectomy so far, is the only method of treatment that has met with any measure of success, although it cannot be stated positively that this operation actually cures the disease; rather it acts by producing an amelioration of the symptoms.

Purpura Hemorrhagica.—Many theories have been advanced in an attempt to explain the nature of this disease. While disturbance in liver function has been held responsible by some observers, the theory of infection advocated by Giffin and Halloway [17] has received the support of most writers on the subject. The disease is usually secondary to localized infection which produces chemical toxins whose action on the endothelium of the blood-vessels probably is the cause of the hemorrhages. The marked reduction in the number of blood platelets, without apparent cause, which is the main feature of the disease led Frank to designate the affection as essential thrombopenia and Eppinger to call it thrombocytopenia.

Brill and Rosenthal [18] regard purpura hemorrhagica as a distinct clinical entity, the main features of which are as follows: Reduction in the number of blood platelets from a normal of two or four hundred thousand to one hundred thousand or lower. A few cases are on record in which the platelets could not be demonstrated and I have treated one case in which two competent hæmatologists were unable to find blood platelets. As a general rule when the platelet count falls below sixty thousand the hemorrhagic tendency of the affection begins to appear, and these manifestations become pronounced when the platelets are under ten thousand in number.

Careful blood examination is of the utmost importance in the study of purpura hemorrhagica. The coagulation time of venous blood is preserved but the bleeding time is prolonged from a normal of one to three minutes to ten minutes or even hours. The capillary resistance test is an important diagnostic feature of the disease and may be demonstrated by applying a tourniquet to the arm sufficiently tight to prevent the return of blood without obliterating the pulse. If a purpuric tendency is present the test is followed by the appearance of multiple petechiæ. Another important blood phenomenon is failure of clot to undergo retraction, even when a fair number of platelets are present in the blood. This observation is useful as a differential test in hæmophilia in which the clotting time may be greatly delayed but when the clot is formed retraction always occurs.

From the clinical point of view purpura hemorrhagica pursues either an acute or chronic course. The acute cases result in a quick recovery, run a short and fatal course or become chronic. In the acute fulminating type the patients are so prostrated and so desperately ill that operative interference is out of the question and transfusion seems to be of little or no benefit. In the chronic form remissions sometimes occur making the diagnosis difficult if the patient is seen during this period. The capillary resistance test may prove of value as an aid in diagnosis at this stage.

When the disease occurs as a chronic affection it is encountered most frequently early in life and particularly in girls. Hemorrhages occur from the mucous membranes, are seen in the skin as multiple petechiæ and appear intermittently, as a continuous oozing or there may be excessive loss of blood following slight degrees of traumatism. Occasionally hemorrhages from the mucosa of the bowel may cause infiltration of the intestinal wall producing abdominal symptoms of pain, rigidity, tenderness, nausea and vomiting, fever, and leucocytosis, all symptoms suggestive of appendicitis. If such symptoms occur before other and more pronounced manifestations of purpura hemorrhagica have been noted there is scarcely any way of avoiding an unnecessary operation unless by some circumstance the surgeon happily should think of using the capillary resistence test.

Little can be expected from the treatment of purpura hemorrhagica by measures non-operative in nature. There is no doubt in some of the less severe forms of the disease that transfusion, preferably by whole blood, is followed by cure and particularly if any underlying infectious process can be removed at the same time. In the case quoted above with complete absence of platelets the bleeding ceased after a single transfusion the platelets increased rapidly in number and the patient has remained well for several years. Such a result must be exceptional for the condition of other patients has been uninfluenced by transfusion and in occasional cases indeed the condition seemed worse after transfusion.

Although the spleen is but slightly enlarged in purpura hemorrhagica there is no doubt that it is to be held responsible for the destruction of platelets. Accordingly its removal has been advocated as a cure for the disease, and in the cases in which this operation has been performed the blood platelets increase rapidly in number and the hemorrhages cease almost immediately. In some cases the platelet increase reaches its maximum only after many weeks, but as a rule the count returns to a low level shortly after the splenec-

tomy. Clopton [19] has reported instances in which there is a tendency toward recurrence of the disease in a greatly modified form in which the hemorrhages are readily controlled.

The immediate cessation of symptoms and apparent cure of the disease after splenectomy show that this mode of treatment offers the best chance of cure. Clopton has tabulated forty-five cases in which twenty-seven are reported as cured, fifteen improved, one unimproved and two operative deaths. The operative mortality is surprisingly low in view of the grave condition of most of these patients at the time of operation.

There are other obscure splenic blood disorders in which further study is necessary in order to properly classify them, and which are so little understood that operative interference by splenectomy is ill-advised. Farley [20] and others have called attention to the difficulty in distinguishing between aplastic anæmia, purpura hemorrhagica and acute myelogenous leukemia. In the light of our present knowledge we must agree with the contention of Hanrahan [21] and most writers on the subject that splenectomy is contraindicated in lymphoid leukemia, polycythemia, and the rapidly progressive fulminating forms of hæmolytic jaundice, splenic anæmia and pernicious anæmia. Providing the spleen has been treated previously by radium, which reduces its size and also at the same time, reduces the number of leucocytes, splenectomy offers the best chance of cure in myelogenous leukemia. While the number of cases so treated is comparatively small, the results seem to justify the operation in carefully selected cases, properly treated by radium before splenectomy is undertaken.

BIBLIOGRAPHY

[1] Inlow: Amer. Jr. Med. Sci., 1921, vol. clxii, p. 325.
[2] Pearce, R. M., Krumbhaar, E. B., and Frazier, C. H.: The Spleen and Anæmia, Philadelphia, 1918 (Lippincott).
[3] Kahn: Amer. Jr. Med. Sci., 1923, vol. clxxxv, p. 214.
[4] Kettle: Brit. Med. Jr., 1922, December 23, p. 1207.
[5] MacKenzie: J. Am. Med. Assn., 1926, vol. lxxxvi, p. 462.
[6] Pool and Stillman: Surgery of the Spleen, 1923, Appleton and Co.
[7] Kahn and Torrey: Penna. Med. Jr., 1926, vol. xxix, p. 394.
[8] Moynihan: The Spleen and Some of Its Diseases, Philadelphia, 1921 (Saunders).
[9] Walterhofer and Schramm: Archiv. fur Klinische Chirurgie, 1923, vol. cliv, p. 407.
[10] Carslow: Brit. Med. Jr., 1922, December 23, p. 1204.
[11] Krumbhaar: Amer. Jr. Med. Sci., 1923, vol. clxvi, p. 329.
[12] Elliott and Kanavel: Surg., Gynec. and Obstet., 1915, vol. xxi, p. 21.
[13] Mayo, W. J.: Jr. Amer. Med. Assn., 1924, vol. lxxxiii, p. 815.
[14] Chaney: Amer. Jr. Med. Sci., 1923, vol. clxv, p. 856.
[15] Mayo, W. J.: Jr. Amer. Med. Assn., 1925, vol. lxxxi, p. 487.
[16] Cushing and Stout: Arch. of Surg., 1926, vol. xii, p. 539.
[17] Giffin and Halloway: Medical Clinics of N. A., 1923, vol. vii, p. 241.
[18] Brill and Rosenthal: Arch. of Int. Med., 1923, vol. xxxii, p. 937.
[19] Clopton: Annals of Surgery, 1925, vol. lxxxii, p. 413.
[20] Farley: Amer. Jr. Med. Sci., 1925, vol. clxx, p. 10.
[21] Hanrahan: Annals of Surgery, 1925, vol. lxxxi, p. 906.
[22] Giffin: Surg., Gynec. and Obstet., 1917, vol. xxv, p. 152.

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